WEAVE

Viva+

THE MD PSYCHIATRY PRACTICAL

The illustrated practical book

Recognise. Locate. Explain.

The original 816-page text | 52 selected plates

Dr. Wilfred Dsouza | Dr. Niharika Reddy

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The original book, illustrated

This owner-approved image-set proof adds the selected captioned plates to the shipped original pages. Every original page remains intact, including its folio, references and credits.

Use the new PDF bookmarks, illustration list and original-page concordance. Printed folios inside original pages still refer to the original edition.

Read the map or topic first. Describe the image using its modality and anatomical plane, then name the pattern and state what this view cannot establish.

Real scans and recordings retain their source attribution. Schematics, when selected, are labelled explicitly. Captions are selectable text outside the artwork.

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THE PRACTICAL

MD and DNB psychiatry, practical examination revision

Weave Viva

The MD Psychiatry Practical

The long case, the short cases including neurology, the OSCE, the thesis viva and the six viva tables, written in the order the examiners ask. Nine complete sets of notes, in one book.

Dr. Wilfred D'souza · Dr. Niharika Reddy

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CENTRE FOR INTEGRATIVE PSYCHIATRY

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01
Your role

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02
The fit

Concern, age, language, format and preferred way of working can all shape the match.

03
The setting

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04
The coordination

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PSYCHIATRY VERTICAL

Weave is the psychiatry vertical of Trijog. Weave Sprint is part of its Psychiatry Outreach Programme, supporting psychiatry residents and clinicians with practical learning and a clear route into Trijog's wider care network.

The notes in your hands are part of that programme. Therapy through Trijog can sit alongside psychiatric care when the patient and clinicians choose.

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Routine referrals only. This is not an emergency route. For urgent risk, follow your local emergency and safeguarding pathway.

About this insert. Weave Sprint is educational outreach from Weave, the psychiatry vertical of Trijog. Therapy is provided through Trijog.

WEAVE SPRINT · THE PRACTICAL

Weave Viva

THE WEAVE SPRINT SERIES

TITLE

Weave Sprint, The Practical: Weave Viva

Part of the Weave Sprint series, MD and DNB psychiatry practical examination revision.

WRITTEN BY

Dr. Wilfred D'souza and Dr. Niharika Reddy

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EDITION

First compiled 2026. Free to read, share and print for study.

The clinical content is written for doctors and doctors in training. It is a study and revision aid, not a substitute for current guidelines or clinical judgement. Verify doses and thresholds against the current references and your local protocols before any clinical use.

For every resident who has ever sat up the night before,
afraid it will not be enough.

ACKNOWLEDGEMENT

A word of thanks

I want to begin by thanking something I cannot quite name. Call it whatever you like. I have never been certain what to call it, and I have stopped needing to be. There is a part of this work that does not feel like mine. The line that arrives before I have thought of it, the connection I did not plan, the steadiness on a morning I had no reason to be steady. I only hold the pen. Something moves through the holding, and I have learned to thank it without asking it for a name.

Someone once asked me why I give all of this away. Why hand out the notes I sat up making, to the very people sitting the same exam beside me. It is a fair question. If I had kept them, I would probably have scored a few marks more than the next person, and I might have been thought of as the better student. I have turned that over honestly, and I have decided it is a small thing to want. What I get instead, by putting the work in your hands, is the chance to take a little of the fear out of a great many nights. That trade is not close. Easing that fear is not a detour on the way to becoming a good psychiatrist. It is the thing itself. I did not come here to be the best student in the room. I came here to be a good psychiatrist.

And to Niharika, who reads everything first and knows when to be honest and when to wait; and to the residents who study from these pages and write back with the corrections and the questions: thank you. The book is better for you, and so am I.

Dr. Wilfred D'souza

FOREWORD

Foreword

This is the practical examination, gathered into one book. It is the part of the MD that is not written but spoken and shown: a patient across the bed, an examiner across the table, and a candidate who must say what was found, what it means and what to do, in the order those questions come. Every chapter here is arranged in that order, and the volume is a companion to the four papers of Weave Sprint, built the same way and for the same reader.

The notes were written to be understood, not just revised. Each chapter is a full account of its subject, from the mechanism up to the point the exam tests, so that you can read to learn the first time and read to recall every time after. Where a fact is worth holding exactly, it is marked. Where a mistake is easy to make, it is named before you make it. The colour code on the next pages tells your eye what kind of fact it is looking at before your mind has to decide, and after a few pages it stops being a key you consult and becomes a sense you carry.

Read it in order if you are meeting the material, or open it anywhere if you are returning to it. The contents at the front and the index at the back both jump to the page you want. However you use it, the hope held in these pages is plain: that one ordered, trustworthy place to study from makes the work a little lighter, and that you walk into the hall steadier than you would have otherwise.

HOW TO USE THIS BOOK

The colour memory code

Every note in this volume speaks one visual language, so once you learn it in the first chapter you read the rest without thinking about it.

Concept and rule	The core idea and the principle that follows from it. When a line is petrol, it is the thing to understand, and the thing worth being able to state.
Key number	A figure to hold exactly: a dose, a threshold, a prevalence, a cut-off. Amber means memorise this number, not just the shape of it.
Trap	The mistake the exam is waiting for. When a line is coral, it is warning you off an error that looks right until it costs you the mark.

Hold this is the single line from a topic you must carry out of it. **Good to remember** gathers, for each named thing, what it is, when it matters, and the one number or formula worth keeping. Both help a tired brain find the centre of a dense page fast.

The contents at the front is clickable, and so is the index at the back: every entry jumps to its page. Read to learn once, and to recall as often as you need.

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PART I

How the Practical Examines You

The scheme, the marks and what examiners are told to assess, then the grammar of a presentation that earns them.

01 The scheme and the marks

02 What examiners assess

03 Presenting: grammar and phrases

04 The thesis viva

How the Practical Examines You

Four sub-areas · 25 topics · the examination architecture

The practical is not a conversation about psychiatry. It is a marking instrument with a fixed arithmetic, a fixed sequence, and a small set of behaviours the people holding the pen have been told to look for. **Learn the instrument before you polish the content.**

What follows is that arithmetic, the order of the examination, the domains every format is scored on, the presentation grammar that earns marks, and the thesis defence. Each topic opens with the question an examiner would ask and the shape of the answer, then the detail, then what earns the marks.

HOW TO USE THESE NOTES

A **Rule** holds across formats. A **Trap** is where candidates lose marks; some are traps because the sources are silent, so the note says verify rather than handing you a figure. A **Hold this** block is the line to carry in, and the shaded cell in a grid is the discriminator.

The scheme and the marks

1 · The frame: what the practical head carries and what passes

SCHEME AND MARKS · TOPIC 01

What does the practical head carry, and what does a candidate have to score to pass it?

VIVA TABLES, AND ANY EXAMINER WHO OPENS ON THE SCHEME | 400 IN THE HEAD, PASS AT 200

THE SHAPE OF THE ANSWER

1 University law	Head of 400 marks, pass at 200, no grace marks
2 The split	300 clinical and practical, 20 of them thesis, plus 100 viva
3 The rival frame	The regulator says 280, 20 and 100; say which is which
4 The gates before it	Attendance, logbook, ethics and life support courses
5 The caveat	The split inside the 300 is departmental, not university law

Start with the arithmetic, because it decides how you spend your effort. The practical and the viva voce sit together as **one examination head**.

- **Key number.** The university ordinance puts that head at 400 marks: 300 for clinical and practical work, inside which 20 marks are the thesis evaluation by the external examiner, plus 100 marks of viva voce.
- **Key number.** Passing is an aggregate rule: 200 of 400, an aggregate of 50 per cent, and no grace marks.
- **Concept.** A candidate who fails under one head re-appears for both theory and practical, so the two are not separable.

Two descriptions of the same money circulate, and **an examiner may be holding either one**.

- **Key number.** The national regulator's notice describes it as clinical and practical 280, dissertation 20, and viva voce 100.

- **Trap.** Quoting one frame as though it were the only one. Be able to say which figure is university law and which is the regulator's frame.
- **Concept.** The internal division of the 300 marks is delegated to the subject syllabus, and none has been published for psychiatry, which is why departmental sheets differ.

WHAT FIXES IT	COMPONENT	MARKS	STATUS IN AN ARGUMENT
RGUHS 2025 post-graduate ordinance, section 10.7.6	Practical and clinical, thesis 20 marks included	300	**University law: quote this first**
RGUHS 2025 post-graduate ordinance, section 10.7.6	Viva voce	100	University law
RGUHS 2025 post-graduate ordinance, section 10.7.6	Head total, and the pass mark	400, pass at 200	**Aggregate of 50 per cent, no grace marks**
National regulator's public notice	Clinical and practical examination	280	Regulator's frame
National regulator's public notice	Dissertation, marked from outside the state	20	**Five per cent of clinical, practical and viva voce together**
National regulator's public notice	Viva voce tables	100	Regulator's frame
Departmental sheet, this cycle	Long case 120, psychiatry short case 60, neurology short case 60, OSCE 40, thesis 20	300	**Local split: confirm against your own circular**
2022 draft psychiatry curriculum, not notified in final form	Long case 100, each short case 50, OSCE 40 sitting inside the viva	200 plus 100	Superseded: do not quote as current

Three descriptions of one head. The first three rows hold up when an examiner presses.

The gates before the gate. Eligibility is checked before any of this arithmetic applies to you.

- **Key number.** Attendance of 80 per cent of working days, spelled out as 751 days out of 939 working days across a three-year course.
- **Key number.** A logbook kept digitally from the 2023 to 2024 batch onwards.
- **Key number.** Courses in ethics and in cardiac life support skills for those admitted from 2021 onwards.

RULE The pass is an **aggregate across the whole head**, 200 of 400, no grace marks. A strong long case does not rescue a silent viva table, and a weak short case is survivable if the tables and the thesis hold up.

INDIAN PRACTICE NOTE

Only the head total and the pass rule are university law. The split inside the 300 marks is departmental, and the sheet in use for your cycle governs how your marks are added.

HOLD THIS

400 marks in the head: 300 practical and clinical with 20 for the thesis inside it, plus 100 viva voce, passing at 200. The rest is local detail.

WHAT EARNS THE MARKS

- **The head total and the pass rule**: the two figures in this topic that are university law, so they hold when an examiner presses
- **Naming the rival frame**: saying which description is the regulator's and which the university's shows you have read both
- **The thesis sitting inside the 300**: a candidate who adds 20 to 300 has not read the ordinance
- **The departmental caveat**: flagging that the internal split is local protects you when the sheet in use differs

SEE ALSO 02 The sequence 06 The viva tables 20 The external examiner

2 · The shape of the examination, in the order it happens

SCHEME AND MARKS · TOPIC 02

Take me through the practical from case allocation to the last viva table.

ANY FORMAT: THE ORIENTATION QUESTION AN EXAMINER OPENS WITH

THE SHAPE OF THE ANSWER

1 The sequence	Allocation, long case, two short cases, circuit, thesis, tables
2 The long case clock	45 minutes to evaluate, then 15 to organise
3 The short case clock	25 minutes each, plus 5 to prepare
4 The circuit clock	5 to 10 minutes a station, rest every 30 to 40 minutes
5 Their standing	The timings come from a draft, so verify your own

The sequence is stable even where the timings are not, so **learn the order first and the clock second**.

- **Concept.** Cases are allocated early in the morning. You work up the long case unobserved, present and defend it at the bedside, do two observed short cases, walk an objective circuit, defend the dissertation, and finish at the viva tables.
- **Key number.** Not more than 8 postgraduates are examined in clinicals in a batch, which is why the clock is enforced.

The timings, and their standing. The written figures attach to the cases, not to the tables.

- **Key number.** The adult psychiatry long case allows 45 minutes for evaluation, covering history, mental state and relevant physical examination, then an additional 15 minutes to organise the presentation.
- **Key number.** Each short case allows 25 minutes plus an additional 5 minutes to prepare.
- **Concept.** Inside the long case window the examiners may interview the patient themselves, and during the viva may ask you to elicit a phenomenon at the bedside.

The circuit runs on **a shorter clock again**.

- **Key number.** Objective stations run at 5 to 10 minutes per station conventionally, or as little as 120 seconds in dense variants.
- **Concept.** Rest stations come every 30 to 40 minutes, or after the twelfth station in larger circuits, and exist so you can organise notes or finish an unfinished written station.

Fig 1.1 The examination as one track: case allocation, long case workup and bedside defence, psychiatry short case, neurology short case, objective circuit with its rest stations, dissertation viva, then the viva tables. The written timings attach to the first three boxes.

TRAP Treating the written timings as settled law. They come from a draft psychiatry curriculum that was not notified in final form, and are the only per-case timings in the university material; the later ordinance fixes marks and conduct, not timing.

TRAP: VERIFY BEFORE THE EXAM How long should the long case presentation itself run, and the viva on it? No university document states either. Get the expected length from your own examination circular or your unit, and rehearse to that.

HOLD THIS

45 plus 15 for the long case, 25 plus 5 for each short case, stations at 5 to 10 minutes or as little as 120 seconds. The sequence is fixed; the durations are local.

WHAT EARNS THE MARKS

- **The order, given without prompting**: the sequence is the part of this topic you can state with certainty
- **45 plus 15, and 25 plus 5**: the two clocks you are being timed against in the cases
- **Naming where a timing comes from**: the figures sit in a draft curriculum, and the later ordinance fixes marks and conduct rather than timing
- **A rehearsed presentation length**: the one duration no document supplies, so you bring your own and say so

SEE ALSO 01 The frame and the marks 03 The long case 05 The OSCE circuit

3 · The long case: what the allotted time buys you

SCHEME AND MARKS · TOPIC 03

You have the long case. What does the time buy you, and in what order do you present?

LONG CASE: BEDSIDE PRESENTATION AND THE VIVA ON IT | LONG CASE 120, LOCAL SHEET

THE SHAPE OF THE ANSWER

1 **Reliability**	Who gave the history, and how good is it
2 **Chronology**	Premorbid baseline to present, with treatment actually taken
3 **Exclusions, then findings**	What you ruled out, then what the mental state showed
4 **Risk**	Said inside the presentation, before anyone asks for it
5 **Formulation and plan**	Four Ps, defended diagnosis, then stepped management

The long case is the spine of the examination. It is the only station where you are alone with a patient long enough to build a whole account, and **the only one marked on synthesis**.

- **Key number.** The 45 minutes buy history, mental state and a relevant physical examination.
- **Concept.** The 15 minutes that follow buy structure, and are also when the examiners form their own impression of the patient.
- **Trap.** A presentation that contradicts what the examiners saw at the bedside costs you twice.

Spend the second window on **the order of the argument**, not on more data. The examiner climbs a predictable ladder, and every rung is a chance to answer the next question before it is asked.

1. **Who gave the history, and how good is it?** The informant, degree of contact, intellectual capability, secondary gain. Reliability stated up front licenses everything after it.
2. **Take me through the illness.** Premorbid baseline to present: onset, episodes, inter-episodic functioning, precipitants, and what treatment was actually taken.
3. **What have you excluded?** Organicity, substance causation, mood episodes in a primary psychosis, psychotic symptoms in a primary mood disorder.
4. **What did the mental state show?** Speech, affectivity, thought form and content, perception, higher mental functions.
5. **What is your risk assessment?** Suicide, self-harm, violence, with protective and aggravating factors, stated rather than waited for.
6. **Give me your formulation.** Predisposing, precipitating, perpetuating, protective, then the categorical diagnosis.
7. **Defend it.** Contrast it with plausible differentials by course and hallmark psychopathology, not by assertion.
8. **What will you do?** Acute, continuation and maintenance: pharmacotherapy, psychotherapy, rehabilitation, and the legal position under the Mental Healthcare Act, 2017.

MNEMONIC

WHO · WHAT · WHAT ELSE · SAW · FEAR · WHY · WHY NOT · NOW

The eight rungs as eight spoken beats. *Who* told me, *what* happened over time, *what else* I ruled out, what I *saw*, what I *fear*, *why* this diagnosis, *why not* the alternatives, what happens *now*. Say the eight without notes and you can present any long case.

RULE Risk is not a section at the end. Examiners commonly probe it as a separate question, so state it inside the presentation, with protective and aggravating factors, before anyone asks.

HOLD THIS

Reliability, chronology, exclusions, mental state, risk, formulation, differential, management. Eight rungs in that order, and the examiner does not have to prompt you.

WHAT EARNS THE MARKS

- **Reliability stated first**: it licenses every statement that follows it
- **A risk sentence you volunteer**: examiners commonly probe risk separately, so saying it early removes a question
- **A differential argued by course and hallmark psychopathology**: contrast, rather than assertion
- **The legal basis inside the plan**: management that names its authority under the Mental Healthcare Act, 2017

SEE ALSO 10 Risk as a competency 15 History in examiner order 17 The formulation 18 Management by acuity

4 · The two short cases: psychiatry and neurology

SCHEME AND MARKS · TOPIC 04

How does a short case differ from a long case, and how do you finish the neurology one?

PSYCHIATRY SHORT CASE AND NEUROLOGY SHORT CASE, BOTH OBSERVED

SHORT CASES 60 EACH, LOCAL SHEET

THE SHAPE OF THE ANSWER

1 **The frame**	One long case and two short cases, psychiatry and neurology
2 **The clock**	25 minutes each, plus 5 to prepare
3 **The psychiatry case**	One syndrome to depth, not a compressed long case
4 **The neurology case**	Cranial nerves through to sensory, in a sequence you can finish
5 **The finish**	Localise the lesion, then give an aetiological differential

The ordinance examines a candidate in a clinical science subject on one long case and two short cases, and here **the marks sit on elicitation**, because you are watched.

- **Key number.** The psychiatry curriculum splits those into one psychiatry and one neurology short case, at 25 minutes plus 5 minutes to prepare.
- **Concept.** What is marked is how you get the sign out, and whether you know what it means.

The psychiatry short case is a focused syndromic presentation: obsessive-compulsive disorder, a substance dependence syndrome, a conversion disorder, or a child and adolescent presentation such as an intellectual developmental disorder or attention-deficit hyperactivity disorder. One syndrome to depth, not a compressed long case.

The neurology short case verifies competence in neuropsychiatric assessment, and it has a ladder of its own.

1. **Cranial nerves**: fundoscopy, extraocular movements, facial symmetry.
2. **Motor system**: tone, separating spasticity from rigidity and paratonia, power, deep tendon reflexes, plantar response.
3. **Involuntary movements**: resting against postural and action tremor, tics, chorea, and the drug-induced disorders, acute dystonia, akathisia, tardive dyskinesia.
4. **Cerebellar signs and gait**: ataxia, dysmetria, dysdiadochokinesia, tandem gait, sensory ataxia.
5. **Sensory system**: pain, touch, vibration, joint position sense, and cortical signs such as graphaesthesia and stereognosis where localisation needs them.
6. **Localise, then investigate.** Map the lesion to cortex, basal ganglia, brainstem, cord or peripheral nerve before proposing imaging.

TRAP Presenting a neurology short case as a list of signs with no localisation. Upper against lower motor neuron, cortical against subcortical, then an aetiological differential: that synthesis is the discriminator. Skipping the sensory examination is the commonest omission.

TRAP: VERIFY BEFORE THE EXAM The expected depth of the sensory examination in this station is not set out in the library sources. Rehearse a sequence you can complete inside the station clock, and check the expectation with your own unit.

CLINICAL ANCHOR

A synthetic case: a man on long-term antipsychotic medication, brought with restlessness he cannot sit through. The station is won by separating akathisia from agitation and from a resting tremor, naming the drug-induced category, then saying what you would change and monitor. It is lost by listing normal cranial nerves for most of your minutes.

HOLD THIS

Short cases are observed, so technique is the mark. Finish every neurology short case with a localisation and a differential, out loud.

WHAT EARNS THE MARKS

- **A visible technique**: the station marks how you elicit, so demonstrate rather than describe
- **A completed sensory examination**: the commonest omission in this station, and the cheapest to avoid
- **A localisation said out loud**: upper against lower motor neuron, cortical against subcortical
- **A named drug-induced category**: acute dystonia, akathisia or tardive dyskinesia, separated from agitation and from tremor

SEE ALSO 03 The long case 05 The OSCE circuit 09 The behaviour grid

5 · The OSCE circuit: stations, taxonomy and the clock

SCHEME AND MARKS · TOPIC 05

Why does the practical include an objective circuit, and how many stations make it reliable?

OSCE CIRCUIT: OBSERVED, UNOBSERVED AND COUPLET STATIONS | OSCE 40, LOCAL SHEET

THE SHAPE OF THE ANSWER

1 **The rationale**	Wide sampling at the shows how and does levels
2 **The counts**	12 to 30 stations, with 20 as the practical ceiling
3 **The reliability**	14 to 18 stations of 5 to 10 minutes each
4 **The station types**	Procedure, question, couplet, rest
5 **Who plays the patient**	Real patients for signs, actors for interpersonal work

The objective structured clinical examination was introduced by Harden in 1975, and **earns its place by sampling widely**.

- **Concept.** It targets the "shows how" and "does" levels of Miller's pyramid rather than recall.
- **Concept.** Competency based medical education guidelines have formalised these stations into regular assessment in India.

The numbers worth knowing. Three of them answer most questions asked about the circuit.

- **Key number.** Circuits run from 12 to 30 stations, with the recommendation that they not exceed 20 so the examination completes within 60 to 150 minutes.
- **Key number.** Psychometric evaluation indicates 14 to 18 stations of 5 to 10 minutes each give adequate reliability.
- **Key number.** Some setups use high-density circuits, for example 25 stations at 120 seconds each, with rest stations every 30 to 40 minutes or after the twelfth station.

STATION TYPE	WHAT HAPPENS	WHAT IS BEING MARKED
Procedure, observed	You perform a defined task: eliciting suicidal intent, counselling a caregiver, explaining electroconvulsive therapy, while an examiner marks a checklist	**Technique and manner: do the task, do not describe it**
Question, unobserved	You read clinical data, a blood report, a tomogram, a radiograph or an electroencephalogram, and write a concise deduction	**The written answer only: write the deduction, not an essay**
Couplet	A procedure station followed immediately by a linked unobserved question station on the same scenario	**Synthesis with cueing minimised: the second half tests the first**
Rest	Unassessed station inside the circuit	**Nothing: finish an incomplete written station**

The four station types. The couplet is the one candidates underestimate.

Who plays the patient follows from **what the station is testing**.

- **Concept.** Real patients suit stations built on stable physical or neurological signs, at the cost of fatigue and fluctuating co-operation.
- **Concept.** Standardised patients, or trained actors, give reproducible and safe simulations where the construct is interpersonal: breaking bad news, managing a behavioural crisis.

HOLD THIS

14 to 18 stations of 5 to 10 minutes is the reliability sweet spot, 20 the practical ceiling.
Observed stations mark technique; unobserved ones mark the written line.

WHAT EARNS THE MARKS

- **Doing the task at a procedure station**: technique is the mark, so perform it rather than describe it
- **A written deduction, not an essay**: the unobserved station marks the line you wrote and nothing else
- **The reliability figures**: 14 to 18 stations of 5 to 10 minutes, with 20 as the ceiling
- **Recognising a couplet**: the second half tests the first, so both halves answer one scenario

SEE ALSO 02 The sequence 08 How a station is scored 09 The behaviour grid

6 · The viva tables: imaging, drugs, instruments, law

SCHEME AND MARKS · TOPIC 06

The viva voce carries 100 marks across tables. What is each table waiting to hear?

VIVA TABLES: IMAGING, INSTRUMENTS, DRUGS, EMERGENCIES, LAW | VIVA VOCE 100 MARKS

THE SHAPE OF THE ANSWER

1 **The structure**	100 marks across distinct tables, six in the local sheet
2 **Identify**	Name the abnormality, or the object in your hand
3 **Consequence**	Say what it changes in management, in the same breath
4 **The legal table**	Name the section that authorises what you just proposed

The viva voce carries **100 marks**, organised across distinct physical tables rather than as one conversation; the departmental sheet for this cycle spreads them over six tables.

- **Concept.** Each table has one thing the examiner is waiting to hear, and the rest of the table follows from it.

TABLE	WHAT IS PUT IN FRONT OF YOU	THE ONE THING THEY LISTEN FOR
Imaging and investigations	Computed tomography, magnetic resonance sequences, electroencephalograms, cerebrospinal fluid reports, drug levels such as lithium or valproate	**Name the abnormality, then what it changes in management**
Instruments and neuromodulation	The modified electroconvulsive therapy machine, stimulus dosing, electrodes, airway adjuncts, monitoring tools	**Indications and contraindications before parameters**
Psychopharmacology	Mechanisms, dosing schedules and adverse reactions of the drug in your hand	**The adverse effect that changes the drug, and its monitoring**
Emergency protocols	Neuroleptic malignant syndrome, serotonin syndrome, lithium toxicity, status epilepticus, acute catatonia	**The first three actions in order, before mechanisms**
Forensic psychiatry and legislation	The Mental Healthcare Act, 2017 in operation: advance directives, the nominated representative, supported admission, capacity assessment	**Which section authorises the thing you just proposed**

Five different questions in the same uniform. The shaded cell buys you the rest of the table.

- **RULE** At a table, lead with the consequence. An identification plus "and that changes my management by ..." scores above a longer answer that describes the object and stops.

HOLD THIS

100 marks of viva, across six tables in the local sheet. Each is won by tying the object in front of you to a decision.

WHAT EARNS THE MARKS

- **Identification plus consequence**: an object named, then tied to a decision you would take
- **Indications before parameters**: at the instrument table, what it is for comes before how it is set
- **The first three actions, in order**: at the emergency table, sequence beats mechanism
- **The authorising provision**: at the legal table, the section arrives with the action

SEE ALSO 01 The frame and the marks 10 Risk as a competency 18 Management by acuity

What examiners assess

7 · The five domains every format is marked on

WHAT EXAMINERS ASSESS · TOPIC 07

Whatever the station is about, what are the examiners actually marking?

ALL FOUR FORMATS: LONG CASE, SHORT CASES, OSCE, VIVA TABLES

50, 30 AND 20 PER CENT IN THE WORKED STATION

THE SHAPE OF THE ANSWER

1	Attitude and conduct	Respect, empathy, sensitivity to culture and distress
2	Consultation management	A logical sequence with the time managed
3	Communication	Listening, reactive questioning, no jargon
4	Assessment and formulation	Elicitation, examination, differentials, formulation
5	Management and risk	A safe, feasible plan with risk mitigation

One question runs under all four formats: has this trainee reached independent clinical competence? The formats differ in what they sample, and **examiners read every one of them through the same five domains.**

1. **Professional attitude and conduct.** Respect, empathy, non-judgemental regard, sensitivity to perspective, culture and distress.
2. **Consultation management.** A logical sequence, fluent direction, core issues prioritised, time respected.
3. **Communication skills.** Active listening, reactive questioning, no jargon, responsiveness to verbal and non-verbal cues.
4. **Clinical assessment and formulation.** Accurate elicitation of psychopathology, physical and cognitive examination, differentials, a structured formulation.
5. **Clinical management and risk planning.** A safe, feasible biopsychosocial plan, with immediate and longer-term risk mitigation.

Weighting follows the construct of the station. Find the construct, then spend your minutes in proportion to it.

- **Key number.** In a structured station on alcohol dependence, eliciting the features of physical and psychological dependence carries 50 per cent of the marks, the biopsychosocial consequences 30 per cent, and interview and communication skills 20 per cent.

KEY IDEA

Content knowledge is assumed by this stage. What is sampled is whether it is organised, safe and usable in front of a patient who is watching, which is why the two neglected domains, attitude and consultation management, most often separate a pass from a borderline fail.

HOLD THIS

Attitude, consultation management, communication, assessment and formulation, management and risk. Five domains, every format, every station.

WHAT EARNS THE MARKS

- **The two neglected domains**: attitude and consultation management most often separate a pass from a borderline fail
- **Minutes spent in proportion**: the construct of the station decides where its marks are
- **A plan given as a plan**: management and risk are marked together, so the risk mitigation sits inside the plan
- **Organisation over volume**: content knowledge is assumed by this stage, so what is sampled is whether it is usable

SEE ALSO 08 How a station is scored 09 The behaviour grid 11 Communication and the role player

8 · How a station is actually scored

WHAT EXAMINERS ASSESS · TOPIC 08

How is a station actually scored, and where is the pass cut set?

OSCE CIRCUIT, AND ANY STRUCTURED STATION WITH A CHECKLIST

THE SHAPE OF THE ANSWER

1 Domain scores	Three to five domains, each on a 5 point scale
2 Global judgement	One separate judgement on six levels
3 The regression	Domain scores regressed on globals set the borderline cut
4 The second gate	Stations passed counts as well as marks scored
5 The veto	Two or more severe fails go to the examinations committee

Objective examinations use the Borderline Regression Method for standard setting, and at each station **an examiner awards two separate things**.

- **Key number.** An analytic domain score on a 5 point scale from 1, poor, to 5, excellent, across 3 to 5 domains.
- **Key number.** One global judgement on six levels: Excellent Pass, Pass, Borderline Pass, Borderline Fail, Fail, or Severe Fail.

The cut is then **derived rather than declared**.

- **Concept.** Regressing domain scores on the global judgements sets the cut for the borderline candidate, adjusted upward by the standard error of measurement to protect patient care.
- **Key number.** In a 16 station examination a candidate must meet the aggregate cut and pass a minimum of 12 stations.
- **Key number.** Borderline candidates should pass at least 8 of the 10 basic skills stations and 4 of the 6 management stations.

The architecture, in its worked form. The 16 station examination splits into 2 circuits in one sitting.

- **Key number.** The stations divide as 5 on history taking including risk, 5 on examination including physical, mental state and capacity, and 6 on management.
- **Key number.** Circuit 1 carries 6 management stations, 1 examination and 1 history station, with 4 minutes of reading and 7 minutes of consultation.
- **Key number.** Circuit 2 carries 4 examination and 4 history stations, with 90 seconds of reading and 7 minutes of consultation.
- **Key number.** A warning bell sounds at 6 minutes and the final bell at 7 minutes.

RULE The global judgement is not a summary of the domain marks; it is a separate act of judgement, and it sets the standard. A candidate who is technically complete but unsafe or discourteous can carry domain marks and still sink the station.

TRAP Playing for the aggregate. Two or more Severe Fail marks trigger review by the examinations committee and can fail the whole examination regardless of marks or stations passed.

HOLD THIS

Domain scores on a 5 point scale plus one global judgement on six levels; regressing one on the other sets the cut. Stations passed matters as well as marks scored.

WHAT EARNS THE MARKS

- **Both marks, named separately**: the domain score and the global judgement are two different acts of judgement
- **The direction of the regression**: the globals set the standard, and the domain scores are regressed on them
- **Stations passed**: the second gate, which an aggregate on its own does not satisfy
- **Safety and courtesy**: a station can sink on the global judgement while the domain marks look complete

SEE ALSO 05 The OSCE circuit 07 The five domains 09 The behaviour grid

9 · Pass, borderline fail, severe fail: the behaviour grid

WHAT EXAMINERS ASSESS · TOPIC 09

What separates a pass from a borderline fail, and a borderline fail from a severe fail?

ALL FORMATS: THE BEHAVIOURAL ANCHORS EXAMINERS ARE GIVEN

THE SHAPE OF THE ANSWER

1 Pass	Organised, courteous, systematic and safe
2 Borderline fail	The content is there and the organisation is not

3 Severe fail	Safety or respect has been breached
4 The fix	A spine for organisation, a risk sentence for safety

Examiners are given **behavioural anchors, not adjectives.** Read the third column first, because that is the column that ends examinations.

DOMAIN	PASS BEHAVIOUR	BORDERLINE FAIL BEHAVIOUR	SEVERE FAIL BEHAVIOUR
Attitude and conduct	Listens actively; warmth and respect; explores health beliefs; validates distress	Mild detachment; rote script; distress addressed superficially	**Rudeness, arrogance, condescension or flippancy; dignity breached**
Consultation management	Open to closed funnel; time managed fluently; core complaints prioritised	Disorganised sequencing; rambling consumes time; critical tasks rushed at the bell	**Disjointed; unlinked questions; time gone with no presenting complaint established**
Communication	Clear speech; jargon avoided; understanding checked; responsive to cues	Occasional jargon; rehearsed phrases; subtle cues missed	**Incomprehensible jargon; speaking over the patient; distress ignored**
Assessment and knowledge	Psychopathology explored systematically; hand hygiene before and after contact; diagnosis justified	Minor criteria omitted; unfocused medical history; cursory physical examination	**Gross psychopathology missed; technical incompetence; unsafe diagnosis**
Management and risk	Safe, feasible biopsychosocial plan; risk assessed systematically; side effects explained	Generic plan; incomplete risk enquiry; vague monitoring	**Lethal drug combinations; active suicidal or violent intent ignored; high-risk discharge with safety unaddressed**

Five domains at three levels. Borderline is about organisation; severe is about safety and respect.

- **Concept.** Borderline behaviour reads as disorganisation: rambling that consumes time, critical tasks rushed at the bell, minor criteria omitted, a cursory physical examination.
- **Trap.** Severe behaviour reads as harm or contempt: a lethal combination prescribed, active intent ignored, a high-risk discharge left unaddressed, dignity breached.

HOLD THIS

Borderline is a failure of organisation; severe is a failure of safety or respect. Fix organisation with a spine, and safety by saying the risk sentence out loud.

WHAT EARNS THE MARKS

- **An audible structure**: organisation is exactly what the borderline column is missing
- **Hand hygiene before and after contact**: a marked behaviour inside the assessment domain, not an aside
- **A completed risk enquiry**: incomplete enquiry sits in the borderline column, ignored intent in the severe one
- **Respect that survives pressure**: flippancy and condescension are scored rather than overlooked

SEE ALSO 07 The five domains 08 How a station is scored 10 Risk as a competency

10 · Risk assessment as an examined competency

WHAT EXAMINERS ASSESS · TOPIC 10

Give me your risk assessment, and the legal basis for what you would do about it.

LONG CASE VIVA, OSCE RISK STATIONS, THE FORENSIC AND LEGISLATION TABLE

THE SHAPE OF THE ANSWER

1 **The frame**	Nature, severity, frequency, likelihood, imminence
2 **The enquiry**	Ideation, intent, plan, means, preparatory acts
3 **The modifiers**	Modifiable factors set against protective ones
4 **Uncertainty**	Name the ambiguity and attach a contingency plan
5 **The statute**	The section that authorises what you propose

Risk is assessed as an **active clinical competency**, not as a tick-box chore.

- **Concept.** Examiners expect a systematic evaluation across five parameters: the nature, severity, frequency, likelihood and imminence of the risk.

For suicide and deliberate self-harm, **specific enquiry fills that frame**.

- **Concept.** Current ideation, intent and planning; the lethality of past attempts, precautions taken against discovery, and regret over survival.
- **Concept.** Access to lethal means, and preparatory acts.
- **Concept.** Modifiable factors, substance use, severe anxiety, insomnia and agitation, set against protective ones, social support, dependent children, an alliance.
- **Concept.** Where the picture is ambiguous, examiners reward naming the uncertainty and attaching a contingency plan to it.

The statutory frame comes next, and marks are won cheaply here. Risk management runs through the Mental Healthcare Act, 2017, with disability entitlements under the Rights of Persons with Disabilities Act, 2016.

- **Concept.** Learn each provision as a pair: what it authorises, and the detail that tests whether you have read it.

PROVISION	WHAT IT AUTHORISES OR REQUIRES	THE DISCRIMINATOR
Capacity, Section 4	Understanding information relevant to a decision on treatment, admission or personal assistance; appreciating its reasonably foreseeable consequences; communicating the decision by any means	**A decision others perceive as wrong does not by itself mean the person lacks capacity**
Independent admission, Sections 85 and 86	For a person with capacity, or needing minimal support, to make treatment decisions; admissions are independent as far as possible	**No treatment without informed consent, and the admission needs neither the consent nor the presence of a nominated representative**

PROVISION	WHAT IT AUTHORISES OR REQUIRES	THE DISCRIMINATOR
Minor, Section 87	On the nominated representative's application, with the opinion of two psychiatrists or the specified combination of professionals	**The application comes from the nominated representative, not the minor**
Supported admission, Section 89	Nominated representative applies; two professionals, one a psychiatrist, examine independently on the day of admission or in the preceding seven days; recent or current self-harm, violence or inability to self-care; certified the least restrictive option	**Capped at thirty days, and the person has to be ineligible as an independent patient**
Reporting, Section 89(9)	A woman or a minor is reported to the concerned Board within three days; any other admission within seven days	**The shorter clock applies to a woman or a minor**
Beyond thirty days, Section 90	Two psychiatrists examine independently in the preceding seven days; the Board permits or discharges within twenty-one days; ninety days, then one hundred and twenty, then one hundred and eighty each time	**The threshold changes from "recently" to "consistently over time"**
Emergency treatment, Section 94	Where immediately necessary to prevent death, irreversible harm, serious harm to self or others, or serious damage to property; seventy-two hours or until assessment, extendable to seven days in a declared disaster	**Electroconvulsive therapy is outside Section 94**
Prohibited procedures, Section 95	Unmodified electroconvulsive therapy, the same therapy for a minor except with guardian consent and the Board's prior permission, sterilisation as treatment, and chaining	**Chaining is prohibited in any manner, with no exception to argue about**
Restraint and seclusion, Section 97	Seclusion prohibited; restraint only as the only means to prevent imminent harm, authorised by the psychiatrist in charge, recorded immediately, nominated representative informed within twenty-four hours	**Not as punishment or deterrent, and not for a shortage of staff**

PROVISION	WHAT IT AUTHORISES OR REQUIRES	THE DISCRIMINATOR
Community living and discharge, Sections 19 and 98	The right not to be segregated from society, with community establishments supported; a discharge plan made with the person, the nominated representative, the care-giver and the psychiatrist taking over	**Continued stay merely because the family will not accept the person home is barred**
Disability certification, Rights of Persons with Disabilities Act, 2016	Benchmark disability is not less than forty per cent of a specified disability not defined in measurable terms; the certifying authority with jurisdiction assesses against Central Government guidelines	**Forty per cent applies to disabilities not defined in measurable terms, and appeal lies to the State Government's appellate authority**

The right-hand column separates a candidate who has read the Act from one who has not.

RULE Name the section that authorises the action, in the same breath as the action. "I would admit under supported admission, Section 89, because ..." beats a paragraph of reasoning with the legal basis implicit.

HOLD THIS

Nature, severity, frequency, likelihood, imminence, then the section that authorises what you propose. Uncertainty named with a contingency plan scores; certainty invented does not.

WHAT EARNS THE MARKS

- **The five parameters, in order**: nature, severity, frequency, likelihood, imminence, said as a frame
- **Protective factors named**: a risk statement carrying only aggravating factors reads as half finished
- **A contingency plan for ambiguity**: uncertainty named scores, and certainty invented does not
- **Section with action**: the provision spoken in the same breath as the decision it authorises

SEE ALSO 03 The long case 09 The behaviour grid 18 Management by acuity

11 · Communication, empathy and the role player

WHAT EXAMINERS ASSESS · TOPIC 11

The station uses a trained role player. What is marked, and what closes a patient down?

OSCE COMMUNICATION STATIONS, AND EVERY OBSERVED CASE

THE SHAPE OF THE ANSWER

1 **The premise**	Communication is scored from behaviour, not from a claim
2 **The opening**	Open and collaborative, then an open to closed funnel
3 **Signposting**	Flag the move into sensitive territory before you make it
4 **Summarising**	Confirms accuracy, and shows the examiner your structure
5 **Pace**	Compress the recitation, not the safety enquiry

Communication is scored from live behaviour, not from a claim about your style. **A professional role player adapts to how you behave.**

- **Concept.** An abrupt, dismissive or interrogative manner triggers withdrawal, hostility, or the withholding of diagnostic information.
- **Trap.** Close the patient down and the information you need disappears, and the marks go with it.

The moves are recognisable, and **each one is a marked behaviour**.

- **Concept.** An open, collaborative opening, then an open-to-closed funnel that lets the narrative emerge before details are clarified.
- **Concept.** Signposting into sensitive territory, from depressive symptoms into suicidal intent.
- **Concept.** Periodic summarising, which confirms accuracy and structures the interview for the examiner as well as for the patient.
- **Concept.** Plain language, and a pace that still leaves room for summarising and for the patient's questions.
- **Trap.** When the clock tightens, compress the recitation and leave the safety enquiry intact.

TRAP The checklist interrogation: closed questions fired in succession without listening or acknowledging distress. It fragments the history, alienates the patient, and lands a borderline fail even when the content is complete.

HOLD THIS

Open to closed, signpost, summarise, plain language. The role player responds to your manner, so your manner is part of the data you are collecting.

WHAT EARNS THE MARKS

- **An opening the patient can work with**: the role player's co-operation is the resource you are marked on
- **Signposting before a sensitive question**: it earns the communication domain and protects the safety enquiry
- **A summary said out loud**: it demonstrates accuracy to the patient and structure to the examiner
- **Distress acknowledged**: ignoring it is anchored in the severe column, not the borderline one

SEE ALSO 07 The five domains 09 The behaviour grid 15 History in examiner order

12 · Why the long case survives, and what the evidence says

WHAT EXAMINERS ASSESS · TOPIC 12

The long case is criticised as unreliable. Why is it still in the examination?

VIVA ON ASSESSMENT METHODS, AND THE EXAMINER WHO ASKS ABOUT VALIDITY

THE SHAPE OF THE ANSWER

1 **The claim for it**	The one format that assesses holistic competence
2 **The weaknesses**	Examiner agreement at 25 per cent, and case specificity
3 **The fix**	Observation lifts reliability, with quotable coefficients
4 **The scoring**	Checklists for discrete steps, global ratings for manner
5 **The standard**	Criterion referencing at a fixed 50 per cent

The long case entered clinical examinations in 1842, and its value is specific: **it is the one format that assesses holistic competence**.

- **Key number.** Traditionally it places a candidate unobserved with a patient for about 60 minutes, then a viva of about 25 minutes on history, examination, formulation and management.

Its psychometric weaknesses are equally documented. Three of them come up at this table.

- **Key number.** Two examiners of the same encounter correlate at only 25 per cent.
- **Concept.** Performance is case-specific, so competence on one condition does not predict another.
- **Concept.** Because the interview is unobserved, communication and examination skills cannot be validly assessed from the presentation.
- **Key number.** A reliability coefficient of 0.8 is estimated to need 5 to 6 long cases of 85 minutes each, or 10 observed by 2 examiners.

Observation is the fix, and the numbers are quotable.

- **Key number.** Wass and Jolly showed that observing history taking gives inter-rater reliability of 0.72 for checklists and 0.71 for global scores, against 0.38 and 0.60 when only the presentation is marked.
- **Key number.** The Objective Structured Long Examination Record is a 10 item instrument.
- **Key number.** The Direct Observation Clinical Encounter Examination reached a generalisability coefficient of 0.84 for four cases and two examiners, with 2 to 3 examiners observing across 4 to 6 patients, a count describing the method rather than the coefficient.

SCORING METHOD	STRENGTH	LIMITATION	WHERE IT BELONGS
Standardised checklist	Objectivity, structured marking, strong agreement between examiners	**Trivialises complex tasks and fragments holistic performance**	Psychomotor steps, neurological examination sequences, structured rating scales
Global rating scale	Captures non-verbal communication, empathy, coherence, clinical judgement	**Greater variation between examiners, and depends on examiner expertise**	Affective domain, psychotherapy role-play, diagnostic formulation

The answer is both: checklists for discrete physical steps, global ratings for communication and interview technique.

Standard setting sits on top of this. Norm-referenced models such as the Angoff method or the borderline group approach exist, but Indian postgraduate clinical examinations generally use **criterion referencing with a fixed pass standard of 50 per cent**, so you are benchmarked against criteria rather than against your batch.

RULE Observation and presentation measure different skills. That sentence answers most viva questions on why the practical pairs an unobserved long case with observed short cases, stations and tables.

HOLD THIS

Two examiners of one unobserved encounter agree at 25 per cent; observing history taking lifts reliability to 0.72 for checklists and 0.71 for global scores. Hence the mixed practical.

WHAT EARNS THE MARKS

- **One sentence on validity**: observation and presentation measure different skills, which answers most of this table
- **Two quotable coefficients**: 0.72 for checklists and 0.71 for global scores once history taking is observed
- **Case specificity**: it explains why the practical samples several formats rather than one long one
- **Criterion referencing**: you are benchmarked against a fixed standard, not against the people beside you

SEE ALSO 03 The long case 05 The OSCE circuit 08 How a station is scored

Presenting: grammar and phrases

13 · Three formats, and which one they asked for

PRESENTING GRAMMAR · TOPIC 13

Present the case. Which of the three structures has that verb just asked for?

LONG CASE BEDSIDE PRESENTATION, AND ANY TABLE THAT SAYS FORMULATE

THE SHAPE OF THE ANSWER

1 The five steps	Detail, summary, formulation, diagnosis, plan
2 Full presentation	All five steps: the default for present the case
3 Case summary	Steps two to five, when they have already seen the patient
4 Case formulation	Steps three to five, with the narrative dropped
5 The cue	Listen for the verb, then choose the entry point

Most marks lost in the long case are lost to the wrong format, not the wrong facts.

- **Concept.** Academic psychiatry recognises three structures built from the same five steps: detailed clinical information, an optional diagnostic summary, the formulation, the diagnosis with its differential, and the plan of management.
- **Concept.** They differ only in **where they start**.

FORMAT	STEPS IT CONTAINS	WHEN IT IS THE RIGHT ANSWER
Full presentation, the 4DP format	All five: history, examination and mental state, then summary, formulation, diagnosis and plan	**"Present the case": the default in a university long case**
Case summary, the 3DP format	Steps two to five: a descriptive account, then synthesis and management	**When the examiner has already seen the patient**
Case formulation, the 2DP format	Steps three to five: analytical synthesis, diagnosis and management only	**"Give me your formulation": the analytic answer, narrative dropped**

Three formats, one set of steps. The examiner's verb tells you where to start.

In the full format, **step one carries the detail**: the history, the physical examination and the detailed mental state.

- **Concept.** Step two, the summary, becomes optional once that detail has been spoken.

- **Concept.** Step three is the formulation, step four the diagnosis argued with points for and against, step five the plan.

- **RULE** Listen for the verb. "Present" invites the full structure; "formulate" invites the analytic one. The wrong format wastes the minutes you needed for the argument, and reads as not listening.

HOLD THIS

Five steps, three entry points. The examiner's verb chooses your entry point; you choose nothing else about the structure.

WHAT EARNS THE MARKS

- **The verb, acted on**: present invites the full structure, and formulate invites the analytic one
- **An entry point, not a new structure**: the steps keep their order whichever one you start at
- **A summary dropped once the detail is spoken**: it stops you saying the same thing twice
- **Minutes kept for the argument**: the wrong format spends them on narrative you did not need

SEE ALSO 14 Summary versus formulation 15 History in examiner order 17 The formulation

14 · Summary versus formulation: two languages

PRESENTING GRAMMAR · TOPIC 14

What is the difference between a diagnostic summary and a diagnostic formulation?

LONG CASE VIVA, AND THE FORMULATION ASKED FOR AT THE BEDSIDE

THE SHAPE OF THE ANSWER

1 Audience	Summary for a lay listener, formulation for colleagues
2 Language	Descriptive and impartial against technical and selective
3 Epistemic claim	An account of the data against a clinical opinion
4 Ordering	Chronological against aetiology before phenomenology
5 Size	About 5 minutes and 10 to 15 sentences for the formulation

A diagnostic summary and a diagnostic formulation are not long and short versions of one thing. They have **different audiences, vocabularies and epistemic claims**, and confusing them is the commonest structural error in a postgraduate presentation.

PARAMETER	DIAGNOSTIC SUMMARY	DIAGNOSTIC FORMULATION
Audience and goal	Communicates data to a lay listener; useful for psychoeducation with patient and family	**Precise communication with professional colleagues and examiners**
Language	Layman's terms, descriptive and impartial	**Technical psychopathological terminology and diagnostic criteria**
Epistemic claim	A descriptive account of the data: objective, impartial, thorough	**A clinical opinion: the analytical weighting of conflicting evidence**

PARAMETER	DIAGNOSTIC SUMMARY	DIAGNOSTIC FORMULATION
Symptom scope	Includes positives, minor and transient symptoms, and negative findings	**Highly selective: salient, discriminatory phenomena only**
Ordering	Chronological: sociodemographics, complaints, past, family, personal, personality, physical, mental state.	**Re-ordered: demographics, then aetiological background (family, past, personality), then presenting psychopathology**
Handling of repetition	Describes mental state, then the psychopathological impression	**Condenses by stating that the mental state concurred with the history of presenting illness**

Six parameters, two languages. The right-hand column is what a postgraduate viva marks.

Size and pace are prescribed. Both have a stated length, and neither is a page count you can pad.

- **Key number.** A formulation runs about 5 minutes spoken and not more than one side of A4 typed, roughly 10 to 15 sentences; a published model runs to 131 words.
- **Key number.** A summary runs not more than 10 minutes and about two sides; its published model runs to 250 words.
- **Key number.** Written records are typically under 10 pages in size 10 font, and page volume does not correlate with marks.

TRAP Reciting a chronological summary of raw biographical detail when a formulation was asked for. A high-scoring formulation is a synthesised argument of about 10 to 15 sentences in technical language, not a compressed biography.

HOLD THIS

Formulation: about 5 minutes, one side of A4, 10 to 15 sentences, technical, re-ordered so aetiology precedes phenomenology. Summary: up to 10 minutes, lay language, chronological.

WHAT EARNS THE MARKS

- **Re-ordering for the formulation**: demographics, then aetiological background, then the presenting psychopathology
- **Selective phenomena**: salient and discriminatory findings, with the minor and transient ones left out
- **The concurrence sentence**: the mental state agreed with the history of presenting illness, said once and moved on from
- **A length you have rehearsed**: about 5 minutes and 10 to 15 sentences, timed before the examination

SEE ALSO 13 Three formats 15 History in examiner order 17 The formulation

15 · History of presenting illness, in examiner order

PRESENTING GRAMMAR · TOPIC 15

Take me through the history of presenting illness.

LONG CASE PRESENTATION, AND OSCE HISTORY TAKING STATIONS

THE SHAPE OF THE ANSWER

1 The opener	One sentence naming the core challenge
2 Demographics	Age, gender, occupation, culture, marital status, living arrangements
3 The illness	Phenomenology with chronology, cluster by cluster
4 Context	Relative negatives, attribution, impact, coping, neurovegetative status
5 Safety	Risk to self and others, with the high-risk populations named

Open with one sentence that orients the examiner to the core diagnostic, management or safety challenge.

- **Concept.** Follow it with demographics in one or two sentences: age, gender, occupation, cultural background, marital status, living arrangements.
- **Concept.** Then the illness, as an argument rather than as a list.

1. **Phenomenological precision.** The patient's own words, selectively quoted, with the technical term attached immediately.
2. **Chronological qualification.** Onset, duration, course, precipitants, severity, per symptom cluster rather than for the illness as a whole.
3. **Relative negatives.** Where psychosis is suspected, report the common delusions, persecutory, of reference, of mind reading, and the hallucinations, pairing each positive with the negative that matters. If hallucinations are present, say whether they issue commands.
4. **Attribution and cultural context.** The patient's own explanation: it drives engagement and the alliance, and it is often the next question.
5. **Illness impact.** Disability and handicap: employment lost, education disrupted, social withdrawal.
6. **Coping.** How the distress has been managed, substance use against seeking support, and whether that is adaptive or hazardous.
7. **Neurovegetative status.** Sleep, appetite, energy, concentration, hedonic capacity, libido, recorded rather than inferred.
8. **Safety stratification.** Risk to self, ideation, intent, plan, preparatory acts; risk to others, homicidal intent, named targets, persecutory delusions involving specific people; self-neglect. Name the high-risk populations, for example a woman with puerperal psychosis, where the risk of infanticide reaches 5 per cent, or a young man recently discharged after a first episode of schizophrenia.

CLINICAL ANCHOR

A synthetic opener that does the work of a paragraph: "This is a woman in her late twenties, a schoolteacher, with a six-week first episode of a persecutory psychosis, in whom the issues are excluding an organic cause and managing an unaddressed risk to her infant." The examiner now knows the diagnosis you favour, the differential you fear and the risk you have considered, before the history begins.

RULE Pair every positive with the negative that discriminates. Hallucinations with no statement about command quality, or a delusion without the forms you sought and did not find, reads as incomplete phenomenology.

HOLD THIS

One sentence of challenge, one of demographics, then phenomenology, chronology, relative negatives, attribution, impact, coping, neurovegetative, safety. In that order, out loud.

WHAT EARNS THE MARKS

- **An opener that does a paragraph's work**: the diagnosis you favour, the differential you fear and the risk you have considered
- **Chronology per symptom cluster**: onset, duration, course, precipitants and severity, rather than one date for the illness
- **Positives paired with negatives**: the delusional forms you sought and did not find, and whether hallucinations command
- **The patient's own attribution**: it drives engagement and the alliance, and it is often the next question

SEE ALSO 03 The long case 10 Risk as a competency 16 Mental state and insight

16 · Mental state and structured insight

PRESENTING GRAMMAR · TOPIC 16

What did the mental state show, and how much insight does the patient have?

LONG CASE PRESENTATION, PSYCHIATRY SHORT CASE, COGNITION STATIONS

THE SHAPE OF THE ANSWER

1 The domains	Appearance, speech, thought, perception, mood and affect, cognition
2 Cognition	A named screen, with the errors recorded rather than summarised
3 Frontal testing	Motor sequencing, proverb abstraction, verbal fluency
4 Insight	Five graded components, each with its own sentence
5 The attribution	Name the psychopathology that impairs the insight

The mental state examination is presented across **its standard domains**: appearance and behaviour; speech, with rate, rhythm, tone, volume and intonation; thought form and content, covering delusions, overvalued ideas, ruminations and passivity; perception; mood and affect; and cognitive function.

Cognitive screening, specified rather than gestured at. Name the instrument and the errors, not the impression.

- **Key number.** A screen such as the Folstein Mini-Mental State Examination scores out of 30, with errors noted in attention and short-term memory at 5 minutes.
- **Key number.** Frontal testing sits alongside it: motor sequencing, proverb abstraction, and verbal fluency, for example 8 words beginning with the letter a in 1 minute with 2 repetitions.

COMPONENT	WHAT IS BEING EVALUATED	THE PHRASE THAT EARNS THE MARK
Awareness of change	Any subjective change from baseline?	**"The patient recognises these experiences as different from their usual state."**
Recognition of illness	Are the changes seen as illness?	**"The patient attributes the symptoms to an illness rather than to external persecutors."**
Attribution to mental illness	Is it identified as psychiatric?	**"The patient accepts that the symptoms stem from a psychiatric disorder."**
Attitude to treatment	Willing to accept treatment?	**"The patient expresses willingness to adhere to pharmacotherapy."**
Appraisal of social consequences	Is the impact on functioning understood?	**"The patient recognises how the illness has compromised occupational roles."**

Insight in five components, each with its sentence. Name the domain, altered mood, psychotic conviction or cognitive deficit, doing the damage.

TRAP Reporting insight as present or absent. A binary label, with no awareness of illness, attribution or attitude to treatment, invites the follow-up asking which phenomenon compromises it.

HOLD THIS

Insight in five graded components, then the psychopathology that impairs it. Mood is the sustained state the patient reports; affect is your observation of range, reactivity and congruence, and conflating them reads as a basic error.

WHAT EARNS THE MARKS

- **An instrument named with its score**: the screen, the total, and where the errors fell
- **Insight in graded components**: awareness, recognition of illness, attribution, attitude to treatment, appraisal of consequences
- **The phenomenon doing the damage**: altered mood, psychotic conviction or cognitive deficit, named as the cause
- **Mood against affect**: the sustained state the patient reports against your observation of range, reactivity and congruence

SEE ALSO 10 Risk as a competency 15 History in examiner order 17 The formulation

17 · The formulation: nomothetic, idiographic, 4P and 8P

PRESENTING GRAMMAR · TOPIC 17

Give me your formulation.

LONG CASE VIVA, AND ANY TABLE ASKING WHY THIS PERSON, NOW

THE SHAPE OF THE ANSWER

1	**Two processes**	Nomothetic classifies, idiographic explains

2 The 4P frame	Predisposing, precipitating, perpetuating, protective
3 The 8P expansion	Presentation, patterns, prognosis and plans added
4 Development	Age at onset changes the formulation and the plan
5 The pairing	Formulation and diagnosis given together, not one alone

Clinical evaluation runs on **two parallel processes**, and naming both is a reliable one-mark discriminator.

- **Concept.** The **nomothetic** process is law-giving classification, a system such as the International Classification of Diseases or the Diagnostic and Statistical Manual, which is what makes generalisation and comparative trials possible.
- **Concept.** The **idiographic** process is the picture of the individual: the formulation that explains why this person, with these vulnerabilities, defences and resources, developed this syndrome now.

The traditional frame is the **4P model**, predisposing, precipitating, perpetuating and protective factors. Academic practice frequently uses the expanded eight-part version, which adds the parts candidates leave out.

PART	WHAT IT HOLDS	WHAT THE EXAMINER LISTENS FOR
Presentation	Core problems and the reason for this encounter	**One sentence, not a recitation of the complaint list**
Predisposing	Genetic, neurodevelopmental, trauma	**Named vulnerabilities, not "positive family history"**
Precipitating	Acute stressors, medical illness, losses	**Why now, tied to a date or an event**
Patterns	Recurrent behavioural or interpersonal templates	**The repetition across episodes, usually the most useful observation you own**
Perpetuating	Ongoing stressors, substance misuse, stigma	**What will keep this going if untreated**
Protective	Strengths, family support, resilience, interests	**Specific assets you will build the plan on**
Prognosis	Risk variables weighed against therapeutic assets	**A balanced statement, neither bleak nor reassuring by default**
Plans	Biological, psychological and social management	**Sequenced by acuity, with the legal basis attached**

Patterns and prognosis are the parts most often skipped, and therefore most often asked about.

Developmental stage belongs inside the formulation. An illness such as schizophrenia beginning at age 14 years disrupts schooling, identity and peers; onset at age 30 years disrupts vocation, marriage and parenting. Same diagnosis, different formulation, different plan.

Fig 3.1 The presentation arc as a funnel: the one-sentence challenge and demographics at the mouth, then aetiological background, then salient psychopathology, narrowing into formulation, defended diagnosis and plan.

RULE A diagnosis without a formulation is a label; a formulation without a diagnosis is an essay. Give both, and say which parts of the account chose the classification.

HOLD THIS

Nomothetic classifies, idiographic explains. Presentation, predisposing, precipitating, patterns, perpetuating, protective, prognosis, plans.

WHAT EARNS THE MARKS

- **Both processes named**: classification and explanation, distinguished in one sentence
- **Patterns and prognosis**: the two parts most often skipped, and therefore the two most often asked about
- **Why now, tied to an event**: the precipitating part is where a date earns its mark
- **Developmental stage**: the same diagnosis at age 14 and at age 30 gives a different plan

SEE ALSO 13 Three formats 14 Summary versus formulation 18 Management by acuity

18 · Management, in the order of acuity

PRESENTING GRAMMAR · TOPIC 18

What will you do for this patient?

LONG CASE VIVA, EMERGENCY PROTOCOLS TABLE, PSYCHOPHARMACOLOGY TABLE

THE SHAPE OF THE ANSWER

1	Safety first	What could kill this patient this week, and where they are cared for
2	Clarification	Collateral interviews, longitudinal records, substance screening
3	Biological	Baseline workup, then the drug with its monitoring
4	Psychological and social	A named modality, family measures, rehabilitation
5	Critique	What your own plan cannot do, said before you are asked

A management plan is marked on **its order** as much as on its content. Present it as a hierarchy that starts with what could kill the patient this week and ends with what decides their next decade.

1. **Safety and risk management.** Immediate measures for suicidality, violence and severe self-neglect, including the level of observation and the admission decision, with its statutory basis.
2. **Diagnostic clarification.** Collateral interviews, longitudinal records, substance screening.
3. **Cultural adaptations.** Interpreters where needed, interventions aligned with the family's belief system.
4. **Biological management.** Baseline workup, then drug selection with titration, adverse effect counselling, and a plan for missed doses.
5. **Psychological interventions.** Psychoeducation, coping skills training, a named disorder-specific modality.
6. **Social and family measures.** Family psychoeducation, caregiver support, financial and housing stability.

7. **Psychosocial rehabilitation.** Community integration, vocational retraining, independent living skills.
8. **Critique of your own plan.** Where past treatment was inadequate, what yours cannot do, which barriers will obstruct it.

Specify the baseline and the monitoring, because that is where safety is visible.

- **Key number.** In severe anorexia nervosa, bradycardia of 48 beats per minute, blood pressure of 110/60 mmHg and a body mass index of 16 change the setting of care on their own.
- **Concept.** Renal and thyroid profiles are documented before lithium.
- **Concept.** Extrapyramidal symptoms and tardive dyskinesia are assessed at baseline and at intervals with an instrument such as the Abnormal Involuntary Movement Scale.

The same plan can be given in the **stepped form** examiners often prefer: setting of care justified by risk, self-care and social support, then biological, psychological, and social and rehabilitative measures, with the obligations flowing from the Mental Healthcare Act, 2017.

- **Concept.** Two statutory hooks carry most of the marks there: the right to community living, and the discharge plan made with the person, the nominated representative, the care-giver and the psychiatrist taking over.

TRAP Long-term pharmacotherapy with no baseline investigation named, or no motor assessment with an antipsychotic. Both read as indifference to the harm your own plan can cause.

INDIAN PRACTICE NOTE

Ask of every management answer: what does the family have to work with? Setting of care, supervision of medication, the feasibility of weekly follow-up and the cost of a second-generation antipsychotic are not footnotes in Indian practice, they are the plan. An elegant plan the family cannot execute will fail.

HOLD THIS

Safety, clarification, culture, biological, psychological, social, rehabilitation, critique. Name a baseline investigation and a monitoring instrument in every biological answer.

WHAT EARNS THE MARKS

- **Safety at the top**: the admission decision and its statutory basis, before any pharmacology
- **A named baseline investigation**: long-term treatment with no baseline named reads as indifference to harm
- **A monitoring instrument**: a motor assessment named alongside an antipsychotic, with its interval
- **A plan the family can execute**: setting, supervision, follow-up and cost are part of the plan in Indian practice

SEE ALSO 06 The viva tables 10 Risk as a competency 17 The formulation

19 · Cognitive traps in your own reasoning

PRESENTING GRAMMAR · TOPIC 19

How confident are you in that diagnosis, and what could have biased you?

LONG CASE VIVA, AND THE METACOGNITION QUESTION AT ANY TABLE

THE SHAPE OF THE ANSWER

1 The ask	Reflect on how you reached the conclusion you are defending
2 Anchoring	Fixation on the referral, or on the first data you saw
3 Framing	The pull of demographics, and of a selective summary
4 Limbic music	Presentation read as truer than declaration
5 The counter	A structured handover you can name and use

Examiners assess metacognition: **your capacity to reflect on how you reached the conclusion you are defending**. Diagnostic errors cluster into a few recognisable traps, and naming the one you avoided shows the reflection is real.

Premature anchoring is fixation on the first data, or on the impression handed over by referring staff.

- **Concept.** In the documented example, patients with known congestive cardiac failure were significantly less likely to be investigated for pulmonary embolism when the chief complaint highlighted the pre-existing failure.
- **Trap.** The psychiatric equivalent is the referral reading "known schizophrenia, relapse", taken as a diagnosis rather than a hypothesis.

Implicit and framing bias is the pull of demographic features, or of a selective summary that steers the listener to a narrow conclusion. It works in the handover you receive and the one you give.

Limbic music, a term attributed to George B. Murray, names the emotional reality emanating from the patient through posture, expression and tone. It is treated as a truer reflection of affective state than verbal declaration, because it bypasses the suppression of affect the same literature calls neocortical squelch.

Structured handover is the counter-measure, because unstructured handover loses information.

- **Concept.** Protocols such as SBAR, or the I-PASS mnemonic: illness severity, patient summary, action list, situational awareness with contingency planning, and synthesis by the receiver.
- **Key number.** I-PASS has been validated across 10,740 patients.

TRAP Premature closure in a late-onset presentation. Depressive symptoms with word-finding difficulty and cognitive slowing, worked up without considering imaging, is the standard illustration of a label closing an organic question too early.

HOLD THIS

Name the bias you checked for. Anchoring on the referral, framing by the summary, and trusting speech over presentation are the three tested when an examiner asks how confident you are.

WHAT EARNS THE MARKS

- **A bias named, not a claim of neutrality**: the reflection itself is what is being marked
- **The referral treated as a hypothesis**: known relapse is a starting point, not a diagnosis you inherited
- **Presentation weighed with speech**: posture, expression and tone set against the verbal declaration
- **A handover protocol you can name**: SBAR or I-PASS, with its components said in order

SEE ALSO 03 The long case 07 The five domains 15 History in examiner order

The thesis viva

20 · The external examiner and the twenty marks

THE THESIS VIVA · TOPIC 20

Who marks the dissertation, what is it worth, and what is being tested?

THESIS VIVA WITH THE EXTERNAL EXAMINER | THESIS 20 MARKS

THE SHAPE OF THE ANSWER

1 The money	20 marks inside the clinical and practical component
2 The examiner	From outside the state, marking the thesis and the defence
3 The percentage	Five per cent of clinical, practical and viva voce together
4 Ownership	Whether the question is yours or was handed to you
5 The rationale	Problem, boundary of the literature, objective in one sentence

The dissertation carries **20 marks** inside the clinical and practical component, and these are the cheapest marks on offer, because you have had three years to prepare for them.

- **Key number.** The regulator describes those marks as five per cent of the total marks of clinical or practical work and viva voce together.
- **Concept.** It is assessed by an external examiner from outside the state, and marks are given on the quality of the dissertation and on your performance in its viva.

What is being tested is **ownership**: whether the question is yours, or whether you completed an assigned project.

- **Concept.** Build the rationale in three parts. First the clinical problem as it appears in practice: treatment non-adherence, caregiver burden, physical comorbidity, symptom severity.
- **Concept.** Then the boundary of the literature, with the foundational studies named.
- **Concept.** Then the objective formulated to address that gap inside a three-year residency, with the primary objective stated in one sentence, separate from the secondary endpoints.

TRAP Misstating what the dissertation percentage is a percentage of. The wording is five per cent of clinical or practical work and viva voce together, not of the clinical and practical component alone. The smaller figure invites an immediate correction.

HOLD THIS

20 marks, an examiner from outside the state, five per cent of clinical, practical and viva voce combined. Marked on the dissertation and on how you defend it.

WHAT EARNS THE MARKS

- **The percentage stated correctly**: five per cent of clinical, practical and viva voce together, not of the clinical component alone
- **A rationale in three parts**: the problem, the boundary of the literature, then the objective
- **One sentence of primary objective**: kept separate from the secondary endpoints
- **A question you own**: the clinical problem described as you met it, rather than as the protocol phrased it

SEE ALSO 21 The five-minute summary 22 Sample size and statistics 25 Limitations and the grade

21 · The five-minute summary

THE THESIS VIVA · TOPIC 21

Summarise your dissertation for me in five minutes.

THESIS VIVA: THE OPENING QUESTION

THE SHAPE OF THE ANSWER

1 **Background and objective**	The clinical problem, then one sentence of aim
2 **Methodology**	Design, setting, criteria, tools, primary scales
3 **Key findings**	Your own numbers, quoted from memory
4 **Conclusion**	The contribution, and one volunteered limitation

Examiners routinely open by asking you to summarise the dissertation in five minutes, often looking for a distillation under 300 words.

- **Trap.** A rambling narrative sets the tone for the whole table, so **rehearse the opening as four blocks** rather than as a story.

BLOCK	WHAT GOES IN IT	THE DISCRIMINATOR
Background and objective	The core clinical problem and the specific aim or hypothesis	**One sentence of aim, distinguishable from the background**
Methodology	Design, setting, participant criteria, diagnostic tools, primary rating scales	**Design named in its technical form, not described loosely**
Key findings	Primary results with exact means, proportions or effect sizes	**Your own numbers, quoted from memory**
Conclusion and impact	The contribution to clinical care, and the major operational limitation	**A limitation volunteered, not extracted**

The four-block opening. An outside examiner should grasp the clinical relevance from this alone.

Length discipline continues afterwards, and **the difference is rehearsal against a clock**.

- **Key number.** Candidates at the top of the range answer in about 2 to 3 minutes on the precise question asked.
- **Concept.** Those in the middle give long answers that stray into general textbook psychiatry.

TRAP: VERIFY BEFORE THE EXAM Per-block timing targets for this opening are not stated in the library sources. Rehearse to the whole five minutes and to your own block order rather than to a split you cannot source.

HOLD THIS

Five minutes, under 300 words, four blocks: background and objective, methodology, findings, conclusion with its limitation. Then answers of two to three minutes to whatever follows.

WHAT EARNS THE MARKS

- **Four blocks in order**: background and objective, methodology, findings, conclusion with its limitation
- **An aim distinguishable from the background**: one sentence, said as the aim rather than buried in the problem
- **Your own numbers from memory**: exact means, proportions or effect sizes, without reaching for the thesis
- **A limitation volunteered**: offered by you, rather than extracted by the examiner

SEE ALSO 20 The external examiner 22 Sample size and statistics 25 Limitations and the grade

22 · Sample size and the statistics defence

THE THESIS VIVA · TOPIC 22

How did you arrive at your sample size, and why those statistical tests?

THESIS VIVA: THE METHODOLOGY AND STATISTICS QUESTIONS

THE SHAPE OF THE ANSWER

1 Why it is asked	An underpowered study yields inconclusive findings
2 The formula	State the one you used, naming every term in it
3 The assumptions	Alpha, power, and a named index study behind them
4 The pipeline	Distribution first, then the test that fits it
5 The honest gap	Carry the figures from your own protocol

Sample size is scrutinised harder than any other methodological choice, because **an underpowered study yields inconclusive findings**.

- **Key number.** In single-centre dissertations, final samples typically land between 80 and 200 participants, so the number is rarely the issue; the justification is.

Be able to state the formula you used, naming each term as you say it.

- **Concept.** For a prevalence or cross-sectional study, n equals Z squared times p times q, divided by d squared, where Z is the standard normal deviate for your alpha, p the expected prevalence from a named index study, q one minus p, and d the absolute allowable precision.
- **Key number.** For a two-group comparison on a continuous scale, it uses the standard deviation from prior literature, the minimum clinically meaningful difference between means, a two-sided alpha of 0.05, and power set at 80 per cent.

The analysis plan is defended as a pipeline, in order. Check the distribution numerically and visually before choosing a test.

- **Concept.** Report normally distributed variables as mean and standard deviation, comparing two groups with an independent samples t-test and three or more with one-way analysis of variance.
- **Concept.** Report non-normal or ordinal data, which includes most rating scale totals, as median and interquartile range, with the rank-based equivalents.
- **Concept.** Analyse proportions with chi-square, moving to an exact test where the table is sparse.
- **Concept.** Pearson for normal data, Spearman for skewed or ordinal, and justify any regression by naming the confounders it controls for.
- **Key number.** Significance at a two-tailed p below an alpha of 0.05.

TRAP: VERIFY BEFORE THE EXAM The standard normal deviate, the precision set for d, the beta behind your power, the names of the normality tests and the expected-cell threshold for an exact test are all unsourced in the library. Carry them from your own protocol, and confirm the threshold in a statistics reference before quoting one.

HOLD THIS

Alpha of 0.05, power of 80 per cent, and a named index study supplying p or the standard deviation. Distribution first, then the test, not the test first.

WHAT EARNS THE MARKS

- **A formula with its terms named**: Z, p, q and d identified as you write them out
- **Alpha and power stated**: 0.05 and 80 per cent, with the index study that supplied p or the standard deviation
- **Distribution before test**: the order of the pipeline is itself what is being marked
- **An unsourced figure flagged**: carried from your own protocol rather than guessed at the table

SEE ALSO 20 The external examiner 21 The five-minute summary 23 Scales

23 · Scales: why that one and not the other

THE THESIS VIVA · TOPIC 23

Why did you use that scale and not another one?

THESIS VIVA: THE INSTRUMENTS AND DIAGNOSIS QUESTIONS

THE SHAPE OF THE ANSWER

1 The shape of it	Construct, rater, and what the alternative would have cost
2 The families	One discriminator per domain, taken from the grid
3 Licence and language	Free for research, and the validated translation used
4 Diagnosis	The classification, and the instrument that verified it

Every instrument in your methods chapter invites the question of why you did not use a different one. The answer takes **the same shape each time**: what construct it measures, who rates it, and what the alternative would have cost you.

DOMAIN	INSTRUMENTS	THE DISCRIMINATOR TO LEAD WITH
Depression	HAM-D, PHQ-9, BDI	**Clinician-rated against self-report; recall period; scoring thresholds**
Anxiety	HAM-A, GAD-7	**Somatic against psychic anxiety; screening against severity**
Psychosis	PANSS, BPRS	**Three subscales; the semi-structured interview requirement**
Mania	YMRS	**Clinician-rated; core mood items operationally separated from behavioural ones**
Obsessive-compulsive symptoms	Y-BOCS	**Checklist separate from severity: time, interference, distress, resistance, control**
Sleep	PSQI	**Subjective sleep quality over the past month; seven component domains**
Substance misuse	AUDIT	**Score cut-offs for hazardous, harmful and dependent use**
Cognition	MMSE, MoCA	**Sensitivity for mild cognitive impairment; educational and cultural adjustment**
Quality of life and burnout	WHOQOL-BREF, Maslach Burnout Inventory	**Domain distribution; cross-cultural reliability**

One family per row, one sentence to defend it. The rest is your own data.

Two further questions follow reliably, and **both have documentary answers**.

- **Concept.** Whether the scale was free for non-commercial research, or needed a licence.
- **Concept.** Which validated vernacular translation you used.

Diagnostic confirmation is examined alongside the scales, because **it is what prevents diagnostic drift across a sample**.

- **Concept.** State the classification you diagnosed against: the International Classification of Diseases in its tenth or eleventh revision, or the fifth edition text revision of the Diagnostic and Statistical Manual.
- **Concept.** State whether it was verified with a structured instrument such as the Mini-International Neuropsychiatric Interview.

TRAP: VERIFY BEFORE THE EXAM

The item count of the mania rating scale is unsourced in the library. Do not quote a number; defend the instrument on who rates it and how its core mood and behavioural items are operationalised, and check the count in the scale's own manual.

HOLD THIS

Who rates it, what it measures, what the alternative would have cost, which translation you used. Four sentences defend any instrument.

WHAT EARNS THE MARKS

- **The rater named before the content**: clinician-rated against self-report decides most of these answers
- **The cost of the alternative**: what you would have lost by choosing the other instrument in the family
- **A licence and a translation**: two documentary facts you can produce on the spot
- **The verifying instrument**: the structured interview that holds the diagnosis steady across the sample

SEE ALSO 21 The five-minute summary 22 Sample size and statistics 24 Ethics and capacity

24 · Ethics, consent and capacity

THE THESIS VIVA · TOPIC 24

How was ethical approval obtained, and how did you consent a participant who lacked capacity?

THESIS VIVA, AND THE FORENSIC AND LEGISLATION TABLE

THE SHAPE OF THE ANSWER

1 **The approval**	Committee, date and protocol number, memorised
2 **The statute**	Consent read through the Mental Healthcare Act, 2017
3 **Capacity**	Assessed and documented before written consent
4 **Absent capacity**	Assent, plus a representative's written consent
5 **Over time**	Capacity re-assessed and recorded as status improves

Research ethics in an Indian postgraduate dissertation runs through the institutional ethics committee and the Mental Healthcare Act, 2017, and **this is one of the few places an examiner can check a fact on the spot.**

- **Concept.** Commit the approval date and the protocol reference number to memory.

Capacity cannot be assumed or dismissed in a psychiatric cohort. The protocol has to say how decision-making capacity was evaluated and documented before written informed consent.

- **Concept.** Where a participant lacks capacity because of acute illness, assent from the patient is accompanied by written informed consent from a legally acceptable or nominated representative.
- **Concept.** In a longitudinal study, capacity is re-assessed and recorded as status improves.

TRAP Either symmetrical error: assuming severe mental illness by itself precludes informed consent, or assuming capacity in an outpatient with no formal assessment. Both read as a failure to read the Act.

HOLD THIS

Approval date, protocol number, how capacity was assessed, who consented when it was absent. Four facts, memorised, and the station is closed.

WHAT EARNS THE MARKS

- **Two facts on demand**: the approval date and the protocol reference number
- **A documented capacity assessment**: how it was evaluated, rather than an assertion that it was
- **Assent plus representative consent**: the pair, where capacity was absent at recruitment
- **Re-assessment in a cohort followed over time**: capacity recorded again as the clinical status changes

SEE ALSO 10 Risk as a competency 20 The external examiner 25 Limitations and the grade

25 · Limitations, and the questions that decide the grade

THE THESIS VIVA · TOPIC 25

What are the limitations of your study, and what would you change?

THESIS VIVA: THE CLOSING QUESTIONS THAT SET THE GRADE

THE SHAPE OF THE ANSWER

1 Design	A cross-sectional design cannot show temporal precedence
2 Sampling	Convenience hospital sampling restricts generalisability
3 Measurement	Recall and social desirability, and inter-rater variability
4 Confounding	What ethics and sample size would not let you control
5 The remedy	A specific change attached to each defect named

Examiners look for **critical self-awareness**, not a claim of flawless execution. The limitations of a single-centre residency dissertation are predictable, and volunteering them in order reads as maturity.

1. **Design.** A cross-sectional design cannot establish temporal precedence or causal direction between symptoms and psychosocial variables.
2. **Sampling.** Convenience or purposive hospital sampling introduces selection bias; tertiary attenders are more severe and chronic, restricting generalisability to community populations.
3. **Measurement.** Self-report scales carry recall and social desirability bias; clinician-rated scales carry inter-rater variability without formal reliability testing.
4. **Confounding.** Concurrent medication, psychotherapy and comorbidity that could not be controlled within ethical and sample size constraints.

Then the question about what you would change, where **acceptable answers are specific**.

- **Concept.** A prospective longitudinal cohort, to follow symptom trajectories.
- **Concept.** Inter-rater reliability with a calculated agreement coefficient, built in before data collection.
- **Concept.** Multi-centre recruitment. Each of the three names a defect and its remedy.

DIMENSION	MIDDLE OF THE RANGE	TOP OF THE RANGE
Delivery	Long, unfocused answers straying into general textbook psychiatry	**Crisp answers of about 2 to 3 minutes on the precise question asked**
Methodological defence	Recites the methodology as written in the thesis	**Explains why that method over the alternatives, and the trade-off**
Diagnostic rigour	Relies on subjective clinical impression	**Names operational criteria and the structured instrument used to verify them**
Handling critique	Becomes defensive or anxious when an error is pointed out	**Concedes the boundary and defends the core data calmly**
Statistical command	Reports p values mechanically	**Articulates distribution, assumptions, effect sizes, and why parametric or not**

Four of these five dimensions are about how you talk, not what you found.

Publication is the closing question. Identify **the publishable unit**, the core empirical finding rather than the demographic tables, and say how the thesis would be condensed into a manuscript.

TRAP Defensive bluffing about an unfamiliar reference or term: examiners recognise it and press harder. Acknowledge the gap, say the analysis was not applied here, and place it as further study. The same immaturity shows in claiming no methodological flaws, or in attributing design decisions to your guide.

TRAP: VERIFY BEFORE THE EXAM Three questions have no answer in the library sources: the similarity index rule, which software and what threshold; the target journals; and the reporting standard for observational studies. Confirm the rule and your own figure with your thesis office, name journals you have actually read, and do not quote a percentage you have not verified.

HOLD THIS

Volunteer the limitation, name its remedy, answer in two to three minutes. Four of the five grading dimensions are about delivery and candour.

WHAT EARNS THE MARKS

- **A limitation volunteered in order**: design, sampling, measurement, confounding, given before they are extracted
- **A remedy attached to each defect**: the change you would make, rather than a regret about the one you did not
- **Answers of two to three minutes**: four of the five grading dimensions are about delivery and candour
- **A conceded boundary**: the limit acknowledged and the core data defended calmly

SEE ALSO 21 The five-minute summary 22 Sample size and statistics 24 Ethics and capacity

The discriminators of this note in one table	
THE SCHEME	400 in the practical head: 300 clinical and practical with 20 for the thesis inside it, plus 100 viva voce. Pass at 200 of 400, an aggregate of 50 per cent, no grace marks. The split inside the 300 is departmental.
THE CLOCK	Long case 45 minutes plus 15 to organise; each short case 25 plus 5. Stations at 5 to 10 minutes, or 120 seconds in dense circuits, rest every 30 to 40 minutes. The presentation length itself is stated nowhere: verify it locally.
LONG CASE LADDER	Informant reliability, chronology, systematic negatives, mental state, risk with protective and aggravating factors, formulation, defended differential, stepped management with its legal basis.
SHORT CASES	Observed, so technique is the mark. Finish the neurology case with a localisation and an aetiological differential; the sensory examination is the commonest omission.
MARKING	Five domains in every format. Domain scores from 1 to 5 plus one global judgement on six levels; borderline regression sets the cut. Two or more severe fails can end the examination regardless of aggregate.
SEVERE FAIL	Unsafe prescribing, ignoring active suicidal or violent intent, discharging a high-risk patient with safety unaddressed, or treating the patient without respect.
RISK	Nature, severity, frequency, likelihood, imminence. Then ideation, intent, plan, means, preparatory acts, modifiable and protective factors, and a contingency plan where the picture is ambiguous.
THE ACT	Section 4 capacity is decision-specific. Independent admission is the default. Section 89 is capped at thirty days; Section 90 changes the threshold to consistently over time. Section 94 excludes electroconvulsive therapy. Section 97 bars seclusion, and restraint is not available for a shortage of staff.
FORMULATION GRAMMAR	Summary is descriptive, lay, chronological. Formulation is analytic, technical, re-ordered: demographics, aetiology, then psychopathology, about 5 minutes and 10 to 15 sentences. Nomothetic classifies, idiographic explains.
INSIGHT AND AFFECT	Insight across five graded components, then the psychopathology that impairs it. Mood is reported by the patient; affect is observed by you.
THESIS	20 marks, examiner from outside the state, five per cent of clinical, practical and viva voce combined. Five-minute summary under 300 words in four blocks, alpha 0.05 and power 80 per cent with a named index study, distribution before test, limitation volunteered.
UNVERIFIED FIGURES	Presentation length, sensory examination depth, the mania scale item count, the standard normal deviate and precision, the normality tests and sparse-cell threshold, the similarity index rule, target journals:

The discriminators of this note in one table	
	each is unsourced here. Carry your own verified figure, or say you will verify it.

HOLD THIS, LAST

The practical marks a small number of visible behaviours: a spine you can hear, a risk sentence said before it is asked for, a legal basis attached to what you propose, and a number you can source.

PART II

The Psychiatry Long Case

The long case as a method: history in examiner order, the mental state with its elicitation, formulation to prognosis, and the ladders of twenty cases.

01 History in examiner order

02 Mental state examination, with elicitation

03 Physical examination and risk

04 Formulation to prognosis

05 Twenty long cases with the examiner's ladder

The Psychiatry Long Case

Five sections · 37 topics · the complete method for the practical

The long case is not a memory test, it is an ordered performance.

Order first, then content. Examiners listen for a proforma they already know, and they mark the places where a candidate has to think: the reliability of the informant, the words used to elicit a phenomenon, the negative history that earns a differential, the risk that is stated rather than implied, and a formulation that explains instead of repeating.

This note follows the sequence the examiner hears, gives the question that pulls each phenomenon out of the patient, and puts the one discriminator beside every near neighbour.

HOW TO USE THIS NOTE

Each topic ends with one line to hold. Grids carry the discriminator in the marked column, which is the cell an examiner is listening for. Where the evidence base does not settle a number, a trap box says so and tells you to verify it before the examination rather than inventing a figure at the table.

Statutory material is marked as an India note; quote a section number only where this note prints one.

History in examiner order

1 · The order of the presentation, and the identifying data

HISTORY · TOPIC 01

Present this patient, and tell me why each item of the identifying data is recorded.

LONG CASE, OPENING PRESENTATION

THE SHAPE OF THE ANSWER

1 The proforma order	Name the consensus sequence before you give any content
2 Identifying data	Each item with the probability or the plan it changes
3 Belief and subculture	Religion is the reference frame before a belief is called delusional
4 The statutory pair	Advance directive and nominated representative, present or absent

The examiner is following a proforma while you speak. Say the parts in the order they expect, and the content lands where they are already listening for it.

- **Concept. A national consensus format fixes the sequence.** It is endorsed by the Indian Psychiatric Society and the Indian Teachers of Psychiatry, and it opens with identifying data, the informant with reliability and adequacy, chief complaints in the patient's and the informant's words, the history of presenting illness, negative history, treatment history and past history.
- **Concept.** The sequence then runs family history, personal and developmental history, premorbid personality, the mental state, the physical examination, and **only then summary and formulation**.

- **Trap.** Treating identifying data as a warm-up. **Each item changes a probability, a plan or the legal position**, so each one is spoken for a stated reason.

ITEM	WHY IT IS RECORDED	WHAT THE EXAMINER LISTENS FOR
Age, gender, address	Age-dependent probability, gender-specific risk, distance from care	**Age as a diagnostic argument; distance as a follow-up problem**
Education, occupation	Cognitive baseline, literacy, baseline function	**The yardstick for any later claim of decline**
Religion	Explanatory model of illness	**Belief tested against the subculture before it is called a delusion**
Socioeconomic status, per capita income	Affordability of long treatment	**Required as an item; name your department's scale**
Marital status	Stability, support, legal standing	**Support quality, not the word "married"**
Advance directive	Treatment preferences recorded before treatment	**Stated present or absent, routinely**
Nominated representative	The statutory decision partner	**Named, with the order of precedence understood**

INDIA NOTE

The statute is the Mental Healthcare Act 2017, Act 10 of 2017.

Capacity is the ability to understand information relevant to a decision on treatment, admission or personal assistance, to appreciate the reasonably foreseeable consequence of the decision or of a lack of decision, and to communicate it by speech, expression, gesture or any other means (Section 4).

Any person who is not a minor may make an advance directive in writing, stating how he wishes and does not wish to be treated and appointing a nominated representative in order of precedence; it is invoked only once he ceases to have capacity, stays effective until capacity returns, and is overridden by a decision made while he has capacity (Section 5).

The nominated representative is appointed in writing, must not be a minor, must be competent to discharge the duties, and must consent in writing (Section 14).

- **TRAP** The socioeconomic status scale. The proforma requires the item but names no instrument. Verify which scale your department expects, and its domains, beforehand.

HOLD THIS

Identifying data closes on the two statutory items. Advance directive and nominated representative, present or absent, separates you from a candidate reciting a pre-2017 proforma.

WHAT EARNS THE MARKS

- **The sequence given unprompted**: it shows you know the proforma the examiner is marking against.
- **A reason attached to every item**: age, distance from care, education and income each move a probability or a plan.
- **Religion framed as an explanatory model**: the subculture is the frame that keeps a belief out of the delusion column.
- **The two statutory items spoken aloud**: advance directive and nominated representative, stated present or absent.

SEE ALSO 02 Informant and complaints 05 Family history and genogram 13 Possession and content 22 Summary and formulation

2 · The informant, the complaints, and the history of presenting illness

HISTORY · TOPIC 02

Who gave you this history, and how do you know it is reliable and adequate?

LONG CASE, HISTORY; OSCE HISTORY-TAKING STATION

THE SHAPE OF THE ANSWER

1 Characterise the informant	Identity, relationship, cohabitation, and the observation window it fixes
2 Defend reliability	Five parameters, each with what it actually tests
3 Chief complaints	Verbatim, chronological, a duration on each one
4 History of presenting illness	ABCDE per symptom, with functional consequence stated
5 Vegetative functions	Sleep, appetite, weight, libido, diurnal variation, bowel habit

Collateral carries the history whenever there is psychosis, mania, cognitive decline or impaired insight, so **characterise the informant before the account is believed**: identity, relationship, whether they live with the patient and for how long, which fixes the observation window.

- **Concept.** Reliability is **defended on the five named parameters, not asserted**. The grid below gives each parameter with the property it tests.

PARAMETER	WHAT IT TESTS
Consistency	Internal coherence of detail across the interview
Confirmability	Verifiable against records, prescriptions or a third party
Credibility	No secondary gain, deception or malice
Constancy	Stable on repeated enquiry
Adequacy	Enough intimacy, observation time and cognitive capacity

- **Concept. Chief complaints go in chronological order of appearance**, with a duration on each, verbatim rather than in jargon.
- **Key point.** Where the patient's and the caregiver's accounts diverge, give them under **separate headings**, because the discrepancy is itself data about insight.

- **Concept.** Then onset, course and precipitating factors, **separating the event that triggered the illness from the event that prompted this presentation**.
- **Concept. The history of presenting illness unpacks every symptom on ABCDE. A**ntecedent factors, meaning triggers, life events and the family's explanatory model; **B**ehaviours, the verbatim phenomenology; **C**onsequence for work, self-care and relationships; **D**uration and chronology; **E**pisodic nature, meaning periodicity, diurnal variation and fluctuation.
- **Concept.** The symptom-level alternative is **NOTEPAD**: nature, onset, triggers, exacerbating and relieving factors, progression, associated symptoms and disability.
- **Key point. Vegetative functions sit inside that narrative**, not in a separate list. Sleep as initial, middle or terminal insomnia, fragmented sleep, or reduced need for sleep without fatigue, which points to mania.
- **Concept.** The rest of the vegetative set: appetite, including refusal from depressive stupor or fear of poisoning; weight; libido; **diurnal variation such as morning worsening in melancholia**; and bowel habit.

- **RULE** **Complaints are quoted, the history of presenting illness is structured.** Verbatim words in the complaints, ABCDE in the narrative, functional consequence on every symptom.

HOLD THIS

"Informant is reliable and adequate" is not an answer. Defend it on the five parameters and name the observation window.

WHAT EARNS THE MARKS

- **The observation window stated as a figure**: how long this informant has lived with the patient bounds everything they can report.
- **Reliability argued parameter by parameter**: consistency, confirmability, credibility, constancy, adequacy, each with its test.
- **Divergent accounts kept apart**: the gap between patient and caregiver is evidence about insight, not an inconvenience.
- **Functional consequence on every symptom**: the C of ABCDE is the clause candidates drop.

SEE ALSO 01 Order and identifying data 03 Onset and course 04 Negative and treatment history 16 Insight and judgement

3 · Onset, course, and the milestone words

HISTORY · TOPIC 03

What was the onset and the course, and what does that pace argue for?

LONG CASE, HISTORY; VIVA TABLE ON COURSE AND OUTCOME TERMS

THE SHAPE OF THE ANSWER

1 Define onset	Interval from a well-adjusted baseline to a detectable abnormal state
2 Nature with time frame	Abrupt, acute, subacute, insidious, each with what it argues
3 Course as pattern plus phase	Episodic, continuous with exacerbations, chronic deteriorating
4 Inter-episodic functioning	Baseline recovery, residual symptoms, occupational capacity between episodes
5 The five milestone words	Response, remission, recovery, relapse, recurrence, in order

Onset is the interval between a well-adjusted premorbid baseline and a detectable abnormal mental state. **Give the nature and the time frame together**, because the pace is itself a differential.

ONSET	TIME FRAME	WHAT IT ARGUES FOR
Abrupt	Within 48 hours	**Organic, toxic or acute stress-related until excluded**
Acute	Clearly within 2 weeks	**Brief psychotic disorder, acute affective episode, substance-related state**
Subacute	Window not settled in the evidence used here	**State the observed span in weeks, quote no boundary**
Insidious	Gradual, undateable by the family	**Schizophrenia, dysthymia, a dementing process**

- **Concept. Course is a pattern plus a current phase.** Episodic with complete recovery, as in bipolar affective disorder and recurrent depressive disorder; continuous with exacerbations, as in schizophrenia; or chronic and deteriorating, as in neurodegenerative disorders.
- **Trap.** Omitting inter-episodic functioning, which is exactly what examiners want: **baseline recovery, residual symptoms and occupational capacity between episodes**.

1. **Response.** Clinically significant reduction in symptom burden during acute treatment.
2. **Remission.** Near-complete absence of symptoms with functioning restored, closing the acute phase.
3. **Recovery.** Sustained remission through the continuation phase.
4. **Relapse.** Syndromal return during continuation treatment, before recovery is established.
5. **Recurrence.** A fresh episode after full recovery.

RULE **Relapse is the same episode flaring before recovery; recurrence is a new episode after recovery.** The line is whether recovery was reached, not how severe the symptoms are.

TRAP Two follow-ups here carry no figure in the evidence used for this note: the definition of the duration of untreated illness and of untreated psychosis, and the episode count and window defining rapid cycling. Verify both from a manual beforehand; the prognostic direction of a long untreated psychosis is in topic 29.

HOLD THIS

Onset within 48 hours is abrupt, within 2 weeks acute. Say what the pace argues for in the same breath.

WHAT EARNS THE MARKS

- **A pace with an argument attached**: abrupt onset points organic or toxic until excluded, insidious onset points to a dementing or schizophrenic process.
- **Course stated as pattern plus current phase**: a pattern alone does not say where the patient is standing now.
- **Relapse and recurrence separated on recovery**: the line is whether recovery was reached, not how severe the symptoms are.
- **A declared gap on the unsettled figures**: the untreated-illness definitions and the rapid-cycling window are verified, not guessed.

SEE ALSO 02 Informant and complaints 25 Differential hierarchy 29 Prognosis

4 · Negative history, treatment history and past history

HISTORY · TOPIC 04

What did you ask about and find absent, and what did each denial exclude?

LONG CASE, HISTORY AND DIFFERENTIAL DEFENCE

THE SHAPE OF THE ANSWER

1 Why a negative history	Hypothesis-driven, placed at the end of the presenting illness
2 The four targets	Organic, substance, competing polarity, risk and function
3 Treatment history	Molecule, dose, duration at target, adherence, response, adverse effects
4 Past history	Every episode, one index episode in full, hospitalisations with legal status

Negative history is where the differential is earned. It is hypothesis-driven, deployed at the end of the history of presenting illness, and **organised so each denial excludes something specific**.

THE FOUR TARGETS

Organic. No fever, headache, vomiting or altered sensorium; no seizures, head trauma, loss of consciousness or focal deficit; no heat or cold intolerance, tremor or unexplained weight change.

Substance. No intoxication, heavy use or withdrawal immediately before onset.

Competing polarity. In suspected mania, no pervasive depressive cognitions and no first-rank phenomena unlinked to affective tone; in psychosis, no sustained elevation or depression and no primary obsessional phenomena; in obsessive-compulsive disorder, no thought alienation, delusions or depressive guilt.

Risk and function. No active suicidal intent or death wishes, no homicidal ideation, no wanderlust, no catastrophic self-neglect.

- Concept. **Treatment history is marked on precision.** Per trial: the molecule, form and route, maximum dose achieved, maximum duration at target dose, adherence, degree of response and specific adverse effects.
- Concept. Then psychological work with modality, duration and outcome, and past electroconvulsive therapy with **indication, response and cognitive effects**.
- Key point. Record the **faith healing and traditional treatment** most Indian patients tried first. It belongs in the record rather than in a joke, because it sets the explanatory model you will work with.

- **Concept. Past history establishes polarity and recurrence.** Every previous episode chronologically, one index episode in full, inter-episodic functioning, and hospitalisations with number, duration and legal status.
- **Concept.** Then the medical spine: epilepsy, central nervous system infection, thyroid disease, head trauma, metabolic disease, operations, and **drug allergy with the reaction pattern**.

TRAP "He was given olanzapine." Without dose, duration at target dose and adherence, nothing is said about whether the trial was adequate. The dose and the minimum weeks defining adequacy are not settled in the evidence used here, so describe what the patient actually received.

RULE **Anything unmentioned in a formulation is presumed absent**, which is why the negative history is spoken in the history and left out of the formulation.

HOLD THIS

A negative history that excludes nothing is a laundry list. Organic, substance, competing polarity, risk.

WHAT EARNS THE MARKS

- **Each denial tied to what it excludes**: a list of absent symptoms with no target attached scores nothing.
- **Dose and duration at target dose**: without them no claim about an adequate trial can be made.
- **Traditional treatment documented**: it is the commonest first contact in Indian practice and it shapes the explanatory model.
- **The negative history left out of the formulation**: anything unmentioned there is presumed absent, which is why it is spoken in the history instead.

SEE ALSO 03 Onset and course 22 Summary and formulation 25 Differential hierarchy 26 For, against, comorbidity

5 · Family history and the three-generation genogram

HISTORY · TOPIC 05

Draw this patient's genogram, and tell me why you took it across three generations.

LONG CASE, HISTORY; OSCE GENOGRAM DRAWING STATION

THE SHAPE OF THE ANSWER

1	Pedigree against genogram	Descent alone, against descent plus relationships and household
2	Why three generations	Polygenic liability, carrier states, variable expression, repeating scripts
3	Structural symbols	Shapes, proband mark, unions, offspring order, consanguinity, household
4	Emotional lines and shading	Between the symbols, only the dyads that bear on the illness
5	The Indian additions	Typology, cohabitants, and the three household roles

A pedigree records biological descent and Mendelian phenotype. **A genogram keeps the lineage and adds emotional relationships**, household boundaries, repeating behavioural scripts and family roles. Murray Bowen developed it within family systems therapy and McGoldrick and colleagues standardised it.

- **Concept. Three generations are mapped because psychiatric liability is polygenic.** Bilateral loading, unexpressed carrier states and variable expression across collateral branches only become visible across three.
- **Key point.** Three generations also expose **repeating scripts of neglect, substance use and trauma**, and recessive conditions that appear to skip a generation. Two generations are markedly less useful for formulation.

ENTITY	SYMBOL	CONVENTION
Male, female	Square, circle	**Uniform size, age inside or adjacent**
Index patient	Double shape or arrow	**Marked, so the case anchor is unambiguous**
Deceased	Diagonal cross through the shape	**Age at death and cause alongside**
Union, separation, divorce	Horizontal line beneath the partners, crossed obliquely if ended	**Male conventionally left; label the crossed line in writing**
Offspring, adopted, foster	Solid vertical line; dashed or dotted if not biological	**Left to right, oldest to youngest**
Pregnancy, miscarriage, abortion, stillbirth	Open triangle; triangle with a cross; with a cross and bar; half-size symbol with a cross	**Age at loss noted**
Twins	Converging sibling lines, bar added for monozygotic	**The bar is the only thing separating the two**
Consanguinity	Double horizontal line	**A single line conceals relatedness**
Household	Dashed perimeter	**Encloses everyone under one roof**

- **Concept. Emotional lines run between the symbols, not beneath them.** Single solid is close and supportive, double or triple parallel is enmeshed with blurred boundaries, dashed is distant, zigzag is conflicted, zigzag over parallel lines is conflicted and enmeshed, and an interrupted line or double slashes is a cutoff.
- **Key point. Chart only the dyads that bear on the illness.** A fully wired chart reads as decoration rather than as formulation.
- **Concept.** Morbidity, substance dependence and completed suicide are shaded or colour-coded with an annotation, **suicide carrying the age at death and the method**.
- **Trap.** Shading without a legend. Unlabelled shading has **no diagnostic validity in an examination**, so every genogram carries a legend defining each fill used.

SCENARIO	RISK	WHY IT MATTERS
First-cousin union	3 to 5 percent risk of malformation, genetic disorder or stillbirth, against a baseline of 2 to 3 percent	**Nearly double baseline; state it as a number**
Both parents recessive carriers	1 in 4 per pregnancy	**Rare neurometabolic disease presenting psychiatrically**
Fragile X premutation in the mother	55 to 200 repeats, expanding above 200 CGG repeats	**The commonest inherited cause of intellectual disability**

Fig 1 The genogram as drawn at the bedside: grandparents on the top line, parents and their siblings on the second, the index patient with siblings, spouse and children on the third, structural lines beneath the symbols, emotional lines between them, a dashed perimeter around the household, and a legend defining every shading used.

INDIA NOTE

Drawing the diagram is half the mark. Record the typology as nuclear, extended or joint, enclose the cohabitants, and name the three roles that rarely sit in the same relative: chief earning member, chief caregiver who gives meals and medicines, and chief decision maker who controls healthcare spending.

Consanguineous parentage justifies looking for inborn errors of metabolism, Wilson disease, organic dementia and treatment-resistant atypical presentations.

TRAP The slash count. One slash for separation and two for divorce is convention, not confirmed in the sources used here, which give one crossed-line symbol for both: draw it, name it in writing, define it in the legend. Paired trap: an empty ancestral branch is not a healthy one, because earlier generations went undiagnosed or hidden.

HOLD THIS

Three generations, proband marked, offspring in birth order, consanguinity a double line, and a legend. Those five omissions are where the marks go.

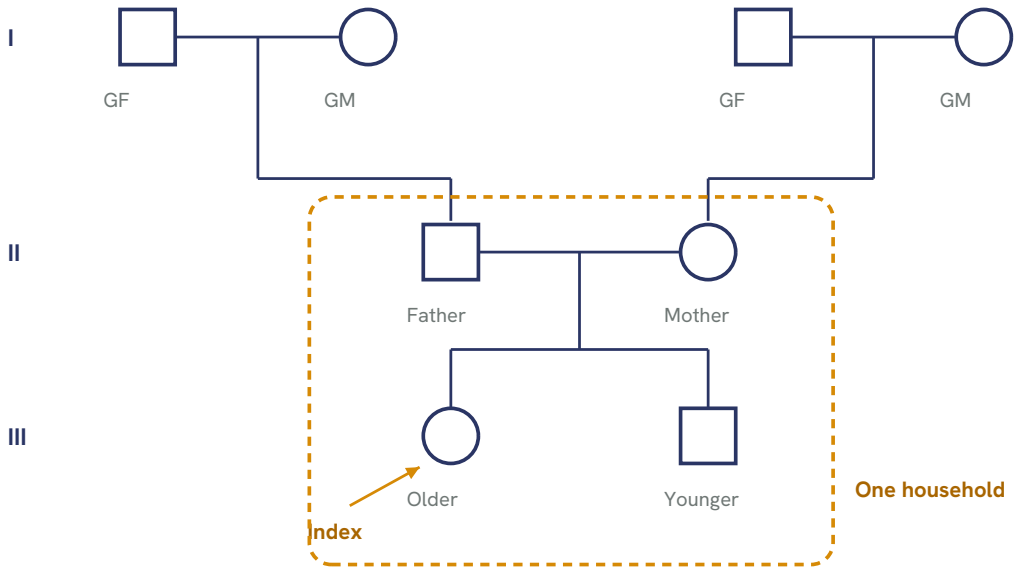
WHAT EARNS THE MARKS

- **Three generations drawn, not described**: the diagram itself carries half the mark in an OSCE station.
- **Consanguinity as a double line**: a single line conceals relatedness and with it a whole metabolic differential.
- **A first-cousin figure quoted**: 3 to 5 percent against a baseline of 2 to 3 percent is nearly double, and a number beats an adjective.
- **The three household roles named**: earning member, caregiver, decision maker, which rarely sit in the same relative.

SEE ALSO 06 Personal history 08 Substance, sexual, marital 23 Biopsychosocial and 5 P

Draw the family, not a list.

A fictional three-generation genogram with an explicit index and household.



READ THE LEGEND

Square: man Circle: woman Arrow: index

Solid lines: union and biological descent

Dashed perimeter: people living in the same household

Oldest sibling on the left. Unfilled does not mean healthy.

GF / GM: grandfather / grandmother. No clinical diagnosis is encoded.

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. A fictional family across three generations, with index arrow, birth order and household perimeter. No clinical diagnosis is encoded. Read from the grandparents to the parents and children. Squares and circles identify the symbols in the legend; the arrow identifies the index person and the dashed perimeter identifies one household. Unfilled symbols do not mean healthy.

Draw the relationships, then explain every symbol and line.

Schematic prepared by Luna for Weave Viva, based on chapter P1-03. Original edition artwork; publication licence to follow the edition.

6 · Personal history and development

HISTORY · TOPIC 06

Take me through the personal history, and tell me what it is the baseline for.

LONG CASE, HISTORY; SHORT CASE IN A DEVELOPMENTAL PRESENTATION

THE SHAPE OF THE ANSWER

1 Why it is taken	It is the yardstick for any later claim of decline
2 Perinatal and milestones	Pregnancy, delivery, cry at birth, then the three normative ages
3 Childhood in four groups	Regulation, conduct, habit and neurotic traits, physical health
4 Education and occupation	Trajectories, not single points; failures and job changes matter
5 Menstrual history	Menarche, regularity, last period, premenstrual mood change

Personal history reconstructs the biological and psychosocial foundation chronologically, and **it is the yardstick against which you later claim decline**.

- Concept. **Perinatal enquiry** covers maternal health in pregnancy, infection or teratogen exposure, whether the pregnancy was planned, gestational maturity, place and mode of delivery, whether the neonate cried at birth, neonatal complications, and parental consanguinity.

MILESTONE	NORMATIVE AGE	WHY YOU ASK
Sits without support	7 months	Gross motor delay, the earliest visible marker
Walks independently	15 months	Motor delay with later coordination implications
Two meaningful words	15 months	Speech delay, the strongest pointer to a neurodevelopmental disorder

- Concept. **Childhood is screened in four groups.** Behavioural and emotional regulation, meaning attention deficits, distractibility, impulsivity and restlessness; then conduct problems, meaning truancy, running away, fights, lying, stealing, destruction of property and cruelty to animals.
- Concept. The other two groups are **habit and neurotic traits**, meaning enuresis, encopresis, somnambulism, nail-biting, stammering, tics, pica and severe tantrums, and physical health, meaning serious illness, head injury, seizures and immunisation.
- Key point. Then **the family atmosphere it happened in**: parenting style, parental discord, domestic violence and adverse childhood experiences.

Essential screening across this territory takes about 5 minutes; where neurodevelopmental pathology or complex trauma is suspected, extended exploration takes between 10 and 20 minutes.

- Concept. **Education and occupation are trajectories.** Age at school entry, scholastic performance, failures, highest qualification, disciplinary action, peer relationships and bullying, which together measure cognitive baseline and early interpersonal style.
- Concept. Then **jobs in order**, with duration, reasons for change, debts, satisfaction and relations with supervisors, which measure reliability, response to authority and occupational impairment.
- Concept. **Menstrual history** covers menarche, cycle regularity, the last menstrual period and premenstrual mood change, identifying premenstrual dysphoria, cyclical exacerbation, and polycystic

ovarian syndrome associated with psychotropic exposure.

INDIA NOTE

The statutory frame is the Mental Healthcare Act 2017, not the British Mental Health Act 1983 that appears in overseas texts, and quoting a British section number in an Indian practical is an immediate discriminator against you.

Admission falls into two categories, independent admission for a person who has capacity and requests it, and supported admission for a person with high support needs; they carry different section numbers, so do not quote one number for both.

TRAP The section numbers for independent and supported admission, and their statutory time limits, are not settled in the evidence used for this note. Read them off the Act and verify each beforehand; a wrong section number spoken confidently is a wrong legal fact.

HOLD THIS

Personal history is the baseline you will later claim the patient fell from. Without school performance, job trajectory and milestones, "deterioration" has nothing to measure against.

WHAT EARNS THE MARKS

- **Milestones with normative ages**: sitting at 7 months, walking and two words at 15 months, each with the delay it flags.
- **School and job history as a trajectory**: it is the only thing a later claim of decline can be measured against.
- **Speech delay singled out**: it is the strongest pointer to a neurodevelopmental disorder in this part of the history.
- **The Indian statute named correctly**: the Mental Healthcare Act 2017, not a British section number.

SEE ALSO 05 Family history and genogram 07 Premorbid personality 15 Cognitive examination 37 Eating, disability, postpartum

7 · Premorbid temperament and premorbid personality

HISTORY · TOPIC 07

What was this patient like before the illness, and how did you establish it?

LONG CASE, HISTORY; VIVA TABLE ON PERSONALITY DISORDER

THE SHAPE OF THE ANSWER

1 Temperament or personality	Childhood onset takes temperament, adult onset takes personality
2 The nine categories	Thomas and Chess, grouped into the three classical child types
3 Six adult headings	Each one carries an anecdote rather than an adjective
4 Informant verification	State against trait cannot be settled by the patient alone
5 Disorder threshold	General criteria, then the prevalence and heritability figures

Temperament is the innate pattern of behavioural reactivity visible from infancy. Personality is the crystallised organisation of attitudes, defences and interpersonal traits built through

environmental interaction. **Childhood onset means you characterise temperament, adult onset means personality.**

- **Key point. The 9 Thomas and Chess categories.** Activity level; rhythmicity of hunger, sleep and bowel habit; approach or withdrawal to novelty; adaptability; threshold of responsiveness; intensity of reaction; quality of mood; distractibility; attention span with persistence.
- **Concept.** They group into **the easy child, the difficult child and the slow-to-warm-up child**. At the bedside temperament is still recorded plainly, shy and introverted against outgoing and extroverted.
- **Concept. Six headings carry adult premorbid personality**, following the classical Mayer-Gross and Schneider framework, and each one needs an anecdote rather than an adjective.
- **Concept. Attitude to self and to others.** Self-esteem against self-deprecation, boastfulness, health preoccupation and moral standards; then sociability, warmth against aloofness, dominance against submission, trust against suspicion, and empathy.
- **Concept. Attitude to work, and predominant mood.** Conscientiousness and perfectionism against procrastination, rigidity and resentment of supervision; then baseline tone, the threshold for anger, and whether feeling is expressed or restricted.
- **Concept. Fantasy life, and habits and leisure.** Whether daydreaming is a creative diversion or a substitute for real relationships; then tobacco, alcohol and other substances.
- **Trap.** Taking premorbid personality from the patient alone. **Informant verification is not optional**, because personality pathology, severe depression and psychosis often leave no insight into interpersonal impact.

FEATURE	SCHIZOID PERSONALITY	PRODROME OF SCHIZOPHRENIA
Time course	Lifelong, stable, ego-syntonic solitude	**Decline from a previously higher level of functioning**
Self-care	Preserved	**Deteriorating**
Thinking	Cool but not odd	**Attenuated positive symptoms, magical thinking, odd beliefs**

- **Concept. The general criteria for a personality disorder** require an enduring pattern deviating markedly from cultural expectation in at least two of four areas: cognition, affectivity, interpersonal functioning and impulse control.
- **Concept.** The pattern must also be inflexible and pervasive, cause significant distress or impairment, be **stable and traceable to adolescence or early adulthood**, and have no better explanation in another disorder, a medical condition or a substance.

PARAMETER	FIGURE	USE AT THE TABLE
Community prevalence	About 9 percent	**The denominator that stops over-diagnosis**
Clinic and inpatient prevalence	Up to 50 percent	**Why comorbidity is the rule in your ward**
Heritability	Approximately 50 percent across most types	**Answers the aetiology follow-up in a line**
Sex ratios	Antisocial 3:1 male to female; borderline 3:1 female to male in clinical settings, not in community samples	**The clinical-against-community caveat is the discriminator**
Below age 18	Features for at least 1 year; antisocial personality disorder is the stated exception and is not diagnosed under 18 years	**Quote the exception, not just the rule**

TRAP State against trait. A patient in a depressive episode describes a lifelong self-deprecating personality and a manic patient describes lifelong grandiosity; interview an informant, or use a period of euthymia, before accepting either as premorbid.

TRAP The constitutional typologies, Kretschmer's body types, Sheldon's somatotypes and Jung's introversion and extraversion, are asked here and are not covered by the evidence used for this note. Take the names and their proposed illness associations from a textbook beforehand.

HOLD THIS

Temperament for childhood onset, personality for adult onset, an anecdote under every heading. A list of pleasant adjectives scores nothing.

WHAT EARNS THE MARKS

- **The right construct for the onset**: temperament for a childhood-onset illness, personality for an adult-onset one.
- **An anecdote under each heading**: an adjective is an assertion, a remembered incident is evidence.
- **State separated from trait**: a depressed patient reports lifelong worthlessness and a manic one lifelong confidence, so use an informant or a euthymic period.
- **The clinical against community caveat on sex ratios**: the borderline ratio holds in clinics and not in community samples.

SEE ALSO 06 Personal history 11 Mood and affect 29 Prognosis 36 Dissociation and borderline

8 · Substance use, sexual and marital history

HISTORY · TOPIC 08

Quantify this patient's drinking, and tell me whether this is dependence or harmful use.

LONG CASE, HISTORY; OSCE SUBSTANCE AND SEXUAL HISTORY STATION

THE SHAPE OF THE ANSWER

1 Quantify the intake	Beverage, millilitres, drinking pattern, longest abstinence
2 Screen, then assess	AUDIT and CAGE prompt an assessment, they do not diagnose
3 The dependence syndrome	Edwards and Gross, then impaired control, salience, physiology
4 Withdrawal and complications	Uncomplicated, complicated, then organ damage by system
5 Sexual and marital history	Patient alone, five domains, then the non-consent question

Quantity and frequency first. Inventory every substance with route, typical daily dose, maximum in a single day, time of the last dose, and who introduced it.

- Key point. For alcohol, document the beverage type, whether Indian made foreign liquor, beer, wine or country liquor, **because country liquor concentration varies**.
- Key point. **Quantify in millilitres** by packaging or household measure rather than the word "peg". Then record drinking days per week, the time of the first drink, continuous against binge pattern, and the longest abstinence.
- Concept. **Name the screens.** AUDIT was developed within a World Health Organization collaborative project on the early detection of people with harmful alcohol consumption. CAGE covers cutting down, annoyance at others' concern, guilt and eye-opener drinking.
- Trap. Reading a positive screen as a diagnosis. **A positive screen prompts a dependence assessment, it does not diagnose dependence.**
- Concept. **Edwards and Gross, 1976**, separated the core dependence syndrome from alcohol-related disabilities. The biaxial point: severe physical harm can occur without dependence, and dependence can exist before end-stage disability.
- Concept. **The seven elements** are narrowing of the drinking repertoire, salience of drink-seeking, subjective compulsion, increased tolerance, repeated withdrawal symptoms, relief or avoidance drinking, and reinstatement after abstinence.

FEATURE	HAZARDOUS USE	HARMFUL PATTERN	DEPENDENCE
Category	Health risk factor, outside the disorders section	Substance use disorder	**Substance use disorder**
Present health harm	Absent, risk only	Present, physical or mental	**Often present, not required**
Impaired control, salience, physiology	Absent	Absent	**Present, and the three core features**

Table: harmful pattern cannot be diagnosed once the presentation meets dependence. ICD-11, published in May 2019, defines dependence by impaired control, increasing priority given to use despite harm, and physiological features meaning tolerance, withdrawal or relief drinking.

The contrast to state: ICD-11 keeps dependence categorical, while the DSM-5 continuum scores 11 criteria in total, with mild requiring 2 to 3, moderate 4 to 5 and severe greater than 6, which is why it captures mild cases that do not meet ICD-11 dependence.

- Concept. **Uncomplicated withdrawal** is autonomic hyperactivity, fine tremor, anxiety, insomnia, sweating and nausea.
- Key point. **Complicated withdrawal has three forms.** Withdrawal seizures, needing epilepsy, metabolic derangement and intracranial trauma excluded; delirium tremens, an emergency, with clouded consciousness, disorientation, coarse tremor, autonomic instability and prominent

hallucinations; and alcohol-induced psychotic disorder, with auditory hallucinations in clear consciousness.

- **Concept. Complications by system.** Fatty liver, hepatitis, cirrhosis, portal hypertension, variceal bleeding, pancreatitis; then cardiomyopathy, arrhythmia and hypertension; then peripheral neuropathy, cerebellar degeneration, Wernicke encephalopathy and Korsakoff psychosis.
- **Concept.** The social column belongs in the same list: **marital conflict, violence, job loss, debt and road traffic accidents**.
- **Trap.** Offering the laboratory markers as a diagnosis. Gamma-glutamyl transferase, mean corpuscular volume, carbohydrate-deficient transferrin and the aspartate to alanine transaminase ratio are **corroboration and abstinence monitoring**.
- **Concept. Motivation is staged, and the answer is the stage-matched strategy.** Precontemplation calls for rapport, information and the effect on the family rather than confrontation; contemplation calls for a decisional balance; preparation calls for concrete goals.
- **Concept.** Action calls for a structured plan with withdrawal management, maintenance for relapse prevention with a significant other, and **relapse is reframed as learning and recycled**.
- **Key point.** **An internal locus of control predicts adherence**, an external locus fuels rationalisation.
- **Concept. Sexual and marital history sits after education and occupation and before premorbid personality.** Take it with the patient alone, confidentiality assured and its limits stated.
- **Key point.** Open with a **normalising bridge**, that these questions are asked of patients of every age, gender and marital status.
- **Concept. Then five domains.** Partners, with number, gender and concurrency; practices, with anatomical site to direct site-specific testing; protection, with barrier consistency, vaccination and pre-exposure prophylaxis; past infections; and pregnancy intention.
- **Key point.** Close with the **non-consent question**, whether anyone has ever made them do something sexual they did not want.
- **Concept.** Record gender with the **two-step method**, sex assigned at birth then current gender identity, and say partner rather than husband or wife.

MARITAL ITEM	WHAT YOU RECORD	WHY IT IS ASKED
Marital profile	Age at marriage, duration of married life, arranged against chosen, consanguinity	**Consanguinity links back to the genogram**
Household	Joint against nuclear, earning member, caregiver, decision maker	**Decides who consents and who supervises medicines**
Adjustment and extended family	Intimacy, trust, conflict resolution; parents-in-law, dowry tension, fertility pressure	**Separates discord as cause from discord as consequence**
Marital sexual functioning	Consummation, frequency, satisfaction, dysfunction, drug effects	**Sexual adverse effects drive silent non-adherence**
Separation and legal	Separation, divorce petitions, maintenance, custody, violence allegations	**Changes both the risk and the plan**

- **Key number.** Two developmental figures are worth carrying. **Postpubertal males have about 10 times the circulating testosterone of females**, and in early adolescence major depressive episodes emerge with a female to male ratio of 2:1.
- **Concept. The Cultural Formulation Interview gives the culturally competent frame**, with 16 core questions across 4 domains: the cultural definition of the problem; perceptions of cause, context and

support; self-coping and past help seeking; and current help seeking.

- **Key point.** Psychosexual distress often presents somatically here, including **semen-loss anxiety**, a cultural explanation of distress in patients who attribute anxiety, fatigue, weakness, weight loss, impotence and depressive mood to the loss of semen.

COMMON TRAP

Interviewing with the spouse or in-laws in the room: abuse, discord, non-heterosexual attraction and undisclosed trauma are not reported in front of family, so a candidate who took this history with relatives present has not taken it. The paired trap is heteronormative assumption, because marriage and children do not establish heterosexuality.

TRAP Where forced sexual acts within marriage are disclosed, examiners ask for the Indian criminal-law position, including the exception relating to marital intercourse and its age qualification, and the contrast with remedies under the Protection of Women from Domestic Violence Act, 2005. That content is not in the evidence used here; verify it beforehand, because it governs what you may document, advise and refer.

RULE **Morbid jealousy against understandable distress.** A delusion of infidelity is fixed and maintained without evidence, with cross-examination, searching of belongings and a serious violence risk; a reaction to infidelity follows verified discovery and stays open to evidence.

HOLD THIS

Dependence is impaired control plus salience plus physiology. Organ damage alone is harmful use, however severe the cirrhosis.

WHAT EARNS THE MARKS

- **Intake in millilitres with a beverage type**: "three pegs" is not a quantity, and country liquor concentration varies.
- **The three core dependence features**: impaired control, salience and physiology, which is what separates dependence from a harmful pattern.
- **The history taken with the patient alone**: abuse, discord and undisclosed trauma are not reported in front of relatives.
- **Morbid jealousy separated from distress**: a fixed belief maintained without evidence carries a serious violence risk and changes the plan.

SEE ALSO 05 Family history and genogram 13 Possession and content 21 Suicide and violence 34 Alcohol and opioid

Mental state examination, with elicitation

9 · The order of the mental state, appearance, attitude and rapport

MENTAL STATE · TOPIC 09

Give me the mental state examination in order, and tell me why that order is fixed.

LONG CASE, MENTAL STATE; OSCE OBSERVATION STATION

THE SHAPE OF THE ANSWER

1 **The fixed sequence**	Appearance to insight, eight headings in a set order
2 **Why that order**	Observable signs before intrusive probing, which protects rapport
3 **Appearance and behaviour**	From the doorway: age, build, dress, hygiene, marks, stigmata
4 **Attitude against rapport**	Two separate observations, each with its own descriptor set
5 **Cultural reading**	Gaze, affect restraint and proximity norms differ by setting

The mental state examination is the psychiatric counterpart of the physical examination, **a cross-sectional snapshot at one point in time.**

- **Concept. Its order is fixed.** General appearance, attitude and behaviour including psychomotor activity; speech; mood and affect; thought as stream, form, possession and content; perception; cognitive functions; then insight and judgement with risk.
- **Key point.** The order moves from what you see without asking anything to the reflective capacity that lets a patient judge their own illness. **Observable signs are documented before intrusive psychopathology is probed**, which is also the sequence that protects rapport.
- **Concept. Appearance starts as the patient enters.** Apparent against chronological age; build and nutrition; dress and grooming, including whether it suits the weather; hygiene and odour, including alcohol or urine.
- **Concept.** Appearance closes on **marks and stigmata**, meaning self-harm scars, needle tracks and bruising, and signs of systemic illness such as jaundice.
- **Concept. Behaviour covers eye contact** as spontaneous, fixed and staring, intermittent or avoidant, then facial expression, gait, posture and mannerisms.

DOMAIN	WHAT IT DESCRIBES	DESCRIPTORS
Attitude towards the examiner	The patient's stance towards you	**Cooperative, guarded, suspicious, evasive, hostile, defensive, withdrawn, apathetic, demanding, overly familiar**
Rapport	Mutual responsiveness between the two of you	**Established; established with difficulty; not established but amenable for interview; not established**

Table: two different observations. If a patient with active persecutory beliefs still answers questions and permits examination, the record is that rapport could not be established but the patient remained amenable for interview.

- **Trap.** Moralistic words. **Replace "uncooperative", "manipulative" and "stubborn" with the observation itself**: the patient sat with arms crossed, gaze averted, and declined to answer questions about personal history.

INDIA NOTE

Non-verbal signs need cultural reading. Reduced eye contact is treated in Western texts as depression, guilt or evasion, but averting the gaze before elders, senior doctors and authority figures is culturally sanctioned deference.

Restrained affect may be emotional dignity rather than blunting, loud distress in a crisis may be a cultural grief norm rather than mania, and norms for proximity and touch differ between urban and rural settings and between genders.

TRAP Opening with "rapport established" as a reflex. If the patient spent the interview silent, suspicious or hostile, that sentence tells the examiner you were not watching.

HOLD THIS

Attitude is what the patient does towards you; rapport is what forms between you. Both get their own line, neither is asserted without a behavioural basis.

WHAT EARNS THE MARKS

- **The eight headings given in order**: the order itself tells the examiner you are working from a method.
- **Attitude and rapport on separate lines**: a guarded patient who still permits examination is rapport not established but amenable for interview.
- **Observation in place of a verdict**: arms crossed and gaze averted is data, "uncooperative" is a judgement.
- **Averted gaze read culturally**: deference before elders and senior doctors is sanctioned, not depressive guilt.

SEE ALSO 10 Psychomotor and speech 11 Mood and affect 16 Insight and judgement 17 Why you examine

10 · Psychomotor activity and speech in eight parameters

MENTAL STATE · TOPIC 10

Describe this patient's speech, and separate akathisia from agitation for me.

LONG CASE, MENTAL STATE; SHORT CASE ON ABNORMAL MOVEMENTS

THE SHAPE OF THE ANSWER

1	Psychomotor level	Increased, normal or decreased, then the involuntary movements
2	Agitation against retardation	Core disturbance, motor signs, speech signs, where each is seen
3	Speech in eight parameters	Give the normal finding as one sentence, then each deviation
4	Relevance against coherence	Independent axes, so answer the four-cell combination
5	The mute patient	Structured substitute, then Kirby's method in stupor

Psychomotor activity is **the physical expression of psychic activity**, read from posture, movement and speech.

- **Concept. Record the general level** as increased, normal or decreased. Then involuntary movements, meaning tremor, tics, grimacing, lip pursing, posturing, choreiform movements and mannerisms.
- **Key point.** Then the **drug-induced signs**: akathisia, parkinsonian rigidity and tardive dyskinesia.

FEATURE	AGITATION	RETARDATION
Core disturbance	Excess motor and cognitive activity from inner tension	**Generalised slowing of action, posture and thought**
Motor signs	Restlessness, fidgeting, pacing, hand-wringing	**Reduced spontaneous movement, fixed posture, delayed initiation**
Speech signs	Rapid rate, increased volume, shortened latency	**Prolonged reaction time, soft volume, reduced output**
Where seen	Mania, severe anxiety, mixed states, akathisia	**Severe depression, catatonic stupor, dementia**

- Key point. **Speech is described in eight parameters, delivered in order**, with the normal finding as one sentence: speech was spontaneous, normal in rate, tone and volume, with normal reaction time, relevant, coherent, and with normal prosody.
- Concept. **Rate**, slowed to pressured, with **pressure in mania and slowing in depressive retardation**. **Volume**, whispered to loud. **Tone**, meaning pitch and modulation, monotonous in depression, parkinsonism and autism, tremulous in anxiety.
- Concept. **Reaction time**, shortened and interrupting or prolonged as in psychomotor slowing. **Productivity**, paucity and monosyllabic answers to verbose. **Relevance, whether the reply addresses the question.**
- Concept. **Coherence**, whether syntax and semantics make it intelligible. **Prosody, the emotional cadence and rhythm.**
- Key point. **Relevance and coherence are independent axes**, and the four-cell combination is a favourite question.
- Concept. **Relevant and coherent**: the patient explains they came because of low mood and poor sleep. Irrelevant but coherent: asked when they came, the patient replies in perfect grammar that the weather is pleasant during the monsoon.
- Concept. **Relevant but incoherent**: asked where it hurts, the patient points to the abdomen and produces fragmented word approximations. Irrelevant and incoherent: word salad.
- Key point. **Pressure of speech** is accelerated, copious, forceful output difficult to interrupt, the audible face of flight of ideas in mania. Describe it as un-interruptible rather than quantifying it.
- Concept. **The mute patient cannot be given the eight parameters**, so run a structured substitute and say that you are doing so.
- Concept. Test comprehension with simple commands and look for appropriate nodding; test writing, gesture and pointing; then **examine the organs of speech** for cranial nerve pathology, vocal cord impairment or pseudobulbar palsy against psychogenic mutism.
- Concept. Note **phonatory effort**, whether the patient whispers or moves the lips silently. Screen catatonic signs, meaning waxy flexibility, catalepsy, negativism, automatic obedience, echopraxia and stupor, and record blinking, visual tracking, pupillary response and tearing.
- Key point. In a stuporous patient, **Kirby's method is the expected structure**: spontaneous posture, facial expression, resistance to passive movement, ocular responses, reaction to commands, and the catatonic signs.

COMMON TRAP

Tone against volume: volume is amplitude, tone is pitch, so a patient can be loud and monotonous or whisper with full modulation. Then dysarthria, impaired neuromuscular articulation with normal grammar and comprehension; aphasia, central language dysfunction; and mutism, absent output that may be functional, catatonic or organic.

TRAP Two numeric cut-offs circulate in notebooks for pressure of speech, a words-per-minute rate and a threshold score on Andreasen's Thought, Language and Communication scale. Neither is supported by the evidence used here nor traced to a manual, so do not offer either as a textbook cut-off.

RULE **Akathisia against agitation is a treatment decision.** Akathisia is a subjective urge to move, relieved by movement, with objective leg restlessness after a dopamine blocker; agitation is affective or psychotic tension. Mistake the first for the second and the antipsychotic dose goes up, which makes it worse.

HOLD THIS

Eight parameters, in order, in one sentence. Rate, volume, tone, reaction time, productivity, relevance, coherence, prosody.

WHAT EARNS THE MARKS

- **The normal finding as one fluent sentence**: it proves you hold the eight parameters as a set rather than a list.
- **Tone kept apart from volume**: volume is amplitude and tone is pitch, so loud and monotonous is a real description.
- **Akathisia separated from agitation**: mistaking the first for the second raises the antipsychotic dose and worsens it.
- **A declared gap on the pressure cut-offs**: the words-per-minute rate and the scale threshold are not traceable to a manual.

SEE ALSO 09 Appearance and rapport 12 Thought stream and form 19 Neurological screen 31 Mania and bipolar depression

11 · Mood and affect: the parameters and the terms

MENTAL STATE · TOPIC 11

Describe this patient's affect, and tell me the difference between restricted, blunted and flat.

LONG CASE, MENTAL STATE; VIVA TABLE ON AFFECTIVE TERMINOLOGY

THE SHAPE OF THE ANSWER

1 **Mood against affect**	Climate against weather, and both get recorded
2 **Mood recorded twice**	Subjective verbatim in quotation marks, then your objective appraisal
3 **The affect parameters**	Range, intensity, reactivity, stability, appropriateness, congruence, communicability
4 **The terminology grid**	What is reduced in each term, and where it is seen

5 Reactivity tested

Three probes, then record it as reactive or fixed

Mood is the pervasive, sustained, internally experienced feeling tone that colours perception and persists over days to weeks. Affect is the immediate, observable expression of emotion now. The analogy the examiner waits for: **mood is the climate, affect is the weather.**

- **Key point.** **Mood is recorded twice.** Subjective mood is the patient's own report, elicited with "How have you been feeling in your spirits recently?", written verbatim in quotation marks with duration and diurnal variation.
- **Concept.** **Objective mood is your appraisal across the encounter**, from behaviour, psychomotor activity, posture and the biological signs of sleep, appetite, weight, concentration, energy and libido.
- **Key point.** Where low mood is found, **screening for suicidal ideation follows in the same breath**.
- **Concept.** **Affect is broken into parameters.** Range, the variety shown; intensity, the depth of what is shown; reactivity, whether it shifts to cues; and stability.
- **Concept.** The remaining parameters are **appropriateness and congruence**, meaning fit with the social situation and fit with the stated mood and thought content, and communicability.

TERM	WHAT IS REDUCED	WHAT YOU SEE, AND WHERE
Broad or full	Nothing; baseline	**Fluid, proportionate facial and vocal response**
Restricted	Range, the variety	**A narrow band with moderate depth: depression, dysthymia, post-traumatic stress disorder, adjustment reactions**
Blunted	Intensity, the amplitude	**Unchanging face, sparse gesture, uninflected voice: schizophrenia as a negative symptom, melancholic depression, brain injury, Parkinson disease**
Flat	All outward display	**Immobile face, negligible eye contact whatever the topic: chronic deficit schizophrenia, stupor, advanced dementia**
Expansive	Nothing; it is excessive	**Boundary-crossing, over-familiar, grandiose: mania and hypomania**

- **Key point.** **Reactivity is tested, not assumed.** A neutral or mildly humorous remark to look for brightening, a sorrowful topic to look for gravity or tears, an empathic comment to look for relief.
- **Concept.** **Record it as reactive or fixed.** A fixed affect that does not shift is characteristic of severe melancholic depression, catatonic states and chronic psychotic illness.
- **Concept.** At the heightened pole, separate **elevated affect**, a cheerful energetic baseline above the patient's usual euthymia, from euphoria, an unwarranted sense of well-being disproportionate to circumstance, and from expansive affect.
- **Key number.** A manic episode requires abnormally and persistently elevated, expansive or irritable mood lasting **at least 1 week**, or any duration if hospitalisation becomes necessary.
- **Concept.** **Labile affect is rapid unpredictable swing on minor stimuli**, seen in mixed and manic states, borderline personality disorder, delirium and intoxication.

FEATURE	PSEUDOBULBAR AFFECT	PSYCHIATRIC LABILITY
Relation to inner feeling	Disconnected; may weep while feeling calm	**The patient genuinely experiences the emotion displayed**
Mechanism	Bilateral corticobulbar disruption disinhibiting brainstem centres for emotional expression	**Affective disorder, personality pathology, delirium or intoxication**
Form	Sudden, stereotyped, unprovoked or disproportionate	**Swings that track topic and interaction**

Table: the PRISM study screened 5,290 patients across neurological disorders and found a symptom prevalence of 36.7 percent, about half of those screened after traumatic brain injury, roughly 45 percent in amyotrophic lateral sclerosis and multiple sclerosis, 37.8 percent after stroke, 29.3 percent in Alzheimer disease and 26 percent in Parkinson disease.

CLINICAL ANCHOR

Asked whether a blunted patient with chronic schizophrenia feels anything, answer with the evidence.

Kring and Neale in 1996 showed that patients watching emotionally evocative films produced significantly less facial movement than controls yet reported comparable subjective emotional intensity and larger skin conductance responses, and a meta-analysis by Cohen and Minor in 2010 pooling 26 laboratory studies confirmed that the capacity to experience pleasant and unpleasant emotion remains largely intact.

Blunting is an expressive deficit, not an experiential one.

RULE **Appropriateness looks outward, congruence looks inward.** Laughing at a funeral is inappropriate, the reference point being the social situation; laughing while describing being poisoned by persecutors is incongruent, the reference point being the patient's own thought content and stated mood.

TRAP Calling a blunted affect flat. Flat requires the near-total absence of outward emotional sign, so a faint smile or any vocal inflection means blunted or restricted. Neighbouring trap: parkinsonian hypomimia, where facial immobility is bradykinesia and affective resonance remains intact.

HOLD THIS

Restricted narrows the range, blunted turns down the volume, flat switches it off. Range is variety, intensity is amplitude.

WHAT EARNS THE MARKS

- **Subjective mood quoted verbatim**: the patient's own words carry duration and diurnal variation with them.
- **Reactivity demonstrated with a probe**: an affect called fixed without a test is an assertion.
- **Appropriateness and congruence distinguished**: one looks outward to the situation, the other inward to the patient's own stated mood.
- **Blunting named as an expressive deficit**: the laboratory evidence shows experience largely intact behind the still face.

SEE ALSO 10 Psychomotor and speech 13 Possession and content 21 Suicide and violence 31 Mania and bipolar depression

12 · Thought stream and form, and how to stress-test them

MENTAL STATE · TOPIC 12

How would you demonstrate formal thought disorder in this patient at the bedside?

LONG CASE, MENTAL STATE; OSCE ELICITATION STATION

THE SHAPE OF THE ANSWER

1	The four aspects of thought	Stream, form, content, possession, each with its own question
2	Content against form	What is said against how it is organised
3	Stream phenomena	Each with the prompt that pulls it out of the patient
4	The provocation protocol	Four phases across a structured interview, not passive listening
5	Targeted clarification	"What did you mean by that?" is the diagnostic pivot

Thought is examined as four things. Stream, meaning tempo and continuity; form, meaning structural organisation as it appears in language; content, meaning what is believed; and possession, meaning ownership.

- Key point. The examinable line: **delusions are a disturbance of content, formal thought disorder a disturbance of organisation.** Content is judged by the truth value and fixity of what is said, form by the coherence of how it is said.

PHENOMENON	DEFINITION	THE QUESTION OR PROMPT
Racing thoughts, pressure of speech	Increased rate, rhythm and volume, hard to interrupt	"Do you experience too many thoughts coming at once, like you cannot control them?"
Inhibition or slowing	Reduced speed and spontaneous generation, prolonged latency	"Does your thinking feel slowed down, as if it is dragging under a heavy weight?"
Circumstantiality	Trivial detail and indirect paths, goal eventually reached	"Tell me in detail what you did yesterday from morning to evening."
Thought blocking	Sudden involuntary cessation mid-pursuit	"Does your train of thought suddenly stop mid-sentence, like hitting a wall, leaving your mind blank?"
Perseveration	Persistence of a previous response past its context	"I would like to change the subject now and ask about your family."

- Key point. **Formal thought disorder is a set of observable signs, not a reported symptom**, and passive listening does not find it.
- Concept. **The descriptor set runs to 20 items.** The ones to hold are poverty of speech; poverty of content of speech; pressure of speech; distractible speech; tangentiality; derailment; incoherence; illogicality; clanging.
- Concept. The rest of the set to hold: **neologisms; word approximations; circumstantiality; loss of goal; perseveration; echolalia; blocking; stilted speech; self-reference; and the phonemic and**

semantic paraphasias.

THE BEDSIDE PROTOCOL, FOUR PHASES

A dedicated 15 to 20 minute interview. Open discourse for 5 minutes, minimal scaffolding, for baseline rate and spontaneous output. Structured provocation for 5 minutes, with proverbs, abstract questions and picture description. Cognitive, affective and social demand for 3 to 5 minutes, with story recall, emotional prompts and perspective-taking.

Subjective verification for 5 minutes, asking directly about thought flow, blocking and interference. Targeted clarification runs throughout. Andreasen's original scoring interview ran to 50 minutes; brief research variants have used 6 questions in a 5 to 10 minute interview, recordings of 1.5 to 3 minutes, or video snippets averaging 1.9 minutes.

- **Key point.** **Free discourse for 5 to 7 minutes** removes scaffolding and forces internal organisation: "Tell me a story about an important event in your life."
- **Concept.** **Abstract stimuli test conceptual synthesis**: "What does it mean when people say, too many cooks spoil the broth?"
- **Key point.** **Cognitive loading taxes working memory**: an 8 to 10 sentence story to be retold, or a polysemic word such as tie or crane.
- **Concept.** **An affective probe establishes coherence on neutral ground**, then introduces a charged topic to see whether syntax collapses and recovers.
- **Concept.** **A perspective-taking prompt taxes social cognition**: "What does your mother think about your current difficulties?"
- **Trap.** Stopping at the objective signs. **Objective disorganisation and subjective awareness do not track each other**, so probe initiative, slowing, blocking or fading, poverty, pressure, interference, rumination, reception and expression.

RULE **Targeted clarification is the diagnostic pivot.** Ask "What did you mean by that?" A patient without thought disorder uses the scaffolding, repairs the slip and restores coherence; one with formal thought disorder derails further, elaborates bizarrely, or becomes visibly perplexed.

PAIR	A	B	THE DISCRIMINATOR
Detour	Circumstantiality	Tangentiality	**Circumstantiality reaches the goal eventually; tangentiality does not return**
Speed against linkage	Flight of ideas	Derailment	**Flight keeps understandable links through puns, rhymes or environmental cues at speed; derailment loses the link at normal tempo**

PAIR	A	B	THE DISCRIMINATOR
Quantity against substance	Poverty of speech	Poverty of content	**Poverty of speech reduces the amount said; poverty of content is a normal or excessive amount conveying little, through vagueness and inappropriate abstraction**
Novel words	Neologism	Paraphasia	**A neologism is a new word with private meaning; phonemic paraphasia mispronounces a real word, semantic paraphasia substitutes a related one**
Sudden stop	Thought blocking	Absence seizure, retardation	**Blocking is a subjective mid-sentence halt with the mind emptied; an absence has motor arrest with no recall; retardation is slow without sudden emptying**

INDIA NOTE

Several Indian languages do not use definite or indefinite articles, so in a patient speaking English as a second language, missing articles, unusual syntax and literally translated idiom can be mistaken for formal thought disorder. Hindi, Urdu and Bengali carry frequent homophones and rhyming idiom, so verify whether rhyming reflects regional cadence before calling it clanging.

Brief an interpreter beforehand to translate verbatim, preserving pauses and disorganisation, because the instinct to tidy speech into fluency masks the sign.

HOLD THIS

Formal thought disorder is provoked, then clarified. A 15 to 20 minute structured interview with clarification on every unclear remark finds what passive listening misses.

WHAT EARNS THE MARKS

- **A named provocation protocol**: four phases across a 15 to 20 minute interview, with clarification running throughout.
- **The repair test on an unclear remark**: a patient without thought disorder uses the scaffolding and restores coherence.
- **Circumstantiality against tangentiality**: the first reaches the goal eventually, the second does not return.
- **Language read before pathology**: missing articles and rhyming idiom can be regional cadence rather than clanging.

SEE ALSO 10 Psychomotor and speech 13 Possession and content 15 Cognitive examination 30 Schizophrenia

13 · Thought possession and thought content

MENTAL STATE · TOPIC 13

What words did you use to elicit thought insertion, and how is it not an obsession?

LONG CASE, MENTAL STATE; OSCE ELICITATION STATION

THE SHAPE OF THE ANSWER

1 **Define possession**	The ordinary certainty that thoughts are yours and private
2 **The four phenomena**	Insertion, withdrawal, broadcasting, obsessional doubt, each with its question
3 **Define a delusion**	Fixed, false, culturally out of keeping, impervious to proof
4 **Primary against secondary**	Arising de novo, against derivable from another morbid phenomenon
5 **The near neighbours**	Overvalued idea, obsession, somatic hallucination, thought blocking

Thought possession is **the ordinary certainty that the thoughts in your mind are yours**, private and under your control. Its disorders are failures of the boundary of the self, and they are elicited with direct questions.

PHENOMENON	THE EXPERIENCE	THE QUESTION
Thought insertion	Thoughts not the patient's own placed in by an external agency	**"Do you feel that thoughts are being put into your head from outside, against your will?"**
Thought withdrawal	Thoughts extracted by an outside power, leaving the mind empty	**"Have you ever felt that your thoughts are being taken out of your mind or stolen?"**
Thought broadcasting	Private thoughts escape and are known by others	**"Do you feel that other people can hear your thoughts without you speaking?"**
Obsessional thoughts and doubts	Intrusive, repetitive, recognised as one's own, resisted	**"Do unwanted thoughts or doubts keep entering your mind against your will, which you know are your own and try to resist?"**

Table: ownership is the axis. Alienated thoughts are attributed outside, obsessions are owned and fought.

- Key point. **A delusion is a fixed, false personal belief based on incorrect inference about external reality**, held with extraordinary conviction despite incontrovertible proof to the contrary, and out of keeping with the patient's cultural, social and religious background.
- Concept. **Name the varieties**: persecutory, grandiose, referential, somatic, erotomanic and nihilistic, the last including the belief that body parts, the self or the world do not exist.
- Concept. **Elicit with an open prompt first**: "What has been occupying your thoughts most of the time?"
- Concept. Then the specifics: **"Do you feel people are following you, talking about you, or trying to harm you?"** and "Do you notice events on television, radio or in newspapers that seem to carry a special meaning meant just for you?"

PRIMARY AND SECONDARY DELUSIONS

Primary delusions arise de novo and are not derivable from another morbid phenomenon; secondary delusions arise understandably from another mental event such as a disturbed mood state, a perceptual abnormality or a personality disorder.

In the primary group, a sudden delusional idea appears fully formed in a single stage; delusional mood or atmosphere is the state in which the patient knows something odd is going on around them which concerns them but cannot say what it is, and accepts it with relief when a delusional perception or idea finally crystallises; and delusional perception is a normal perception given an interpretation of delusional and abnormal importance.

Delusional memory is named in the same group, but its definition and elicitation cues are not settled in the evidence used here, so verify them beforehand.

PAIR	DELUSION	NEAR NEIGHBOUR
Against an overvalued idea	Fixed, false, culturally out of keeping, impervious to proof	**An overvalued idea is solitary, comprehensible within the life history, defended passionately, not recognised as senseless, and not bizarre: morbid jealousy, dysmorphophobia, the weight concern of anorexia nervosa**
Against an obsession	Ego-syntonic, firmly believed, not resisted	**An obsession is ego-dystonic, recognised as irrational, resisted, anxiety-provoking**
Against a somatic hallucination	A somatic delusion is a false belief about bodily function, such as infestation, with no sensory experience	**A somatic hallucination is the direct perception of a bodily sensation, such as an organ twisting**
Withdrawal against blocking	Withdrawal attributes the emptying to an outside agency stealing the thought	**Blocking is an internal halt with no external agency invoked**

INDIA NOTE

Shared cultural belief in black magic, astrological influence, spirit possession or ritual is not a delusion unless it departs significantly from the subcultural consensus and is accompanied by pervasive functional impairment. The test is departure from the patient's own community, not from the examiner's.

RULE **Suicidal and homicidal ideation is screened in every mental state examination, directly and without preamble.** Ruminations, hypochondriacal preoccupation and guilt go in the same paragraph, because they are the cognitions that carry the risk.

HOLD THIS

Possession asks whose thought it is; content asks what the thought says. Insertion, withdrawal and broadcasting are boundary failures; obsessions are owned and resisted.

WHAT EARNS THE MARKS

- **The exact question quoted**: naming the words that produced the phenomenon is what separates elicitation from recitation.
- **Ownership used as the axis**: alienated thoughts are attributed outside, obsessions are owned and fought.
- **Primary and secondary delusions distinguished**: delusional mood, sudden delusional idea and delusional perception sit in the primary group.
- **The subculture as the reference frame**: departure from the patient's own community is the test, not departure from yours.

SEE ALSO 12 Thought stream and form 14 Perception 21 Suicide and violence 30 Schizophrenia

14 · Perception: hallucinations, illusions, and the dissociative pair

MENTAL STATE · TOPIC 14

How did you establish these voices, and what makes them a hallucination rather than a pseudohallucination?

LONG CASE, MENTAL STATE; OSCE ELICITATION STATION

THE SHAPE OF THE ANSWER

1 Define the pair	Hallucination without a stimulus, illusion misperceiving a real one
2 Progressive elicitation	Setting, then noise against speech, then location and clarity
3 Characterise the voices	Number, person, complexity, commentary, command and tone
4 The other modalities	Visual, olfactory, gustatory, tactile, each with localising weight
5 Denial with signs	Document the denial and the observed response side by side

A hallucination is a perception in the absence of an external sensory stimulus, in any of the 5 modalities, carrying the quality, vividness and immediate reality of a genuine perception and occurring without voluntary control.

- Concept. **An illusion is the misperception of an actual external stimulus**, so wind becomes whispering and rustling leaves become figures.
- Key point. **Illusions are common in healthy people** during reduced sensory acuity, high arousal or transitional states, while hallucinations more often signify psychiatric disorder, drug toxicity or neurological disease.
- Trap. The generic opening question. **Asking whether someone hears voices invites denial in the suspicious and false endorsement in the suggestible**, so elicitation is progressive.
- Concept. **Open with the setting**: "When you are sitting quietly alone, do you ever notice unusual sounds or noises around you?" Then separate noise from speech.
- Concept. **Then locate it.** "Do these sounds seem like your own thoughts echoing inside your head, or do they sound like real voices coming through your ears from the outside world?" and "Do you hear them as clearly as you hear my voice right now?"

PARAMETER	THE QUESTION	WHAT IT TELLS YOU
Number, identity, gender	"One voice or several? People you know or strangers?"	**Multiple interacting voices suggest psychotic illness; familiarity separates trauma-related voices**
Grammatical person	"Do they speak to you, or about you?"	**Third-person commentary is a first-rank symptom**
Complexity	"Single words, or full sentences?"	**Simple noises point organic; complex syntax points functional**
Running commentary	"Do they narrate what you are doing or thinking?"	**A core first-rank symptom**
Command and tone	"Do they order you to do things? Hostile or soothing?"	**Commands to harm raise risk and change the setting of care**

- **Key point. Schneider's first rank symptoms, formulated in 1959, have perceptual members.** Hearing thoughts spoken aloud, or thought echo; voices arguing or discussing the patient in the third person; voices commenting on the patient's actions.
- **Concept.** The perceptual list closes on **somatic hallucinations attributed to external manipulation**, and delusional perception, a real perception given an abnormal, self-referential meaning without logical connection.
- **Concept. The non-perceptual members** are thought withdrawal, insertion, interruption and broadcasting, and the passivity experiences of made affect, impulses and volitions.
- **Trap.** Treating them as diagnostic. **Their sensitivity and specificity are not absolute**, so they cannot confirm schizophrenia alone, and they occur in affective psychoses, substance-induced psychoses and neurological disorders.

CONDITION	VISUAL DISTURBANCE	THE CLUE BESIDE IT
Migraine	Scintillating scotoma, fortification spectra	**The single most common cause of visual hallucination and illusion**
Dementia with Lewy bodies	Recurrent, formed, detailed people or animals	**A core feature, with fluctuating cognition, parkinsonism and dream enactment**
Charles Bonnet syndrome	Vivid complex images in severe visual impairment	**Insight preserved**
Seizure disorder	Flashes, geometric patterns, complex auras	**Paroxysmal and ictal, from occipital or temporal foci**
Delirium and toxic states	Vivid frightening scenes, small animals or insects	**Clouded consciousness, fluctuating orientation, autonomic arousal**
Narcolepsy	Hypnagogic or hypnopompic imagery	**Daytime sleepiness, sleep paralysis, cataplexy**

- **Key point. The other modalities carry localising weight.** Olfactory and gustatory hallucinations, asked about as unusual, foul or chemical smells nobody else detects and food tasting tainted, raise

suspicion of uncinate fits in temporal lobe epilepsy, head trauma and intracranial space-occupying lesions.

- **Concept.** Those same modalities **also occur in schizophrenia with delusions of poisoning**, so the localising weight is a prompt to look, not a verdict.
- **Concept.** **Tactile and somatic hallucinations**, asked about as crawling, burning, electric shocks or insects under the skin, include formication in withdrawal and stimulant intoxication.
- **Key point.** **Not all perception is pathology.** Hypnagogic perceptions occur at sleep onset and hypnopompic on waking, and hearing the voice or briefly seeing the figure of someone who has died is a recognised grief phenomenon in the acute period.
- **Concept.** **A divine voice offering benign encouragement may fall inside culturally normative religious experience**; one commanding self-harm or abrupt travel does not.

FEATURE	DEPERSONALISATION	DEREALISATION
What feels detached	The self, the body, one's own thoughts	**The external world, surroundings, other people**
Elicitation	"Do you ever feel detached from your body, as though you are observing yourself from the outside?"	**"Do your surroundings ever feel artificial, dreamlike, or two-dimensional?"**
Reality testing	Preserved	**Preserved, which separates both from psychosis**

Mood congruence is analysed against the prevailing affective state. Congruent: a depressed patient hears voices mocking their unworthiness or urging suicide; an elated patient hears voices proclaiming a chosen status. Incongruent: a paranoid patient hears peaceful soothing voices, or a euphoric manic patient hears that their organs are rotten.

CLINICAL ANCHOR

Patients deny hallucinations for good reasons: persecutory belief, voices ordering silence, fear of admission, stigma.

Document the denial and the observation side by side, recording the signs of response to internal stimuli: abrupt speech pauses without external distraction; head tilting, ear cupping or gaze at empty corners; unprompted smiling or muttering; earplugs or loud music; darting eye movements tracking invisible trajectories.

The record reads: patient verbally denies auditory and visual hallucinations, however the patient was observed pausing mid-sentence, cocking the head towards the wall and muttering inaudibly, consistent with active response to internal stimuli.

RULE **Space, not vividness, separates a hallucination from a pseudohallucination.** A true hallucination is in external objective space with full perceptual reality; a pseudohallucination is in internal subjective space, keeps its sensory quality, and the patient retains the insight that it is not an external physical reality.

TRAP The Fish subtypes are a standard probe. Extracampine hallucinations are visual hallucinations located outside the field of vision, usually behind the head; the definitions of functional and reflex hallucinations are not settled in the evidence used here, so read them from a primary psychopathology text rather than improvising at the table.

HOLD THIS

Illusion has a stimulus, hallucination has none, delusional perception has a normal stimulus and an abnormal meaning. The third is a disorder of content.

WHAT EARNS THE MARKS

- **Progressive rather than generic questioning**: setting, then noise against speech, then location and clarity.
- **Space named as the discriminator**: external objective space for a true hallucination, internal subjective space for a pseudohallucination.
- **Denial documented beside the observation**: pausing mid-sentence and muttering towards a wall is recordable evidence.
- **Depersonalisation and derealisation kept apart**: reality testing is preserved in both, which is what separates them from psychosis.

SEE ALSO 13 Possession and content 15 Cognitive examination 30 Schizophrenia 36 Dissociation and borderline

15 · The cognitive examination in sequence

MENTAL STATE · TOPIC 15

Examine this patient's cognition, and tell me why attention comes before memory.

LONG CASE, MENTAL STATE; SHORT CASE AND OSCE COGNITIVE STATION

THE SHAPE OF THE ANSWER

1 The fixed order	Consciousness, orientation, attention, memory, intelligence, abstraction, judgement
2 Consciousness operationally	Stimulus needed, response given, how long the level holds
3 Attention tests	Serial sevens, spelling backwards, digit span, with their limits
4 Memory in three compartments	Registration, recent, remote, then cue to localise the failure
5 Cortical closers	Abstraction, parietal signs, praxis, then the named instruments

The content of consciousness cannot be characterised unless the patient is awake and alert, so **the order is consciousness, orientation, attention, memory, intelligence and fund of information, abstraction, judgement.**

- Key point. **Attention is tested early because an inattentive patient cannot register**, and every later result then becomes uninterpretable.
- Concept. **Consciousness is recorded operationally**, not as the bare label drowsy or stuporous: the exact stimulus needed to arouse, whether auditory, tactile or noxious; the specific response, meaning eye opening, vocalisation, motor withdrawal or cooperation; and how long that level is sustained.
- Key point. **Fluctuation is itself a finding**, so record it rather than averaging it away.

- **Concept. Orientation covers three fields.** Time, the exact date, day, month, season and year, which fails earliest; place, the hospital, ward, floor, city and state; and person.

- **RULE** **Isolated loss of orientation to person, with surroundings intact, is not a focal structural lesion.** It points to dissociative amnesia, malingering or severe functional disturbance, and organic disease produces the reverse pattern.

TEST	ADMINISTRATION	SCORING OR NORMATIVE POINT
Serial sevens	Subtract 7 from 100, then 7 from each remainder	**14 possible correct subtractions down to 2, each an independent step, so an arithmetic slip followed by accurate continuation penalises only the first step**
Spelling backwards	A 5-letter word such as WORLD in reverse	**Orthographic processing and working memory rather than calculation**
Serial threes, reverse sequences	Count down by threes, or months and days in reverse	**Threes from 40, or backwards from 20 to 1, replaces WORLD where the patient does not read English**
Digit span	Digits at one per second, forward then backward	**Normal forward span 5 to 7 digits, book range 5 to 9; reverse span tests working memory**

COMMON TRAP

Treating serial sevens and WORLD backwards as interchangeable. Serial sevens is harder, has a lower completion rate and demands mental calculation; the two correlate only weakly and both are strongly influenced by education, so failure can come from dyscalculia, anxiety or low schooling rather than a memory syndrome. The task was documented in clinical use as far back as 1944.

- **Key point. Memory is tested in three compartments.** Immediate registration, by repetition of 3 unrelated words or forward digit span; recent memory, by unprompted recall of the same 3 words after a delay and by verifiable events earlier the same day.
- **Concept. Remote memory is tested by verifiable biography**, meaning date of marriage, schools attended, place of birth.
- **Trap.** Scoring memory in an unregistered patient. **If the patient cannot repeat the words immediately the defect is attention or registration**, and memory is then untested rather than impaired.

- **RULE** **Cueing separates retrieval failure from storage failure.** If spontaneous recall fails at 2 to 5 minutes, give a category cue or a multiple choice: if cueing restores the word the deficit is frontostriatal retrieval, and if cueing does not help it is hippocampal encoding and consolidation failure, the Alzheimer pattern.

Intelligence and fund of information are interpreted against education, culture and background. Names of current political leaders, prominent landmarks and major events;

vocabulary and category fluency by naming as many animals as possible in 1 minute; and calculation in local currency.

Inability to calculate, with agraphia, finger agnosia and right-left disorientation, points to **Gerstmann syndrome and the dominant angular gyrus**.

Abstraction uses similarities and proverbs. Similarities such as orange and banana as fruit, then proverbs graded in three tiers: abstract scoring 2 points where the generalised meaning is captured, semi-abstract scoring 1 point where the generalisation is partial, and concrete scoring 0 points for a literal reading.

A concrete response accompanies **frontal pathology, schizophrenia and moderate to severe dementia**.

INDIA NOTE

Bedside fund-of-knowledge and vocabulary tasks estimate intelligence, they do not measure it. For an illiterate or minimally educated patient, name an Indian-standardised instrument: the Binet-Kamat Test and the Bhatia Battery are the commonly used tests in Indian practice. Proverbs must be locally familiar, because the metaphor is the test, not the translation.

INSTRUMENT	STRUCTURE	USE AND LIMITATION
Mini-Mental State Examination	A 30-point scored screen	**Weighted to verbal memory and orientation, insensitive to frontal executive impairment, no substitute for the mental state examination**
Montreal Cognitive Assessment	Scored out of 30	**Samples attention, executive function, memory, language, visuospatial skill, abstraction, calculation and orientation, so it is preferred for broad screening**
Addenbrooke's Cognitive Examination, third edition	15 to 20 minutes, out of 100, cut-offs 82 to 88	**Five domains with high sensitivity for dementia, light on frontal executive function, visual memory, apraxia and agnosia**
Frontal Assessment Battery	A standardised composite bedside frontal instrument	**Name the battery first, then verbal fluency, cognitive estimates, the Luria fist-edge-palm sequence, go and no-go, reverse digit span, alternating graphical sequencing**

- **Concept. Parietal testing and praxis close the cortical screen.** Right-left orientation, finger recognition, astereognosis and dysgraphaesthesia.
- **Key point. Construction by drawing a clock face to a set time or intersecting pentagons.** Impairment is more pronounced in dementia with Lewy bodies than in Alzheimer disease.

- **Concept. Praxis by pantomime.** Using a body part as the object, scrubbing the teeth with an index finger, demonstrates subtle ideomotor apraxia.

TRAP Calling an inattentive patient amnesic, and calling depressive pseudodementia a degenerative dementia. In pseudodementia the patient gives "I do not know" answers with poor effort; in neurodegenerative dementia the patient makes near-miss errors, minimises the deficit, or confabulates.

HOLD THIS

Attention first, or nothing after it counts. A failed immediate repetition is a registration failure, and memory is then untested rather than impaired.

WHAT EARNS THE MARKS

- **Attention placed before memory with a reason**: an inattentive patient cannot register, so later results become uninterpretable.
- **Cueing used to localise the failure**: restored recall points frontostriatal retrieval, unhelped recall points hippocampal encoding.
- **An Indian-standardised instrument named**: the Binet-Kamat Test and the Bhatia Battery for a minimally educated patient.
- **Pseudodementia separated by error type**: "I do not know" with poor effort, against near-miss errors and confabulation.

SEE ALSO 14 Perception 16 Insight and judgement 19 Neurological screen 35 Delirium and dementia

16 · Insight and judgement

MENTAL STATE · TOPIC 16

Grade this patient's insight, and tell me why judgement is not graded the same way.

LONG CASE, MENTAL STATE; VIVA TABLE ON INSIGHT AND CAPACITY

THE SHAPE OF THE ANSWER

1 Define insight	Continuous, multidimensional and dynamic, not present or absent
2 The three dimensions	Acknowledgement, need for treatment, relabelling of symptoms
3 Grade it	Six grades, with what the patient says at each one
4 Judgement in three components	Personal, social, test, reported intact or impaired
5 The statutory link	Insight is not capacity, but a poison delusion destroys appreciation

Insight is the degree to which a patient recognises that they are ill, understands the consequences, attributes symptoms to a morbid psychological state and accepts treatment.

- **Concept.** Aubrey Lewis framed it as **the patient's correct attitude to a morbid change in themselves**.
- **Key point.** The modern correction the examiner waits for: **insight is continuous, multidimensional and dynamic rather than present or absent**.
- **Concept. Anthony David, in 1990**, defined three dimensions: acknowledgement of illness; recognition of the need for treatment and compliance; and relabelling of symptoms, meaning the capacity to identify unusual experiences as abnormal and arising from a disease process.

- **Key point.** A patient can hold one dimension and not another, **which is why partial insight is a meaningful record.**

GRADE	LABEL	WHAT THE PATIENT SAYS
Grade 1	Complete denial	**Rejects any notion of illness or need for help**
Grade 2	Slight awareness with simultaneous denial	**Ambivalent, fluctuating awareness of being unwell**
Grade 3	External attribution	**Accepts being ill but blames others, malicious influence or a bodily disease**
Grade 4	Awareness of an unknown internal cause	**Accepts something within is wrong, without connecting it to a psychiatric condition**
Grade 5	Intellectual insight	**Names the illness and calls the symptoms abnormal, without applying it to behaviour or adherence**
Grade 6	True emotional insight	**Emotional awareness of the motives behind the symptoms, applied to life decisions and sustained adherence**

Table: the classic six grades. Concise records may instead say absent, partial or full.

- **Key point.** **Elicit it with five sequential non-leading questions.** Do you feel you are having any health problem or change in your thoughts, mood or behaviour? Do you think these difficulties are physical, or related to a nervous or mental condition?
- **Concept.** Then: **What do you think is causing them? Do you think you need treatment, medication or admission?**
- **Key point.** The fifth question is the discriminator: **looking back at the unusual experiences, do you believe they were real or part of an illness?** It separates grade 5 from grade 4, because it tests relabelling rather than agreement.

The instrument to name is David's Schedule for Assessment of Insight, a clinician-rated semi-structured interview of 8 questions.

The first 7 items cover treatment acceptance, treatment request, knowledge of illness, knowledge of mental illness, explanation of illness, belief in the truth of delusions and explanation of experiences, **giving a maximum of 14**. The eighth item on reaction to disbelief raises the maximum to 18, higher meaning better insight.

For contrast, **the Birchwood Insight Scale is an 8-item self-report** scored 0 for poor insight, 1 for unsure and 2 for insightful.

The Scale to Assess Unawareness of Mental Disorder rates global and symptom-specific awareness, and **the Beck Cognitive Insight Scale measures metacognitive flexibility** across self-reflectiveness and self-certainty rather than awareness of illness.

THE INSIGHT PARADOX

Gaining insight is not uniformly good.

Higher awareness of having a mental illness is associated with depressive symptoms, lower self-esteem and reduced quality of life, and structural modelling attributes this to internalised stigma and shame: awareness predicts increased shame with a standardised beta of 0.36, higher internalised stigma with a beta of 0.27, and reduced self-esteem with a beta of 0.25, correlating with shame at r = 0.53.

The dimension covering need for treatment does not correlate with stigma or shame. Clinically, as psychosis clears and awareness dawns, patients may internalise the stereotype and become vulnerable to post-psychotic depression and suicide, so psychoeducation is paired with work that challenges internalised stigma.

Insight deficits occur in 50 percent to 80 percent of patients with schizophrenia, and previous hospitalisation predicts higher illness awareness.

COMPONENT	HOW IT IS ASSESSED	WHAT IMPAIRMENT LOOKS LIKE
Personal judgement	Realistic plans for discharge, livelihood, finances, safety, treatment	**Impossible, hazardous or delusionally motivated plans, such as giving away all savings on a grandiose belief**
Social judgement	Observed ward conduct towards staff, patients and boundaries	**Disinhibition, sexual indiscretion, unprovoked aggression, bizarre public conduct**
Test judgement	Hypotheticals: the stamped letter on the pavement, smoke in a crowded cinema, a lost child in a market	**Answers read against culture, intelligence and schooling, not a textbook script**

Table: judgement is not graded numerically. It is reported as intact or impaired.

RULE **If any one component is impaired, judgement is impaired**, and ward behaviour outweighs a textbook answer to a hypothetical. Grade 5 talks, grade 6 changes: a patient who agrees they have an illness and intends to stop medication at discharge has intellectual insight, not emotional insight.

INDIA NOTE

This part of the mental state carries statutory weight: documented impairment of insight and judgement, with severity of illness and risk of harm, is the clinical material a supported admission decision rests on, and capacity under the Mental Healthcare Act 2017 turns on understanding the relevant information, appreciating the reasonably foreseeable consequences, and communicating the decision (Section 4).

Insight is not legally identical to capacity, but a delusional reading of medication as poison destroys the ability to appreciate consequences.

TRAP Two examiner favourites the evidence used here does not settle: the section number governing supported admission with its permitted duration, the number of examiners and their professional categories; and the contrast between psychologically mediated poor insight, meaning denial, rationalisation and external attribution, and anosognosia, meaning unawareness attributed to a neurocognitive or organic mechanism. Verify both beforehand.

HOLD THIS

Insight is graded, judgement is not. Grades 1 to 6 for insight, intact or impaired for judgement, and ward behaviour overrides the hypothetical.

WHAT EARNS THE MARKS

- **Insight offered as three dimensions**: a patient can accept illness, refuse treatment and still relabel nothing.
- **The fifth elicitation question**: relabelling is what separates grade 5 from grade 4, and agreement alone is not insight.
- **Ward behaviour placed above the hypothetical**: observed conduct outweighs a textbook answer about a stamped letter.
- **The insight paradox raised**: rising awareness can bring shame and internalised stigma, so psychoeducation is paired with stigma work.

SEE ALSO 01 Order and identifying data 13 Possession and content 21 Suicide and violence 28 Management

Physical examination and risk

17 · Why you examine, and the general physical examination

EXAMINATION AND RISK · TOPIC 17

Why does a psychiatrist examine the patient physically, and what did you find?

LONG CASE, PHYSICAL EXAMINATION; SHORT CASE ON GENERAL SIGNS

THE SHAPE OF THE ANSWER

1 The quantitative case	Mortality, misattribution and documentation figures, in that order
2 The five goals	Organic separation, comorbidity, baseline, drug safety, comparison
3 Setting and chaperone	Privacy, the kit, and a chaperone matched to the patient's sex
4 The general survey	Anthropometry, pallor, skin, hands, mouth and nodes
5 Documentation	Specific positives and negatives, with a plan for what you could not do

The physical examination is yours, not the referring doctor's. Examiners open here by asking why a psychiatrist examines at all, and **the answer is quantitative**.

FINDING	FIGURE	THE ARGUMENT IT MAKES
Mortality in mental disorder	Age-adjusted annual all-cause mortality 2 to 4 times the general community	**Physical disease is more prevalent, not less, in your patients**
Misattribution	Between 6 percent and 20 percent of patients with primary physical disease are mistakenly diagnosed with a psychiatric illness	**Relying on another's examination perpetuates the error**
Documentation in practice	Central nervous system examination recorded in under 60 percent of admissions; 23.4 percent of inpatients received none	**The omission is systemic, which is why a checklist improves it**
Delay	Mean delay of 62 hours, extending to 612 hours	**Why the standard is examination on admission or within 24 hours**

Five goals justify it. Separating organic disorders from functional illness; detecting untreated comorbidity; establishing baseline parameters before psychotropics; guiding safe drug selection; and giving a standard for comparison if the patient deteriorates.

Patients in acute mental disturbance **under-report serious somatic symptoms even with life-threatening disease**, which is why the examination cannot be delegated.

- Concept. **Setting and kit.** Privacy, quiet, warmth, light and a couch, with stethoscope, sphygmomanometer, ophthalmoscope, tendon hammer, tuning fork, pen torch, tape measure, tongue depressor and urine dipsticks.
- Key point. **Anything beyond observation, pulse and blood pressure is done with a chaperone matched to the patient's sex.** It protects the patient from harm and the clinician from allegation, and it is equally advisable for a female psychiatrist examining a male patient.

Where agitation prevents a full examination, **document the reason and the plan to re-examine within 24 hours** rather than leaving a blank.

- Concept. **The general examination reads self-care, endocrine state, nutrition and substance use off the surface.**
- Concept. **Anthropometry**: weight, height, body mass index and waist circumference. Loss suggests depressive anorexia, psychotic self-neglect or occult malignancy, and the same chart later tracks drug-induced gain.
- Concept. **Pallor and cyanosis**: palmar and conjunctival pallor for nutritional anaemia, a lemon tint for vitamin B12 deficiency which itself causes cognitive and affective disturbance, central cyanosis for cardiopulmonary compromise or aspiration in the elderly and in neuroleptic-induced dysphagia.
- Concept. **Skin**: a dull sallow complexion in depression, facial plethora in chronic alcohol misuse, jaundice from alcohol, hepatitis or drug-induced cholestasis, a malar rash for lupus with neuropsychiatric features.
- Concept. **Hands**: a warm moist handshake in thyrotoxicosis and anxiety, cold clammy hands in panic disorder, needle tracks and thrombosed veins in the antecubital fossae, and splinter haemorrhages with fever in an injecting user meaning endocarditis.
- Concept. The hands also carry the liver signs: **palmar erythema, Dupuytren contracture, spider naevi and clubbing** for chronic liver disease.

- **Concept. Mouth and nodes**: caries and periodontal disease as the signature of neglect, cervical lymphadenopathy for tuberculosis and opportunistic infection, a supraclavicular node in a smoker for metastatic carcinoma.

TRAP Writing "NAD" across the examination. Nothing abnormal detected, without specific positives and negatives, reads as perfunctory and has no clinical or legal value; documented specific negatives are what defend you.

HOLD THIS

You examine because between 6 percent and 20 percent of physical disease presenting as psychiatric illness is misdiagnosed, and mortality runs 2 to 4 times higher.

WHAT EARNS THE MARKS

- **Three figures given before any opinion**: mortality 2 to 4 times higher, 6 to 20 percent misattribution, and a mean delay of 62 hours.
- **The examination claimed as your own**: relying on another clinician's note perpetuates the original error.
- **A chaperone stated as routine**: it protects the patient and defends the clinician.
- **Specific negatives written out**: "nothing abnormal detected" has no clinical or legal value.

SEE ALSO 18 Vitals and systems 19 Neurological screen 25 Differential hierarchy 27 Investigations

18 · Vital signs and the systemic examination

EXAMINATION AND RISK · TOPIC 18

The pulse is 118 and the patient is febrile. What is your differential and what do you do?

LONG CASE, PHYSICAL EXAMINATION; SHORT CASE ON AN EMERGENCY PATTERN

THE SHAPE OF THE ANSWER

1 Read the pattern	The pattern carries the diagnosis, not the single value
2 Each abnormality with its action	Tachycardia, bradycardia, rhythm, hypertension, postural drop
3 The emergency pattern	Labile pressure with pyrexia, then rigidity or abdominal pain
4 System by system	Cardiovascular, respiratory, gastrointestinal, each with its sequence
5 The neglected parts	Remove the socks, look in the mouth, screen where indicated

Temperature, pulse, blood pressure and respiratory rate are recorded at admission, and **the pattern, not the single value, carries the diagnosis.**

FINDING	DIFFERENTIAL	THE ACTION IT FORCES
Resting tachycardia	Alcohol or sedative withdrawal, thyrotoxicosis, anxiety, anti-cholinergic toxicity, clozapine myocarditis, infection, neuroleptic malignant syndrome	**Do not write it off as anxiety; it is the sign most often dismissed**

FINDING	DIFFERENTIAL	THE ACTION IT FORCES
Bradycardia	Anorexia nervosa, hypothyroidism, raised intracranial pressure, beta-blocker, lithium or acetylcholinesterase inhibitor toxicity	**Review the drug chart before the psychiatry**
Irregular rhythm	Atrial fibrillation as a source of cerebral emboli and vascular dementia	**Anticoagulation and rate control join the psychiatric plan**
Sustained hypertension	Arousal at admission, then alcohol, thyroid disease, clozapine, high-dose venlafaxine, renal disease including lithium nephropathy	**Serial readings over days before calling it essential; dipstick for proteinuria**
Postural drop	Autonomic failure in diabetic neuropathy or Lewy body dementia; chlorpromazine and tricyclics; resting hypotension in anorexia nervosa and Addison disease	**Measure supine and standing, record both**
Labile pressure with pyrexia	Neuroleptic malignant syndrome with rigidity and raised creatine kinase, or acute intermittent porphyria with abdominal pain and urine darkening on standing	**The emergency pattern of the whole table**

Phaeochromocytoma is rare, mimics severe panic disorder and may drive self-medication with alcohol; screening starts with urine glucose and confirmation is by urinary adrenaline and noradrenaline assay over 24 hours.

SYSTEM	SEQUENCE	SIGNS THAT CHANGE THE PSYCHIATRIC PLAN
Cardiovascular	Patient at 45 degrees for venous pressure, apex beat leaning forward, sounds and murmurs, peripheral pulses and bruits	**Raised venous pressure for failure; mitral stenosis predisposing to embolic vascular dementia; a murmur with fever in an injecting user; absent foot pulses for neglected diabetic or vascular disease; carotid bruits**
Respiratory	Expansion, tracheal position, percussion, auscultation	**Dullness, bronchial breathing or crackles for pneumonia, chronic obstructive pulmonary disease or tuberculosis in the self-neglected; haemoptysis in a smoker warrants a radiograph**
Gastrointestinal	Flat unless orthopnoeic, contour, palpation, shifting dullness, bowel sounds	**Caput medusae for portal hypertension; shifting flank dullness for ascites; tender hepatomegaly in steatohepatitis against a hard shrunken nodular liver in cirrhosis**

CLINICAL ANCHOR

Remove the socks and look in the mouth. Omitting oral inspection misses a dental abscess the patient has been calling facial pain, and leaving the socks on misses gangrene and diabetic ulceration in a patient who has not complained of either.

Breast examination is recommended as a screen in female inpatients over 65 years, and audit recorded a compliance rate of zero, which is the scale of the omission being discussed.

RULE **Iatrogenic parkinsonism against idiopathic Parkinson disease.** Drug-induced disease is symmetrical with coarse resting tremor and bilateral lead-pipe or cogwheel rigidity after a dopamine blocker; idiopathic disease starts asymmetrically with a pill-rolling tremor, and Lewy body dementia adds fluctuating cognition, visual hallucinations and autonomic instability.

HOLD THIS

Pyrexia, tachycardia and swinging blood pressure is a syndrome, not three observations. Rigidity with raised creatine kinase points to neuroleptic malignant syndrome; abdominal pain with darkening urine points to porphyria.

WHAT EARNS THE MARKS

- **Tachycardia not written off as anxiety**: it is the sign most often dismissed, and it sits in front of withdrawal, myocarditis and sepsis.
- **An action attached to each abnormal vital sign**: serial readings, a drug chart review, a dipstick, a supine and standing pair.
- **The emergency triad recognised as one syndrome**: pyrexia with labile pressure and rigidity changes the plan immediately.
- **Iatrogenic parkinsonism separated from idiopathic**: symmetry and a coarse resting tremor after a dopamine blocker, against asymmetric pill-rolling onset.

SEE ALSO 17 Why you examine 19 Neurological screen 27 Investigations 34 Alcohol and opioid

19 · The rapid neurological screen, organic against functional

EXAMINATION AND RISK · TOPIC 19

Show me a neurological screen you could finish in this long case, and how you separate organic from functional weakness.

LONG CASE, PHYSICAL EXAMINATION; SHORT CASE ON FUNCTIONAL SIGNS

THE SHAPE OF THE ANSWER

1 Purpose and overlap	Anatomical localisation, overlapping the mental state at four points
2 Record consciousness	Glasgow Coma Scale at admission, three subscales, 3 to 15
3 The rapid screen	Seventeen steps in about 3 minutes, in a fixed order
4 Organic against functional	Hoover sign, sensory boundaries, tone, reflexes
5 The mute patient	Examined rather than excused, with a structured catatonia assessment

The purpose of the neurological examination is **anatomical localisation**, and it overlaps the mental state at conscious level, orientation, memory and speech.

- Key point. **Record the admission level of consciousness in each case with the Glasgow Coma Scale**, which gives a baseline if the state later changes.

The Glasgow Coma Scale has three subscales, eye opening, verbal performance and motor response, with a total score range of 3 to 15. A documented level of consciousness was present in only 40 percent of patients in baseline inpatient audits.

- Concept. **Speech assessment separates dysphasia from dysarthria.** Dysphasia is a localising cortical deficit for which hand dominance must be established; dysarthria follows neuroleptics, high-dose anticonvulsants, cerebellar disease, stroke, multiple sclerosis or tardive dyskinesia.

A full neurological examination takes about 40 minutes, which the long case does not allow. **The rapid screen comprises 17 steps and can be completed in about 3 minutes.**

STEPS	MANOEUVRE	PATHWAY AND INTERPRETATION
1 and 2	Romberg test: feet together, then eyes closed	Dorsal columns; marked wobbling without ataxia that vanishes on tandem walking suggests a non-organic difficulty

STEPS	MANOEUVRE	PATHWAY AND INTERPRETATION
3 and 4	Tandem walk forwards and backwards, then tiptoes and heels	**Midline cerebellar function, then ankle plantarflexion and dorsiflexion power**
5 and 6	Arms outstretched, palms up, eyes closed	**Pronation with drift indicates a contralateral corticospinal lesion; drift without pronation suggests parietal neglect**
7 to 9	Finger to opposite finger then nose; rapid tapping; alternating patting of back and palm	**Light touch, coordination, cerebellar dysmetria and dysdiadochokinesia, extrapyramidal bradykinesia**
10 and 11	Screw the eyes tight, then show the teeth and grin	**Facial power and symmetry, upper against lower motor neuron; pupils and Horner syndrome**
12 and 13	Protrude the tongue and wiggle it side to side	**Hypoglossal power, and the spastic tongue of pseudobulbar palsy**
14 and 15	Visual fields by wiggling fingers, then bilateral simultaneous movement; follow a finger in an H pattern	**Hemianopia and parietal sensory inattention; then ocular motility, nystagmus and gaze palsies**
16 and 17	Deep tendon reflexes in relaxed limbs; fundoscopy	**Symmetry, hyperreflexia and clonus against hyporeflexia; papilloedema, optic atrophy, hypertensive and diabetic retinopathy**

Extrapyramidal signs are sought explicitly, meaning resting tremor, lead-pipe or cogwheel rigidity, bradykinesia and festinant gait, because spontaneous abnormal involuntary movements in a neuroleptic-naive patient predict sensitivity to drug-induced extrapyramidal effects and to tardive dyskinesia later.

ORGANIC AGAINST FUNCTIONAL, AT THE BEDSIDE

Hoover sign. With the patient supine, cup a hand under the unaffected heel and ask them to lift the affected leg against resistance: in functional weakness no downward thrust is felt. Then ask them to lift the normal leg against resistance and feel involuntary downward counter-pressure under the supposedly paralysed heel, confirming intact corticospinal pathways.

Sensory boundaries. Organic loss follows dermatomes or nerve territories; functional loss shows sharp midline cut-offs or glove-and-stocking lines without transition zones. **Tone.** Spasticity is velocity-dependent with clasp-knife release in antigravity muscles; rigidity is velocity-independent and uniform; paratonia, or gegenhalten, is variable resistance rising with the force you apply, marking diffuse frontal dysfunction.

Reflexes. Hyperreflexia with clonus and an extensor plantar response cannot be voluntarily simulated; sluggish reflexes with delayed relaxation occur in hypothyroidism.

- Key point. **The uncooperative, mute or catatonic patient is examined, not excused.**
- Concept. **Start with gross physiological stability.** A patient standing and shouting without breathlessness has a patent airway, adequate gross respiratory function and no severe hypotension.
- Concept. Then **observational inspection** of clothing, self-care, dentition, pupils, goitre, facial masking, tremor, sweating and wasting.
- Concept. Then the structured catatonia assessment: **general reaction and posture**; facial movement and gaze, including resistance to eye opening; and motor responses to command, looking for automatic obedience, ambitendency, echopraxia and negativism.
- Concept. Then **muscular phenomena on gentle passive movement**, meaning waxy flexibility, catalepsy, lead-pipe rigidity, gegenhalten, mitgehen and mitmachen.
- Key point. Close on the **autonomic and excretory check** for urinary retention, faecal impaction or incontinence, which are emergencies in catatonic stupor.

TRAP Saying the examination was impossible because the patient was mute. Demonstrate the observational examination covering consciousness, airway, vital signs, pupils, tone and bladder fullness, document the behavioural reason for what you could not complete, and state the plan to re-examine within 24 hours.

HOLD THIS

Seventeen steps in about 3 minutes, and the Glasgow Coma Scale recorded from 3 to 15 at admission. A screen you can name is a screen you can defend.

WHAT EARNS THE MARKS

- **A screen with a step count and a time**: 17 steps in about 3 minutes is a method, not an intention.
- **The Hoover sign demonstrated**: involuntary counter-pressure under the paralysed heel proves the corticospinal pathway is intact.
- **Signs that cannot be simulated named**: clonus and an extensor plantar response settle the organic question.
- **The mute patient still examined**: an observational examination plus a documented reason beats "examination was impossible".

SEE ALSO 10 Psychomotor and speech 15 Cognitive examination 18 Vitals and systems 36 Dissociation and borderline

20 · Risk: from prediction to prevention, and the four factor classes

EXAMINATION AND RISK · TOPIC 20

Can you predict whether this patient will attempt suicide, and if not, what are you doing instead?

LONG CASE, RISK ASSESSMENT; VIVA TABLE ON RISK METHOD

THE SHAPE OF THE ANSWER

1 **Define risk assessment**	Identifying hazards in order to reduce the probability of occurrence
2 **The base-rate argument**	Low base rate means false positives, so prediction is the wrong goal
3 **Foreseeability as the standard**	Use the data available at evaluation, plan, and follow through
4 **Three approaches compared**	Unstructured, actuarial, structured professional judgement
5 **The four factor classes**	Static, stable, dynamic, future, each with what it is used for

Risk assessment is the process of identifying and studying hazards in order to reduce the probability of their occurrence, and **the shift to state plainly is from prediction to prevention**.

- **Key point.** Suicide and severe interpersonal violence are **low base-rate events**, so even an accurate model produces an unacceptable rate of false positives, misallocating scarce resources and restricting autonomy without benefit.

- **RULE** **Foreseeability, not predictability, is the standard of care.** Predicting an individual suicide is not achievable because of the base rate; using the clinical data available at the time of evaluation to anticipate plausible hazards, plan treatment and follow through is what is required and audited.

APPROACH	METHOD	STRENGTH AND LIMITATION
Unstructured clinical judgement	Subjective, intuitive evaluation from experience	**Flexible and individualised, but low reliability, poor accuracy, open to bias; not sustainable alone**
Actuarial assessment	Algorithmic scoring producing a numerical probability	**High inter-rater reliability, but rests on static and stable factors, insensitive to change, silent on imminence and context**
Structured professional judgement	Empirically supported factors integrated with an individualised formulation	**The current standard: captures dynamic and future factors, transparent for governance, guides prevention. An approach, not an instrument, and it structures judgement rather than replacing it**

CLASS	EXAMPLES	WHAT IT IS USED FOR
Static	Family history of suicide, childhood abuse, past self-harm, seriousness of previous attempts, age, sex, marital status	**Fixes the lifelong baseline propensity; cannot be treated**
Stable	Personality disorder or maladaptive traits, chronic substance use disorder, persistent deficit states	**Shapes long-term supportive and maintenance care**
Dynamic	Active ideation and intent, hopelessness, agitation, active psychotic symptoms, intoxication or withdrawal, non-adherence, acute loss	**The targets of acute treatment, and what converts baseline vulnerability into an imminent crisis**
Future	Impending discharge, access to a preferred method, anticipated stressors, anticipated non-response, pending legal proceedings	**Drives proactive safety planning and transitional care**

Table: under fluid vulnerability theory, static and stable factors set an enduring baseline on which dynamic episodes fluctuate, and multiple attempters carry a lower threshold for activating a crisis under stress.

HOLD THIS

Static sets the floor, dynamic moves the ceiling, future tells you when to worry. Structured professional judgement holds all three together.

WHAT EARNS THE MARKS

- **Prevention offered in place of prediction**: the base-rate argument is the reason, and it is worth saying out loud.
- **Foreseeability named as the standard of care**: it is what is audited, and it is achievable where prediction is not.
- **Structured professional judgement identified as an approach**: it structures judgement rather than replacing it, and it is not an instrument.
- **The four classes used functionally**: static fixes the baseline, dynamic supplies the treatment targets, future drives the safety plan.

SEE ALSO 16 Insight and judgement 21 Suicide and violence 23 Biopsychosocial and 5 P 28 Management

21 · Suicide, violence, self-neglect, and how to present risk

EXAMINATION AND RISK · TOPIC 21

Present this patient's risk, and tell me what you will do about each part of it.

LONG CASE, RISK PRESENTATION; OSCE RISK ASSESSMENT STATION

THE SHAPE OF THE ANSWER

1 **Ask properly**	Normalise, then work the timeline from lifetime to this crisis
2 **Assess intent**	Purpose, preparatory acts, self-rated wish to die, practical execution
3 **Violence and the wider field**	Historical, clinical and risk management scales, then self-neglect

4 Iatrogenic hazards	Prescribed overdose, akathisia, abrupt stopping, discharge transition
5 Present it in five beats	Static, dynamic, protective, formulation narrative, then the plan

A denied ideation is not an absent risk. Patients with severe hopelessness who have resolved to end their life **may conceal the plan precisely to prevent interference**.

Passive ideation, meaning wishing not to wake up, **carries risk comparable to active ideation**.

The chronological approach to suicide events gives the sequence. Normalise first, by validating that severe distress commonly brings thoughts of ending one's life. Then work the timeline from lifetime ideation and attempts, narrowing to the preceding 12 months, then the past month, then the presenting crisis, which builds rapport and reduces withholding.

- **Key point. Distinguish three acts.** A suicide threat is a verbal declaration without self-harm; instrumental behaviour is an act for an external goal without intent to die; a genuine attempt is self-directed injury with suicidal intent.

Intent is assessed through four structured inquiries: the purpose of the act, meaning why the patient wishes to die; preparatory acts, such as finalising a will, giving away possessions or writing a note; the patient's own rating of the wish to die on a scale of 1 to 10; and practical execution, meaning rehearsal, precautions against discovery and availability of lethal means.

MNEMONIC

IS PATH WARM

Consensus warning signs of near-term crisis: **I**deation with desire, plan or means; **S**ubstance abuse escalating; **P**urposelessness; **A**nger and desire for vengeance; **T**rapped, no viable exit from the pain; **H**opelessness about self, others and future; **W**ithdrawal from supports; **A**nxiety, agitation, panic or acute insomnia; **R**ecklessness; **M**ood changes that are rapid and dramatic.

These are proximal and acute, so they are intervention targets rather than score items.

INDIA NOTE

India accounts for approximately 170,000 officially reported suicides annually, and verbal autopsy work suggests actual rates may be at least 6 times higher because of underreporting linked to historical legal sanction.

Western psychological autopsy studies find psychiatric morbidity in nearly 90 percent of decedents with major depression in approximately 50 percent, whereas Indian studies identify major psychiatric disorders in 25 percent to 35 percent of cases, with acute social crises, interpersonal discord and financial distress carrying substantial weight.

Rao and Nammalvar showed that familial obligation towards spouse and parents, economic duty towards children, and religious or moral prohibition act as significant protective buffers against acting on suicidal urges, the protective factor a Western-trained candidate omits.

SCALE OF THE VIOLENCE-RISK INSTRUMENT	COMPOSITION	WHAT IT CAPTURES
Historical	10 items	**Static: history of violence, young age at first incident, relationship instability, employment problems, substance misuse, major mental disorder, personality disorder, early maladjustment, prior supervision failure**
Clinical	5 items	**Current dynamic: lack of insight, violent ideation or intent, active symptoms, instability, poor treatment response**
Risk management	5 items	**Future dynamic: lack of services, unstable living situation, lack of personal support, nonadherence, stress and coping deficits**

A systematic review of 12 studies examined its longitudinal properties across 3 dimensions of sensitivity. Internally, clinical, risk management and total scores show small decreases during inpatient forensic treatment while historical scores do not change.

Relatively, the dynamic scores correlate with other dynamic tools. Externally, **there was little evidence that reductions predict lower subsequent violent recidivism.**

- **Trap.** Reading a falling dynamic score as safety. **It demonstrates therapeutic progress and cannot be read as an assurance about future violence.**
- **Concept. Self-neglect is the inability to sustain basic nutrition, hygiene or medical self-care** to the point of physical deterioration.
- **Concept. Vulnerability is susceptibility to physical, sexual or financial exploitation** because of psychosis, mania or cognitive impairment.
- **Trap.** Reporting suicide and violence and stopping there. **A candidate who omits self-neglect and vulnerability has assessed half the risk.**

IATROGENIC HAZARD	FIGURE	WHAT IT CHANGES
Psychotropic self-poisoning	Over one third, 33.3 percent, of suicides among psychiatric patients within 12 months of contact involve overdose of prescribed psychotropic medication	**Limit quantities dispensed and supervise supply through a family member**
Akathisia and abrupt discontinuation	Antipsychotic-induced restlessness raises suicide risk; withdrawal of maintenance mood stabilisers, including lithium, precipitates sharp elevations in suicidal behaviour	**Look for akathisia at every review, and plan stopping rather than stopping abruptly**

IATROGENIC HAZARD	FIGURE	WHAT IT CHANGES
High-risk transitions	16 percent of suicides under mental health services occur on inpatient wards, and 30 percent of community suicides within 3 months of discharge, with the initial 28 days the peak	**Admission is not a protective bubble, and the discharge plan is a risk intervention**

THE SIX QUESTIONS A RISK FORMULATION ANSWERS

A formulation is a narrative, not the words low, moderate or high. How serious is the risk, meaning severity and lethality? How immediate, over hours, days or weeks? Specific or general, targeted at a named person or diffuse? How volatile, meaning how fast it moves with triggers, substances or affective dysregulation?

What are the signs of increasing risk that alert carers, such as sleeplessness, irritability, withdrawal or medication refusal? And which treatment and management plan will reduce it?

- **Key point.** **Presenting risk follows five beats.** Beat one is the static baseline: age, sex, family history, past attempts or violent acts.
- **Concept.** Beat two is the **current dynamic state**: ideation, intent, affect, psychotic symptoms, agitation and substance use.
- **Concept.** Beat three is the **protective buffers**: family support, rapport, reasons for living and socio-cultural duties.
- **Concept.** Beat four is **the formulation narrative**, answering the six questions above.
- **Concept.** Beat five is the plan, starting with **setting**: a named observation level, or supervised outpatient care.
- **Key point.** The plan then carries **pharmacotherapy including lithium and clozapine for their anti-suicidal properties**, with active management of akathisia.
- **Concept.** The plan closes on **psychological work targeting hopelessness and problem-solving**, and environmental safeguards restricting lethal means and involving the family.

TRAP Two items asked here are not settled in the evidence used for this note: the structured suicide-severity instrument expected alongside the interview, with its ideation severity, ideation intensity and behaviour subscales, and the statutory material on decriminalisation of attempted suicide and on supported admission, meaning section numbers, the number of professionals required and the initial duration. Verify each from the manual and the bare Act; a severity rating structures the inquiry but carries no predictive cut-off.

RULE **Scale scores do not substitute for a formulation.** Risk instruments have no predictive cut-off, so they organise the inquiry and feed the narrative; the mark is given for the narrative.

HOLD THIS

Risk is foreseeable, not predictable, and it is presented as a narrative in six answers. Static, dynamic, protective, formulation, plan.

WHAT EARNS THE MARKS

- **Denial recorded as a finding, not a clearance**: concealment protects the plan, and passive ideation carries comparable risk.
- **The Indian protective factors named**: familial obligation, economic duty towards children, and religious or moral prohibition.
- **An iatrogenic hazard raised unprompted**: a third of suicides within 12 months of contact involve prescribed psychotropic overdose.
- **The narrative given in place of a label**: low, moderate or high is not a formulation, and the mark is for the narrative.

SEE ALSO 11 Mood and affect 20 Risk factor classes 28 Management 32 Psychotic depression, OCD

Formulation to prognosis

22 · Summary against formulation, and what a formulation must do

FORMULATION · TOPIC 22

Give me your formulation. Not the summary, the formulation.

LONG CASE, FORMULATION; VIVA TABLE ON CASE WRITE-UP

THE SHAPE OF THE ANSWER

1 Separate the two	A summary condenses findings, a formulation explains mechanism
2 The three questions	What is happening, how did this person reach it, how do we intervene
3 The rules of form	Scientific terminology, 10 to 20 sentences, no negative history
4 Components in order	Baseline, phenomenology, aetiology, conclusion, therapeutic direction
5 Delivery	About 5 minutes, spoken tentatively as a testable hypothesis

Confusing the two is an immediate tactical error, **because they are asked for separately**.

- Concept. **A case summary is a descriptive, cross-sectional condensation** of the history, the positive and pertinent negative symptoms, the mental state and the physical findings, ending in a provisional categorical diagnosis.
- Key point. **A diagnostic formulation is an explanatory, hypothesis-generating synthesis** that explains the mechanism of the illness.
- Concept. It answers three questions: **what is happening with this individual, how did this person reach this state of distress at this specific point in time**, and how can the team intervene to relieve the suffering and restore functioning.

The consensus proforma sets rules examiners apply directly. Scientific medical terminology rather than vernacular phrasing; a length of typically 10 to 20 sentences; and negative history omitted, with any feature not mentioned presumed absent.

Components go in order. The identifying baseline of age, sex, marital status, education, occupation and family background; the phenomenological synthesis of positive psychopathology; the aetiological analysis using the biopsychosocial model or the 5 P framework.

The order closes on **the multi-axial conclusion with differentials and comorbidity**, and the therapeutic direction including the setting of care.

Delivery matters. The verbal formulation runs to about 5 minutes and is spoken tentatively, the findings suggest, or it is probable that.

- Key point. **A formulation presented as absolute fact invites the examiner to dismantle it**, while one presented as a testable hypothesis invites them to test it with you.

FEATURE	CASE SUMMARY	DIAGNOSTIC FORMULATION
Function	Condenses what was found	**Explains why it happened to this person now**
Negative history	Included	**Excluded, and absence presumed for anything unmentioned**
Language	May carry the patient's phrasing	**Scientific terminology throughout**
Ends with	A provisional categorical diagnosis	**Differentials, comorbidity and a broad biopsychosocial direction**
Epistemic status	A record	**A hypothesis, framed tentatively and open to revision**

TRAP Starting the formulation by repeating the history of presenting illness in chronological order. The examiner will stop you: a formulation presents functional interactions, not a second pass through the timeline.

HOLD THIS

A summary condenses, a formulation explains, in 10 to 20 sentences and no negative history. If it does not answer why this person and why now, it is still a summary.

WHAT EARNS THE MARKS

- **The two products named as different tasks**: one is a record, the other a hypothesis, and examiners ask for them separately.
- **Why this person and why now answered**: a formulation that skips those two clauses is a summary in disguise.
- **Tentative phrasing used deliberately**: it invites the examiner to test the hypothesis with you.
- **Negative history left out**: anything unmentioned is presumed absent, which is the rule that makes the omission safe.

SEE ALSO 04 Negative and treatment history 23 Biopsychosocial and 5 P 24 ICD-11 and DSM-5-TR 29 Prognosis

23 · The biopsychosocial model, the 5 P grid, and its critics

FORMULATION · TOPIC 23

Set out the aetiology on a biopsychosocial grid, and tell me what is wrong with the model.

LONG CASE, FORMULATION; VIVA TABLE ON MODELS OF ILLNESS

THE SHAPE OF THE ANSWER

1 Origin and target	Meyer then Engel, aimed at two kinds of reductionism
2 Cross it with the P framework	Weerasekera's four categories, plus the presenting row
3 Fill the grid	Three domains against each category, with this patient's material
4 Name the treatment targets	Perpetuating factors, because the other two are historical
5 The critiques	No weighting, additive, superficiality, social neglect

Adolf Meyer promoted the psychobiological approach in 1948, and **George Engel developed and operationalised the biopsychosocial model in 1977 and 1980 against reductionism.**

- **Concept. Reductionism takes two forms.** Medical materialism, where biology alone explains psychiatric illness, and psychological or social dogmatism, where intrapsychic process or environment is held to be the sole cause.
- **Key point.** Left alone the model becomes an inventory, so **it is crossed with the 4 P framework systematised by Weerasekera in 1993**: predisposing, precipitating, perpetuating and protective factors. The 5 P variant adds the presenting problem.
- **Trap.** Presenting the five-row grid as published. **The published grid crosses three domains with the four functional categories**, so the presenting row below is a revision aid rather than a published framework.

CATEGORY AND QUESTION	BIOLOGICAL	PSYCHOLOGICAL	SOCIAL
Presenting **What is the active problem?**	Physical complaints, somatic distress, medical disorder	**Subjective distress with a scale anchor, such as a GAD-7 of 16 or a PHQ-9 of 17**	Impairment at work or school, interpersonal strain
Predisposing **Why vulnerable?**	Genetic loading, family history, difficult temperament, perinatal insult	**Insecure attachment, adverse childhood experiences, trauma, maladaptive schemas**	Poverty, disadvantage, marginalisation, household dysfunction
Precipitating **Why now?**	Acute illness, medication discontinuation, intoxication, endocrine shift	**Interpersonal loss, bereavement, threat to self-worth, reactivated trauma**	Unemployment, academic failure, legal problems, relocation, separation
Perpetuating **Why does it persist?**	Untreated illness, chronic insomnia, continuing substance use, non-adherence	**Avoidance, rumination, catastrophising, secondary gain, immature defences**	Isolation, family accommodation of avoidance, financial distress
Protective **What buffers?**	Good physical health, no substance misuse, known treatment responsiveness	**Psychological mindedness, cognitive flexibility, motivation, adaptive coping**	Warm family support, stable employment, community or spiritual ties

- **RULE** **Perpetuating factors are the primary targets of treatment**, because predisposing and precipitating factors are historical. If family accommodation maintains agoraphobic avoidance the intervention is family psychoeducation; if rumination maintains depression it is cognitive restructuring. A sub-therapeutic dose is perpetuating, and chronic non-adherence in a patient unmedicated for years is perpetuating, while the abrupt stoppage that triggered this relapse is the precipitant.

- **Concept. The critiques are asked as a set.** No weighting rules, so the model is silent on which domain takes precedence, an objection associated with Ghaemi.
- **Concept. Additive rather than integrative**, juxtaposing domains without a unified causal theory, which is McLaren's argument.
- **Concept. An invitation to superficiality**, where clinicians pay lip service to domains outside their interest, noted by Jellinek and McDermott.
- **Key point. Neglect of the social domain**, noted by Grunebaum, despite social distress being what usually brings the patient in.

PERSPECTIVE	EXPLANATORY CONCEPT	GOAL, RISK, AND WHAT IT REQUIRES OF THE PATIENT
Disease	What the patient has: broken biological machinery	**Cure or symptom reduction; risk is medication toxicity; requires adherence**
Dimension	What the patient is: a position on a continuum	**Guidance and psychoeducation; risk is paternalism; requires identified strengths**
Behaviour	What the patient does: goal-directed maladaptive action	**Interruption and retraining; risk is stigmatisation; requires the will to change**
Life story	What the patient experiences: a narrative of events	**Re-scripting and insight; risk is premature interpretation; requires psychological insight**

Table: McHugh and Slavney's four perspectives, offered in 1998 as pluralism rather than eclecticism, refining distinct methods instead of mixing them loosely.

- **Concept. Cultural formulation is the fifth element, in four parts.** Cultural identity, including languages and involvement with the culture of origin against the host society; and cultural explanations of illness, meaning idioms of distress.
- **Concept.** The other two parts are **cultural factors in the psychosocial environment**, meaning culturally specific supports and stressors, and cultural elements of the clinician-patient relationship, meaning differences in status, belief and communication style.

CLINICAL ANCHOR

Formulations are communication instruments too. In an audit of 150 psychiatric assessment letters from an outpatient service, 94 percent contained no case formulation, depriving the receiving clinician of the diagnostic rationale entirely. A consultation letter states the rationale, synthesises the explanation, documents suicide and violence risk, outlines the treatment steps, and exercises discretion about sensitive disclosures.

TRAP Writing "family is supportive" as a protective factor. Unless you say how the support buffers decompensation, who provides it and what it can be relied on to do, a protective factor is clinically useless and scores nothing.

HOLD THIS

The grid organises, the narrative explains, and perpetuating factors are where treatment bites. Understanding grasps meaning from within the patient's experience; explanation applies a theory from outside.

WHAT EARNS THE MARKS

- **The grid filled with this patient's material**: a blank grid recited from memory is an inventory, not a formulation.
- **Perpetuating factors identified as the targets**: predisposing and precipitating factors are historical and cannot be treated.
- **A protective factor specified**: "family is supportive" scores nothing without who, how, and what it can be relied on to do.
- **One critique named with its author**: it shows the model is held critically rather than repeated.

SEE ALSO 20 Risk factor classes 22 Summary and formulation 28 Management 29 Prognosis

24 · ICD-11 against DSM-5-TR, and the multiaxial habit

FORMULATION · TOPIC 24

Which classification are you coding in, and how does the other one differ?

LONG CASE, DIAGNOSIS; VIVA TABLE ON CLASSIFICATION

THE SHAPE OF THE ANSWER

1 Declare the system	India is a member state, so code in the global standard and say so
2 Publisher and purpose	Global public health reporting against national diagnostic standardisation
3 The structural contrasts	Thresholds, personality, substance use, complex trauma
4 Disorder-level divergences	Trauma, grief, childhood irritability, sexual behaviour, gender
5 The multiaxial habit	Say which system you are using before you list axes

Diagnose in the World Health Organization system and say so. India is a member state, ICD-11 is the global standard for recording and reporting conditions, and national statistics and hospital records follow it.

- Key number. **The 72nd World Health Assembly adopted it in May 2019**, and it took effect for morbidity and mortality reporting in January 2022.
- Concept. **DSM-5 was published by the American Psychiatric Association in May 2013** with its text revision in 2022. It remains a national classification produced by one professional body, valuable for operational criteria and research.

FEATURE	ICD-11	DSM-5-TR
Publisher and purpose	World Health Organization; global utility and public health reporting	**American Psychiatric Association; diagnosis and research standardisation**

FEATURE	ICD-11	DSM-5-TR
Where mental disorders sit	Chapter 6, mental, behavioural and neurodevelopmental disorders, codes beginning with 6, the range starting at 6A00	**A dedicated manual with no independent statistical coding, mapped to ICD codes for billing**
Diagnostic philosophy	Descriptive requirements with flexible thresholds, qualitative terms such as several symptoms	**Polythetic criteria with strict numerical counts and explicit durations**
Personality disorders	Fully dimensional: severity plus trait domain qualifiers	**The traditional 10 categorical types retained in the official section**
Substance use	Harmful pattern and dependence kept separate	**A single unified substance use disorder continuum**
Complex post-traumatic stress disorder	A distinct official diagnosis	**Not recognised as a distinct entity**

- **Key point.** **Personality disorder is the most radical divergence.** ICD-11 eliminated the 10 categorical diagnoses and grades pathology by severity as mild, moderate or severe, determined by dysfunction in self-functioning, meaning identity and self-direction, and in interpersonal functioning.
- **Trap.** Calling personality difficulty a mild personality disorder. **It is a non-disease classification coded among factors influencing health status**, sitting below the disorder threshold.
- **Concept.** **Prominent facets are described with 5 trait domains**: Negative Affectivity, Detachment, Dissociality, Disinhibition and Anankastia. A borderline pattern qualifier is retained to preserve clinical continuity and access to treatment.
- **Concept.** **DSM-5-TR keeps the general criteria and the 10 types officially**, with the alternative dimensional model confined to the research section.

DISORDER AREA	ICD-11	DSM-5-TR
Post-traumatic stress disorder	Tightened to 3 core clusters: re-experiencing in the present, avoidance of reminders, persistent sense of heightened current threat	**Includes non-specific dysphoric and cognitive symptoms, which inflates comorbidity**
Complex post-traumatic stress disorder	Distinct: the 3 core symptoms plus 3 disturbances in self-organisation, meaning severe affect dysregulation, persistent negative self-concept, marked interpersonal difficulty	**No such diagnosis exists**
Grief and acute stress	Prolonged grief disorder established; acute stress reaction is a normal response, not a mental disorder	**Acute stress disorder is a formal psychiatric diagnosis**
Severe childhood irritability	Rejected as a mood entity; oppositional defiant disorder with a chronic irritability and anger specifier	**Disruptive mood dysregulation disorder, under depressive disorders, to curb overdiagnosis of paediatric bipolar disorder**

DISORDER AREA	ICD-11	DSM-5-TR
Compulsive sexual behaviour	Included, under impulse control disorders rather than behavioural addictions	**Not included as a diagnostic entity**
Gender	Moved out of the mental disorders chapter to Chapter 17, conditions related to sexual health, renamed gender incongruence, with separate names for childhood and for adolescence and adulthood	**Retained inside the psychiatric manual as gender dysphoria for all ages, with clinically significant distress required**
Substance use	Harmful pattern and dependence separate	**One disorder across 11 criteria: 7 from the old dependence set, 3 from abuse omitting legal problems, 1 new craving item**

- **Concept. The reasoning behind the gender move is examinable.** Placement in the sexual health chapter reflects the conclusion that these conditions are not appropriately viewed as mental disorders.
- **Concept.** That conclusion rests on **field studies indicating that distress and impairment in transgender people is predicted by stigmatisation and victimisation rather than being intrinsic**.
- **Concept.** The aim was to **remove the doubly burdensome stigma of transgender status and mental disorder** while keeping the condition codeable so access to care is preserved.
- **Key point.** The framing to offer: **ICD-11 depsychiatrised the condition while keeping it codeable; DSM-5-TR kept it in the manual with a distress requirement.**

INDIA NOTE

Indian curricula still expect the multiaxial habit. The ICD-10 framework carries clinical syndromes on the first axis, disabilities in personal care, occupational role, familial role and broader social participation on the second, assessed with the disability schedule, and contextual and environmental factors on the third.

Candidates trained on DSM may recite the historic five-axis schema, clinical syndromes, personality disorders and intellectual disability, general medical conditions, psychosocial and environmental problems, and global assessment of functioning, while current DSM practice is non-axial with separate disability staging. Say which system you are using before you list axes.

COMMON TRAP

Five errors examiners fish for: calling ICD-11 codes F-codes, which belongs to ICD-10 published in 1992; calling acute stress reaction a formal ICD-11 disorder; calling personality difficulty a mild personality disorder; importing the DSM alternative-model trait Psychoticism into the ICD-11 domain list, which contains Anankastia instead; and saying both systems treat gender incongruence as a mental disorder.

- **RULE** **Complex post-traumatic stress disorder against borderline personality disorder.** The complex trauma diagnosis requires exposure to severe traumatic stressors, the core trauma symptoms and a negative self-concept of guilt or shame; borderline pathology centres on pervasive fear of abandonment, unstable self-image and recurrent self-harm.

HOLD THIS

ICD-11 is dimensional for personality and categorical for dependence; DSM-5-TR is the reverse. That single inversion answers most comparison questions.

WHAT EARNS THE MARKS

- **The coding system declared first**: it settles which thresholds and which axes the rest of your answer is using.
- **The one inversion held**: dimensional personality with categorical dependence, against categorical personality with a dependence continuum.
- **The gender move explained, not just stated**: the field-study reasoning is what the follow-up question is after.
- **F-codes kept out of an ICD-11 answer**: those belong to the 1992 revision.

SEE ALSO 22 Summary and formulation 25 Differential hierarchy 33 Panic, anxiety, trauma 36 Dissociation and borderline

25 · Differential diagnosis: the hierarchy, and organic first

FORMULATION · TOPIC 25

Give me your differentials in order, and show me how you excluded each tier above your diagnosis.

LONG CASE, DIFFERENTIAL; VIVA TABLE ON THE DIAGNOSTIC HIERARCHY

THE SHAPE OF THE ANSWER

1 State the principle	Parsimony: one entity should account for the whole complex
2 The six tiers	Organic, substance, psychotic, affective, neurotic, personality
3 Organic red flags	By domain, each with how you excluded it
4 Manual then duration	Name the system before you quote any duration threshold
5 Inside the middle tier	Schizophrenia, schizoaffective and psychotic mood, sorted longitudinally

Differentials are presented as an ordered, defensible hierarchy on the principle of parsimony, that a single entity should account for the whole symptom complex where possible.

- Concept. **Jaspers in 1923 recognised that Kraepelinian classification worked hierarchically** to assign one diagnosis to a complex presentation, and Schneider in 1950 reinforced it with first-rank symptoms.
- Concept. **Foulds and Bedford in 1975 proposed four classes of personal illness**, running from delusions of disintegration, through integrated delusions, to neurotic symptoms, to dysthymic states.
- Key point. In classical theory **a higher-level disorder embraces the symptoms of all lower members and excludes the lower diagnoses**.

1. **Organic mental disorders and general medical conditions.** Delirium, dementia, secondary psychotic and mood syndromes.

2. **Substance-induced disorders.** Intoxication, withdrawal, substance-induced psychosis or mood state.
3. **Severe psychotic disorders.** Schizophrenia, schizoaffective disorder, delusional disorder.
4. **Major affective disorders.** Bipolar disorder, major depressive disorder.
5. **Neurotic, stress-related and somatoform disorders.** Obsessive-compulsive disorder, panic disorder, generalised anxiety disorder.
6. **Personality and developmental disorders.**

The corollary is about time as well as rank: symptoms low on the hierarchy, such as anxiety and depression, appear early in decompensation and remit last, while psychotic symptoms develop late and remit earlier.

DOMAIN	ORGANIC RED FLAG	HOW YOU EXCLUDE IT
History of presenting illness	Acute or subacute onset, fluctuating consciousness, rapid progression, atypical age at onset	**A timeline establishing clear sensorium, no systemic illness, gradual deterioration**
Review of systems	Head trauma, fever, nausea and vomiting, weight loss, seizures	**A negative systemic enquiry and no focal deficit**
Biological functions	Sleep-wake reversal, marked anorexia, incontinence	**Detailed history of biological rhythms and sphincter control**
Mental state examination	Clouding of consciousness, disorientation, impaired attention, visual hallucinations, fluctuating deficit	**Serial orientation, digit span, three-compartment memory, proverb interpretation**
Physical and neurological examination	Abnormal vital signs, focal signs, papilloedema, tremor, primitive reflexes	**A full neurological examination including cranial nerves, fundoscopy, motor, sensory and cerebellar testing**

STATE THE MANUAL, THEN THE DURATION

Before defending a psychotic-tier diagnosis, say which system you are coding in, because Indian practice codes in ICD while DSM is taught alongside it and the two set different duration requirements for schizophrenia.

The book position for ICD is that symptoms must have persisted for at least one month for the diagnosis to be assigned, whereas the DSM requirement is a longer total illness duration counting prodromal and residual phases.

A short illness therefore obliges you to justify the duration criterion explicitly and to exclude acute and transient psychotic disorder, where the ICD version involves a period of 3 months compared with 1 month for the DSM equivalent, along with the brief and schizophreniform categories, before settling on schizophrenia.

Quote the threshold from the manual you are using and verify it there.

- **Key point. Within the middle hierarchy, separating schizophrenia from schizoaffective disorder and psychotic mood disorder is longitudinal work.**
- **Concept. Kasanin coined schizoaffective disorder in 1933** for episodic psychoses followed by recovery. Later work by Vaillant in 1964 and Stephens and colleagues in 1966 observed that affective symptoms predicted remission.
- **Concept. The operational demand is full-threshold mood episodes concurrent with the psychosis**, plus a period of delusions or hallucinations without prominent mood symptoms.
- **Key point.** In psychotic depression and mania with psychosis **the psychotic symptoms occur only during the mood disturbance.** If they precede it by months or persist after the mood resolves, the schizophrenia spectrum takes precedence.
- **Concept. Baseline investigations in the exclusion frame** are the complete haemogram, fasting blood glucose, renal and liver function, electrolytes, thyroid function, lipid profile and an electrocardiogram.
- **Concept. Neuroimaging is specifically indicated** in late-onset psychosis, focal signs, sudden unexplained cognitive decline or treatment-refractory illness.

HOLD THIS

Organic, then substance, then psychotic, then affective, then neurotic, then personality. Rank the list, justify the rank, and show how you excluded each tier above your provisional diagnosis.

WHAT EARNS THE MARKS

- **Organic placed first and excluded explicitly**: the tier order is the argument, and skipping the top tier invalidates the rest.
- **An exclusion method beside each red flag**: a timeline, a negative systemic enquiry, serial orientation, a full neurological examination.
- **The manual named before the duration**: the two systems set different thresholds, so the figure needs its source.
- **The lower tiers sorted longitudinally**: whether psychosis sits inside the mood episode is what separates the three middle diagnoses.

SEE ALSO 04 Negative and treatment history 24 ICD-11 and DSM-5-TR 26 For, against, comorbidity 27 Investigations

26 · Points in favour, points against, and comorbidity

FORMULATION · TOPIC 26

Defend your diagnosis against your first two differentials, point by point.

LONG CASE, DIFFERENTIAL DEFENCE; VIVA TABLE ON COMORBIDITY

THE SHAPE OF THE ANSWER

1 Two columns per entity	What argues for it, and what excludes the alternative
2 The substance-using patient	Odds ratios with their direction, not an impression
3 Diagnostic reductionism	The opposite failure: everything blamed on the one diagnosis
4 Comorbidity figures	Community and chronic-illness rates, then what each predicts
5 Iatrogenic mimics	Akinesia, akathisia, drug-induced obsessions, each with its wrong action

The examiner will ask you to defend the provisional diagnosis against the first two differentials. **The expected structure is a two-column defence for each entity**: what argues for it, and what excludes the alternative.

ENTITY	POINTS IN FAVOUR	POINTS AGAINST, OR HOW EXCLUDED
Provisional: schizophrenia	Continuous illness with social deterioration; bizarre delusions; formal thought disorder; hallucinations running a commentary	**No affective episodes meeting criteria for mania or major depression; no focal deficit, no fluctuating sensorium**
Substance-induced psychotic disorder	Concurrent cannabis or stimulant use; symptoms flaring with heavy intake	**Psychosis persisting through verified abstinence; bizarre delusions and thought disorder point away; no prior detoxification or maintenance admissions**
Mania with psychotic symptoms	Hyperactivity, decreased need for sleep, irritable affect	**Affective symptoms mood-incongruent and secondary to persecutory beliefs; no sustained elation or pressure of speech; psychosis persists outside affective episodes**
Organic delusional disorder	Late age at onset or atypical presentation	**Clear sensorium throughout; normal physical and neurological examination; no fever, trauma or seizures**

The substance-using patient is where this is tested hardest, and there is a statistical answer: a discriminant model evaluated 211 inpatients with psychoactive substance use disorders to separate substance-induced psychosis from schizophrenia, classifying 76.2 percent correctly in the derivation dataset and 70.4 percent in cross-validation.

FEATURE	ODDS RATIO	WHICH WAY IT POINTS
Bizarre delusions	6.09:1	**Strongly towards schizophrenia**
Formal thought disorder	3.55:1	**Towards schizophrenia**
Suicidal ideation	0.32:1	**Towards substance-induced psychosis**
Intravenous cocaine use	0.18:1	**Towards substance-induced psychosis**
History of detoxification	0.26:1	**Towards substance-induced psychosis**

Table: two odds ratios in the right direction are worth more than a paragraph of impression. Severe thought disorder and bizarre delusions favour primary schizophrenia even with substance misuse present.

- **Trap. Diagnostic reductionism is the opposite failure**, attributing each sign in a psychotic patient to the schizophrenia alone.
- **Concept. DSM-III carried exclusion rules** forbidding a diagnosis of depression, panic disorder or obsessive-compulsive disorder in the presence of schizophrenia, and DSM-IV retained a remnant in the phrase "not better accounted for". The epidemiology does not support the exclusion.

FINDING	FIGURE	CONSEQUENCE FOR CARE
Associated syndromes, community	85 percent had one or more, with lifetime rates of 54.2 percent for depression, 59.2 percent for obsessive-compulsive disorder and 29.5 percent for panic disorder	**Comorbidity is the rule**
Associated syndromes, chronic illness	76 percent had symptoms of one or more, and 48.6 percent met criteria for full syndromes	**Half of a chronic ward carries a second treatable syndrome**
Depression in schizophrenia	Modal prevalence approximately 25 percent, range 7 percent to 75 percent	**Predicts poorer function and higher risk of recurrent psychosis**
Obsessive-compulsive phenomena	12.9 percent, 26 percent for full disorder and 31.7 percent across studies, with obsessive delusions in 21.6 percent of chronic patients	**Predicts poorer function, earlier onset, more hospitalisation**

Treating the comorbidity helps: adjunctive antidepressant treatment improved affective symptoms and protected against psychotic relapse without destabilising psychosis in stable outpatients, which answers the reflex objection that antidepressants worsen schizophrenia.

COMMON TRAP

Three iatrogenic mimics, each leading to the wrong action. Antipsychotic-induced akinesia mimics postpsychotic depression and deficit schizophrenia with blunted affect, reduced movement and poverty of speech, but lacks depressive cognitions and shows extrapyramidal signs that improve with anticholinergics or a dose reduction. Akathisia mimics worsening anxiety and prompts an erroneous dose increase.

And second-generation antipsychotics, particularly clozapine and risperidone, can provoke or worsen obsessive-compulsive symptoms, then misread as refractory psychosis.

RULE **Severe anxiety in a persecuted patient is not automatically understandable.** "Who would not be anxious if they were being followed?" is the sentence that leaves a disabling, treatable syndrome unaddressed, and examiners quote it back at candidates who use it.

HOLD THIS

Bizarre delusions and formal thought disorder argue for schizophrenia; suicidal ideation and an addiction-treatment history argue for substance-induced psychosis. Name the direction, not just the feature.

WHAT EARNS THE MARKS

- **Each feature given a direction**: bizarre delusions at 6.09:1 and intravenous cocaine use at 0.18:1 point opposite ways.
- **Comorbidity treated as the rule**: 85 percent of a community sample carried one or more associated syndromes.
- **An iatrogenic mimic named**: akinesia read as postpsychotic depression, akathisia read as worsening anxiety.
- **Understandable anxiety refused as an explanation**: "who would not be anxious?" leaves a treatable syndrome unaddressed.

SEE ALSO 25 Differential hierarchy 27 Investigations 30 Schizophrenia 34 Alcohol and opioid

27 · Investigations: yield, baseline, targeted, monitoring

FORMULATION · TOPIC 27

What would you investigate in this patient, and what is the yield of what you have ordered?

LONG CASE, INVESTIGATIONS; VIVA TABLE ON YIELD AND MONITORING

THE SHAPE OF THE ANSWER

1 Lead with yield	History, examination and routine screening, as three figures
2 Drop the wrong phrase	Medical screening and stabilisation, not medical clearance
3 Where yield rises	Older adults, the agitated or disoriented, inpatients
4 The baseline set	Each test with the justification attached to it
5 Monitoring as a separate question	Tolerability, organ toxicity and levels once a drug is planned

Investigations are targeted hypotheses, not a panel. Examiners are testing cost awareness and pretest probability, and **the evidence is unambiguous.**

MODALITY	POOLED YIELD	WHAT IT MEANS FOR YOUR ANSWER
Routine screening investigations	1.1 percent, confidence interval 0.5 percent to 2.2 percent	**Low utility in unselected cohorts; unindicated batteries waste resources and start false-positive cascades**
Clinical history	15.6 percent, confidence interval 9.1 percent to 25.6 percent	**The single highest-yielding diagnostic modality**
Physical and neurological examination	14.9 percent, confidence interval 8.1 percent to 25.9 percent	**Comparable to history, yet vital signs are documented in only 52 percent of psychiatric emergency presentations and a complete neurological examination in 8 percent**

Table: say out loud that investigations complement, and do not substitute for, the history and the examination.

Medical clearance is a phrase to abandon. It is imprecise, it implies an absolute exclusion no brief evaluation can assure, it splits medical from psychiatric practice and it produces misplaced

reassurance. Call the step medical screening and stabilisation.

Protocolised testing, meaning fixed panels ordered regardless of signs, yields 1.6 percent, which does not differ significantly from physician-directed testing at 0.4 percent.

Mandatory screening also **costs money per patient, prolongs emergency stays and triggers invasive cascades** without changing psychiatric disposition.

SUBGROUP	YIELD	WHICH TESTS RISE
Older adults	**Overall 48.5 percent; 27 percent of patients over 60 years had a change in diagnosis or treatment after medical evaluation, against 4.5 percent of patients under 30**	Urinalysis 15.5 percent, complete blood count 7.8 percent, chest radiography 7.7 percent to 8.5 percent, electrocardiography 4.7 percent to 14.3 percent
Agitated, violent or disoriented	**Pooled 22.0 percent; history alone 27.0 percent**	Complete blood count rises to 8.8 percent, creatine kinase elevated in 13.9 percent from exhaustion, immobility, intramuscular injection or dehydration
Inpatient against emergency	**2.9 percent against 0.5 percent**	Referral selection and deeper evaluation during admission explain the difference

- Key point. **Give the baseline set with the justification attached to each test.**
- Concept. **Complete blood count** for occult infection, severe anaemia contributing to fatigue, and a baseline leucocyte and platelet count. Urinalysis is high-yield in the elderly for occult urinary infection driving delirium.
- Concept. **Thyroid function** to exclude hypothyroidism presenting as depression and thyrotoxicosis presenting as mania, and as a requirement before lithium.
- Concept. **Fasting glucose** for undiagnosed diabetes and as a mandatory baseline before a second-generation antipsychotic.
- Concept. **Renal function and electrolytes** for dehydration, renal failure, hyponatraemia driving confusion and hypokalaemia predisposing to arrhythmia, and as a requirement before lithium.
- Concept. **An electrocardiogram** for conduction defects, arrhythmia, ischaemia and baseline QTc.
- Concept. **Syphilis serology** where there is dementia, atypical psychosis or pupillary and reflex abnormality.
- Concept. **Vitamin B12 and folate** as a modifiable cause of cognitive impairment and mood or psychotic presentation. Chest radiography for respiratory signs, suspected tuberculosis, or an elderly admission.

SPECIALISED INVESTIGATION	WHEN IT IS JUSTIFIED	THE YIELD ARGUMENT
Neuroimaging	First episode at an unusual age, focal signs, papilloedema, head trauma, unremitting headache, rapid unexplained cognitive deterioration, atypical dementia, catatonia unresponsive to a benzodiazepine challenge, treatment resistance	**Unselected yield 3.5 percent to 8 percent, which is why it is not routine in a young patient with a normal neurological examination**

SPECIALISED INVESTIGATION	WHEN IT IS JUSTIFIED	THE YIELD ARGUMENT
Electroencephalography	Suspected non-convulsive status epilepticus or complex partial seizures with episodic confusion, automatisms or psychosis; delirium of undetermined cause; rapidly progressive cognitive impairment; severe catatonia with altered vital signs	**Not indicated for uncomplicated functional disorder, diagnostic when the question is episodic or ictal**
Cerebrospinal fluid and autoimmune panels	Suspected central nervous system infection with fever, altered sensorium or meningism; suspected autoimmune encephalitis where psychosis, agitation or catatonia comes with seizures, autonomic instability, orofacial dyskinesias or extreme neuroleptic sensitivity	**Unselected yield zero in uncomplicated emergency psychosis, 8 percent in disoriented cohorts; send paired serum and cerebrospinal fluid neuronal surface antibodies**

- Key point. **Monitoring is a separate question from diagnosis.** Once a drug is planned the investigations shift to tolerability, organ toxicity and levels.
- Concept. **The shared monitoring set**: weight, body mass index, waist circumference, blood pressure and pulse, fasting glucose and lipids, and a movement disorder rating at baseline, then repeated on a schedule.
- Concept. **Lithium requires baseline renal function, electrolytes, thyroid function, glucose, an electrocardiogram and a pregnancy test**, with serum levels drawn at a fixed interval after the dose.
- Concept. **Valproate requires baseline liver function and a platelet count.** Carbamazepine requires a baseline haemogram, liver function and serum sodium, with monitoring for leucopenia, hyponatraemia and transaminitis.

TRAP The intervals are where candidates over-claim. The repeat schedules for metabolic and movement monitoring, and the clozapine haematological protocol, meaning the baseline count, the monitoring intervals and the neutrophil stopping thresholds, are not settled in the evidence used for this note. Verify them from a prescribing source and quote the schedule you have checked; clozapine monitoring is asked whenever antipsychotic baselines are discussed.

COMMON TRAP

The pan-scan, ordering imaging, electroencephalography and a wide panel for a young patient with classical symptoms and a normal examination. The belief that normal bloodwork excludes secondary illness, which misses toxic, autoimmune and seizure-related encephalopathy. Treating asymptomatic bacteriuria in an elderly patient with no systemic infection and no delirium.

And accepting a verbal handoff that the patient is medically cleared without examining them yourself.

- **RULE** **Primary against secondary psychosis, on the investigation axis.** Primary functional psychosis presents in late adolescence or early adulthood with a clear sensorium, third-person or commentary hallucinations, systematised delusions and a low screening yield; secondary organic psychosis presents at an atypical age with fluctuating consciousness, disorientation, prominent visual, olfactory or tactile hallucinations, autonomic instability and focal signs, and targeted testing in the agitated or disoriented group yields 22.0 percent.

HOLD THIS

History yields 15.6 percent, examination 14.9 percent, routine screening 1.1 percent. Say those three numbers and the case against the reflex panel is made.

WHAT EARNS THE MARKS

- **The three yield figures offered first**: they make the case against the reflex panel before you list a single test.
- **A justification attached to each baseline test**: a bare list reads as a protocol rather than as reasoning.
- **Subgroups where yield rises**: 48.5 percent overall in older adults, and 22.0 percent in the agitated or disoriented.
- **Monitoring separated from diagnosis**: once a drug is planned the question changes to tolerability, organ toxicity and levels.

SEE ALSO 17 Why you examine 25 Differential hierarchy 28 Management 35 Delirium and dementia

28 · Management: setting, immediate, master plan, follow-up

FORMULATION · TOPIC 28

How will you manage this patient? Start with where, not with what drug.

LONG CASE, MANAGEMENT; VIVA TABLE ON THE TREATMENT PLAN

THE SHAPE OF THE ANSWER

1 Locus, focus, modus	Where, what first, and by which modalities, in that order
2 Justify the setting	Medical necessity, acute risk, least restrictive environment
3 The first 24 hours	Assessments, observation level, urgent treatment, consent, discharge planning
4 The master plan	Twelve components, running on three axes
5 Review and discharge	Cadence, the stagnation rule, then follow-up within 7 days

Management is presented as **locus, focus and modus**: where, what first, and by which modalities. Starting with drugs is the commonest structural error.

- Key point. **Setting is decided on medical necessity, acute risk and the least restrictive environment.**
- Concept. Acute inpatient care is justified by at least one of these. **Imminent danger to self**, meaning active suicidal ideation with plan, intent and means, or a recent severe attempt.
- Concept. **Imminent danger to others**, meaning recent threats, escalating violence or homicidal ideation with plan, intent and means; or command hallucinations directing harm.
- Concept. **Recent life-threatening self-mutilation or profound loss of impulse control**; or grossly disorganised behaviour preventing safe survival at a lower level of care.

- **Concept. Severe or life-threatening psychotropic adverse effects**; or impairment so severe that stabilisation requires a 24-hour medical environment.
- **Concept. Below that sit four lower levels**: subacute inpatient care, partial hospitalisation for structured daytime stabilisation and nutritional rehabilitation, intensive outpatient programmes, and routine outpatient care.

The first 24 hours. An initial plan of care is formulated by the psychiatrist and the nurse. The nursing assessment and the psychiatric evaluation begin immediately, with most parts complete within 8 hours and both documented within 24 hours, and a general medical history and physical examination completed within 24 hours.

Set the observation level from stratified suicide, violence and fall risk. Then deliver urgent treatment for severe agitation, catatonia, dehydration or medical decompensation.

Involve the patient and caregivers, obtain informed consent, and **begin discharge planning from admission** rather than near the end of the stay.

DISCIPLINE	ASSESSMENT	COMPLETION
Psychiatry, medicine, nursing	Psychiatric evaluation; medical history and examination; nursing assessment and initial plan of care	**Within 24 hours, most of the nursing assessment within 8 hours**
Psychiatry	Cognitive screen and involuntary movement rating	**Within 5 days**
Social work, psychology, rehabilitation	Psychosocial history, psychological and functional assessment, patient strengths and goals	**Within 6 days**
Interdisciplinary team	The comprehensive master treatment plan	**Within 7 days of admission**

- **Key point. The master plan carries twelve components.** Identifying details, diagnostic codes, presenting problems in the patient's own words, strengths, barriers, and long-term goals.
- **Concept.** The remaining components are **measurable short-term objectives, specific interventions, responsible clinicians, target review dates, discharge criteria, and the signatures of patient and clinician**.
- **Concept. Biologically**: baseline investigations, then rational drug selection guided by symptoms, previous trials, adverse-effect profile, metabolic indices and patient preference, with explicit titration.
- **Concept.** The biological axis also carries **electroconvulsive therapy** where rapid resolution of severe catatonia, intractable depression or severe psychosis is indicated, and physical monitoring.
- **Concept. Psychologically**: the modality matched to the pathology, with technique, session length, weekly frequency and therapist named, plus skills work in cognitive restructuring, worry postponement and problem-solving.
- **Concept. Socially**: family psychoeducation and caregiver burden work, social skills training, and rehabilitation addressing entitlements, housing, vocation and legal support.

GOALS AGAINST OBJECTIVES

Goals are broad long-term outcomes, typically 2 to 4 per plan, at least one linked directly to the discharge criteria, and in an inpatient plan the life goal carries a direct quote from the patient. Objectives are the measurable milestones under each goal, typically 2 to 3 per goal, each specific, measurable, achievable, relevant and time-bound.

"Patient will feel less depressed" fails; "patient will score 9 or below on the depression scale, down from 14, by 60 days, measured weekly in session" passes, as does "patient will report sleeping 7 or more hours per night on at least 5 nights per week by 30 days" and "patient will identify 5 relapse triggers and 5 coping responses by 14 days".

Review cadence. Progress notes are linked to active plan goals, because that substantiates medical necessity and continuity.

An institutional inpatient schedule **reviews at 21, 35, 49 and 63 days of admission and every 30 days thereafter**, with a review within 7 days of a unit transfer and formal quarterly reviews every 90 days.

Programme-level intervals run at **a minimum of 30 days for residential and partial hospitalisation and 60 to 90 days for outpatient care**.

The stagnation rule is examinable. If no measurable progress is shown on an objective for 2 consecutive months, the team revises it or records a clear rationale for continuing.

An unscheduled focused review is triggered by constant or line-of-sight observation, restraint or seclusion, acute physical deterioration or transfer, sudden escalation of symptoms or aggression, emerging ideation or an attempt, a documented fall, or treatment disengagement defined as persistent unexcused absence within one week.

In that review **the on-duty physician and charge nurse document an interim plan with orders**, and the full team convenes on the following working day.

- **Concept. Discharge from acute care requires four things.** Absence of imminent danger to self or others; resolution of gross psychotic disorganisation sufficient for safe functioning at a lower level; substantial achievement of the stabilisation goals; and supports organised at that lower intensity.

An outpatient appointment with a licensed mental health clinician is **scheduled within 7 days of discharge**, and the social worker confirms transmission of the discharge summary, medication reconciliation and community connections before the patient leaves.

INDIA NOTE

Name the two admission routes the Act itself uses. Independent admission covers the person who has capacity to make admission and treatment decisions and who requests or agrees to admission.

Supported admission covers the person with high support needs who cannot make that decision independently, and it is the term that replaced the involuntary admission language of the earlier legislation.

A supported admission is time-limited in the first instance, and continuation beyond that period requires a fresh statutory review by the Mental Health Review Board rather than an extension written by the treating team. Emergency treatment is permitted to prevent immediate harm.

The Act presumes capacity unless the contrary is shown and grants the right to a nominated representative and an advance directive. Electroconvulsive therapy without muscle relaxants is prohibited, and its use in minors requires specific permissions.

Long-term rehabilitation connects to the District Mental Health Programme and the National Mental Health Programme for affordable medication, community follow-up and disability certification.

TRAP Calling a supported admission an involuntary admission, which reads as a candidate who has not absorbed the change in the law. The paired trap is quoting numbers: the section numbers, the first-instance duration, the renewal period and the permitted duration of emergency treatment are not settled in the evidence used for this note, so read them off the bare Act and verify each beforehand.

RULE **Separate personal barriers from systemic barriers.** Poor distress tolerance and cognitive impairment are personal barriers to achieving a goal; homelessness, pending court proceedings and missing welfare entitlements are systemic barriers to discharge, and they need different interventions.

HOLD THIS

Setting, then the first 24 hours, then the master plan on three axes, then follow-up within 7 days. Drugs are a paragraph inside the plan, not the opening line.

WHAT EARNS THE MARKS

- **Setting argued before any prescription**: locus, focus and modus is the order, and drugs sit inside the plan.
- **An objective written so it can be measured**: "will feel less depressed" fails, a scored target with a date passes.
- **The two admission routes named in the Act's own words**: independent admission and supported admission.
- **Personal barriers separated from systemic ones**: distress tolerance and homelessness need different interventions.

SEE ALSO 20 Risk factor classes 23 Biopsychosocial and 5 P 27 Investigations 29 Prognosis

29 · Prognosis, and how to state it

FORMULATION · TOPIC 29

What is the prognosis in this patient, and on what basis?

LONG CASE, PROGNOSIS; VIVA TABLE ON OUTCOME PREDICTORS

THE SHAPE OF THE ANSWER

1 **Refuse the single word**	Prognosis is a forecast on axes, not the word "guarded"
2 **Three axes**	Temporal, outcome, risk, each stated separately
3 **The classical predictors**	Onset, precipitant, symptom profile, episode history, constitution
4 **The modern additions**	Duration of untreated psychosis, and the inverted subtype ranking
5 **Close on modifiability**	Psychoeducation, social rhythms, family and community follow-up

Prognosis is the forecast of the probable course, duration, symptomatic remission, functional recovery and treatment responsiveness, and **a single word is criticised**.

- **Concept. Temporal**: immediate prognosis for this episode against long-term prognosis for recurrence, inter-episode stability and preserved functioning.
- **Concept. Outcome**: symptomatic recovery against functional recovery in independent living, work and relationships.
- **Concept. Risk**: lifetime risk of suicide, accidental harm, substance relapse, metabolic comorbidity and non-adherence.
- **Key point. Close with a synthesis built from the 4 P factors.** The immediate prognosis for symptomatic stabilisation in this episode is favourable given the acute onset, an identifiable precipitant and a prompt response to pharmacotherapy.
- **Concept.** The second half of that synthesis: **the long-term functional prognosis remains guarded** in view of the young age at onset, family loading for affective illness and residual socio-occupational impairment.

DOMAIN	FAVOURABLE IN SCHIZOPHRENIA	UNFAVOURABLE IN SCHIZOPHRENIA
Onset and duration	Acute onset, short duration of illness	**Insidious onset, long duration before presentation**
Precipitating factors	Obvious exogenous precipitant	**Absent or obscure precipitant**
Symptom profile	Prominent confusion; atypical symptoms, especially affective; minimal process symptoms	**Prominent process symptoms, blunted affect, negative symptoms, no affective or confusional element**
Episode history	A previous episode with a long interval, complete recovery in the preceding remission, dissimilar episodes	**Prolonged unremitting symptoms, short remissions, identical stereotyped deteriorating episodes**
Subtype, classical series	Catatonic best; acute unclassifiable atypical cases	**Paranoid least favourable classically, hebephrenic and simple intermediate**

DOMAIN	FAVOURABLE IN SCHIZOPHRENIA	UNFAVOURABLE IN SCHIZOPHRENIA
Constitution	Pyknic habitus, extravert temperament with adequate pre-psychotic adjustment	**Asthenic habitus, introverted temperament with inadequate adjustment**
Heredity and course	Family history of manic-depressive psychosis; improvement or a fluctuating course	**Family history of chronic deteriorating illness; a progressive unremitting hospital course**

Table: the classical criteria. Sex, formal education, individual abilities and psychosexual history carried no clear prognostic significance in that analysis, and relatively late age at onset was there considered unfavourable.

RULE **The classical list is not the whole modern answer.** Add the duration of untreated psychosis: a longer period of unmedicated psychosis before treatment is associated with a reduced likelihood of remission, more prominent negative psychopathology and poorer functioning. Note also that in contemporary practice paranoid schizophrenia often shows better functional outcome than hebephrenic or simple forms, inverting the classical ranking.

QUESTION	FACTORS THAT MAKE THE ILLNESS CHRONIC	FACTORS THAT KEEP IT CHRONIC
Obsessive-compulsive disorder, prospective cohort	Childhood psychotrauma, odds ratio 1.98; recent negative life events, odds ratio 1.42; a partner is protective, odds ratio 0.28	**Longer illness duration, odds ratio 1.46; higher baseline severity, odds ratio 1.09**

Table: external stressors and isolation govern the transition into chronicity, while intrinsic illness variables maintain it once established, the argument for early stress-focused intervention and partner or family engagement.

Comorbid bipolar disorder changes the prognosis of obsessive-compulsive disorder, and this is a favourite table.

Bipolar disorder affects **between 4 percent and 16 percent of patients with obsessive-compulsive disorder**, and between 11 percent and 21 percent of patients with primary bipolar disorder have comorbid obsessive-compulsive disorder. In a multicentre cohort of 955 adults, lifetime bipolar comorbidity was 7.75 percent.

Comorbidity brings **more panic disorder with agoraphobia, more impulse control disorder, more suicide attempts, and more poor insight and sensory phenomena**.

Contamination obsessions are fewer, odds ratio 0.71, while sexual obsessions are commoner, odds ratio 1.77.

The course becomes episodic in up to 75 percent of comorbid patients against 3 percent of those without bipolar disorder.

Obsessive symptoms **worsen in depressive episodes in 78 percent and improve in manic or hypomanic episodes in 64 percent**, and depressive burden is higher, with a mean of 8.9 episodes against 4.1.

TRAP Prescribing serotonergic antidepressant monotherapy for severe obsessions without screening for bipolarity. The switch rate to hypomania or mania is 60 percent in comorbid bipolar-obsessive-compulsive disorder against 4.1 percent in unipolar cases, which is why mood stabilisers with an antipsychotic add-on are preferred there.

BIPOLAR DISORDER	FAVOURABLE	UNFAVOURABLE
Onset and episode count	Later age at onset, fewer prior episodes	**Early onset in childhood or adolescence, high episode frequency or rapid cycling**
Inter-episode state	Good adherence, no psychotic features	**Persistent subthreshold depressive symptoms between episodes, psychotic features**
Comorbidity and support	No substance misuse, strong family support	**Comorbid substance use disorder, cardiometabolic or other psychiatric comorbidity**

The mortality figures carry the risk axis. Life expectancy in bipolar disorder is shortened by approximately 12 to 14 years, and cardiovascular death is increased 1.6- to 2-fold and occurs earlier.

In bipolar disorder **the lifetime risk of suicide attempts is 25 percent to 50 percent, and 15 percent to 20 percent die by suicide**.

Obsessive-compulsive disorder is not benign either. Pooled lifetime prevalence of suicide attempts is 13.5 percent, current suicidal ideation 27.3 percent and lifetime suicidal ideation 47.3 percent, rising further with comorbid bipolar disorder, panic disorder or impulse control disorders.

The course of bipolar disorder often reflects **kindling**, in which recurrent episodes become progressively autonomous and less dependent on external precipitants, and inter-episode recovery may become less complete as the episode count rises.

- **Key point. Prognosis is modifiable, and saying so finishes the answer.**
- **Concept. Structured psychoeducation reduces relapse, improves adherence and decreases admissions in bipolar disorder**, without directly altering baseline symptom severity or functional deficit, so it is an adjunct rather than a substitute.
- **Concept. Regular sleep-wake cycles and social rhythms give useful prophylaxis** against affective relapse.
- **Concept.** In Indian practice, **involving the family as care partners, appointing a nominated representative and establishing community follow-up through District Mental Health Programme clinics** supports continuity, reduces financial burden and sustains rehabilitation.

TRAP Two prognostic factors in obsessive-compulsive disorder are standard probes and only one is settled here: poor or absent insight is associated with a lower probability of remission and a need for more medication trials, while the independent prognostic effect of the hoarding dimension is not in the evidence used for this note. Verify it beforehand rather than estimating.

HOLD THIS

Three axes, then one synthesis sentence. Temporal, outcome, risk, with symptomatic and functional prognosis stated separately, because they often diverge.

WHAT EARNS THE MARKS

- **Symptomatic and functional prognosis separated**: they diverge often enough that merging them loses the answer.
- **Duration of untreated psychosis added to the classical list**: a longer untreated period predicts less remission and poorer function.
- **The classical subtype ranking flagged as inverted**: paranoid illness often does better functionally in contemporary practice.
- **Modifiability stated at the close**: psychoeducation, social rhythms and family engagement are the levers you actually hold.

SEE ALSO 03 Onset and course 23 Biopsychosocial and 5 P 28 Management 31 Mania and bipolar depression

Twenty long cases with the examiner's ladder

30 · Schizophrenia, and the psychoses beside it

LONG CASES · TOPIC 30

Which psychosis is this, which antipsychotic will you start, and for how long will you maintain it?

LONG CASE, PSYCHOSIS; VIVA TABLE ON THE PSYCHOSIS DIFFERENTIAL

THE SHAPE OF THE ANSWER

1	Summarise and formulate	History, biopsychosocial formulation, then the criteria you are using
2	Take the right exit	Four exits sorted by duration and the psychosis to mood relation
3	Setting in law	Independent or supported admission, with the certifications
4	Prescribe and monitor	Stratify by aggression, then baseline and scheduled monitoring
5	Resistance and psychosocial care	Define resistance, set out clozapine, then the interventions

One ladder, four exits. Every psychosis case is asked in the same order, and **the four exits are schizophrenia, an acute and transient psychosis, a schizoaffective episode and a persistent delusional disorder.**

- Key point. **Learn the exits as durations and as the relation between psychosis and mood**, and the differential answers itself.

1. Summarise the history and give the biopsychosocial formulation.
2. State the diagnostic criteria and how the classification frames the disorder.
3. Give the psychiatric and organic differentials with a discriminator for each.
4. Decide the locus of care and justify admission in law.
5. Choose the first antipsychotic, and say how aggression changes that choice.
6. State the duration of maintenance after remission of a first episode.

7. Give the baseline investigations and the monitoring schedule.
8. Define treatment resistance and set out the clozapine pathway.
9. Name the psychosocial interventions you would integrate.

Criteria. In the classification you can source, schizophrenia sits at F20 and needs characteristic symptoms for a minimum of 1 month. A shorter presentation is F23.2, acute schizophrenia-like psychotic disorder.

The domains to recite, first half. Thought echo and interference; delusions of control, influence or passivity and delusional perception; voices giving running commentary or discussing the patient in the third person.

The domains to recite, second half. Other persistent culturally inappropriate delusions; incoherent speech; catatonic behaviour; and negative symptoms.

Exclusions. Not attributable to a medical or central nervous system disorder, not secondary to substance intoxication or withdrawal, and not diagnosed where extensive affective symptoms are present unless the schizophrenic symptoms clearly antedate them.

TRAP The examiner asks what the newer classification changed: the subtypes are gone, and it states its own minimum symptom count, its required core positive symptom and its duration. Those figures are not carried by the sourced material here, so **verify them in the classification itself before the exam** and answer the frame you can source rather than reciting a count you cannot.

EXIT	WHAT IS PRESENT	DURATION AND COURSE DISCRIMINATOR
Acute and transient psychotic disorder (F23)	Polymorphism with shifting turmoil, or stable schizophrenia-like symptoms	**Abrupt onset within 2 weeks, resolving within 1 to 3 months; the schizophrenia-like form lasts less than 1 month**
Schizoaffective disorder (F25)	Prominent schizophrenic and prominent affective symptoms in the same episode	**The two develop concurrently and balance each other**
Persistent delusional disorder (F22)	One delusion or a related system, affect and cognition intact, no third-person voices or passivity	**Insidious and long-standing, often lifelong**
Mood disorder with psychotic features	Delusions and hallucinations secondary to affective polarity	**Psychosis only within affective episodes, with inter-episode recovery**
Substance-induced psychotic disorder	Direct effect of ingestion or withdrawal, prominent illusions and tactile or visual hallucinations	**Resolves with detoxification and sustained abstinence**

EXIT	WHAT IS PRESENT	DURATION AND COURSE DISCRIMINATOR
Psychosis due to a medical condition	Fluctuating sensorium, neurological signs, late onset, abnormal laboratory or imaging findings	**Follows the course of the underlying disease**

- RULE **Duration sorts the psychoses; the relation between psychosis and mood sorts the rest.** Under 1 month and remitting is acute and transient; concurrent and balanced is schizoaffective; mood-bound only is a mood disorder with psychotic features.

- Key point. **First antipsychotic: monotherapy**, chosen on efficacy, side effects, comorbidity, formulation preference and past response, then stratified by aggression, which is the step candidates omit.
- Concept. **Without violence, four oral agents have comparable efficacy, discontinuation and mortality**: aripiprazole, olanzapine, perphenazine and risperidone, with amisulpride a viable alternative in Indian practice.
- Concept. **With violence or marked hostility, olanzapine is first-line** for its sedative and anti-aggressive effect, and inadequate response moves the patient to clozapine rather than to another first-line agent.

THE MAINTENANCE ANSWER

Offer maintenance antipsychotic treatment for a minimum of 7 to 12 months to an adult with a first episode in remission, balancing efficacy, side effects and preference. That is the 2023 update; earlier 2012 guidance said at least 12 months after sustained remission. Note the strength asymmetry: continuing an antipsychotic is a recommendation, continuing the *same* antipsychotic only a suggestion.

Monitoring and movement disorders. Baseline weight, height, waist circumference, blood pressure, pulse, fasting glucose, HbA1c, fasting lipids and a rating of involuntary movements, with an electrocardiogram where there is cardiac risk.

Then **weight at 6 weeks and the full panel at 12 weeks and annually**.

- Concept. **Acute dystonia takes a parenteral or oral anticholinergic.** Parkinsonism takes dose reduction, a switch or an anticholinergic.
- Concept. **Akathisia takes dose reduction, a switch, a benzodiazepine or propranolol.** Moderate to severe tardive dyskinesia takes a reversible inhibitor of vesicular monoamine transporter 2.

The clozapine pathway. Resistance is persistent symptoms and impairment despite adequate trials of different antipsychotics at therapeutic doses for adequate duration with adherence verified, and the number of failed trials required is 2.

- Concept. **Indications are confirmed resistance, substantial persistent suicide risk, and substantial persistent risk of violence.**
- Concept. **Workup**: neutrophil count, blood count, fasting metabolic profile, liver and renal function, electrocardiogram and troponin.
- Concept. **Contraindications**: severe cardiac disease, active marrow suppression, previous clozapine-induced agranulocytosis.

- **Key point.** Name the adverse effects: **agranulocytosis, myocarditis, adynamic ileus, metabolic syndrome and dose-dependent seizures**.

TRAP The clozapine follow-ups are the starting dose, the neutrophil monitoring intervals, the thresholds that decide continuation, closer monitoring or withholding with haematology referral, and the target plasma concentration. None is carried here. **Take them from a cited protocol and verify before the exam**; a remembered threshold is the worst answer in this part of the viva.

The same holds for catatonia complicating schizophrenia, the benzodiazepine challenge and the place of electroconvulsive therapy in it.

THE STABILITY QUESTION

Short-term recovery is good and long-term retention is not. Among 3,074 patients followed for a mean of 1,495 days, 61.26 per cent still held the index diagnosis at 8 years, cumulative transition to any non-organic psychosis reached 46.25 per cent, and transition to a schizophrenia-spectrum disorder reached 36.14 per cent.

Onset is a shift from non-psychotic to clearly psychotic within 2 weeks, with resolution within 1 to 3 months, and the remission ceilings below are what separate these states from schizophrenia at one end and persistent delusional disorder at the other.

Management is a low to moderate dose second-generation antipsychotic, then tapering considered within 3 to 6 months of full remission while you watch for transition.

Schizoaffective disorder is asked as **a contrast of two models**.

The longitudinal model needs a major mood episode concurrent with active-phase schizophrenia criteria, delusions or hallucinations for 2 or more weeks free of any mood episode across the lifetime illness, mood symptoms for the majority of total active and residual illness, and exclusion of substances and medical causes.

The episode-level model instead needs the full requirements for schizophrenia and a concurrent moderate to severe affective episode, simultaneously or within a few days, both present at least 4 weeks.

- **Trap.** Dropping the lifetime psychosis-free-of-mood criterion. **Dropping it turns each psychotic mood disorder into schizoaffective disorder.**
- **Concept.** **Treatment is a second-generation antipsychotic backbone**, paliperidone extended-release carrying a specific approval.
- **Concept.** Add **a mood stabiliser for the bipolar subtype**, an antidepressant for the depressive subtype after screening for switch risk, and clozapine for refractory illness or persistent suicidality.

Delusional disorder is the encapsulated case, and **the examiner wants both duration thresholds**: 1 month in one classification, 3 months in the other.

- **Concept.** **Permissible hallucinations differ too.** Non-prominent tactile or olfactory ones congruent with the theme, against transitory hallucinations in any modality apart from third-person or running-commentary voices.
- **Concept.** **Bizarre delusions are permitted in the first and excluded in the second**, while thought alienation, passivity and negative symptoms are absent in both.

- **Concept. Subtypes** are persecutory, jealous, erotomanic, somatic, grandiose, and mixed or unspecified.
- **Key point.** In the interview **hold impartial neutrality**. Refuting the belief makes you part of the persecutory system, and agreeing destroys your credibility the moment you prescribe.
- **Trap.** Treating delusional jealousy as low risk. **It carries risk of domestic violence, partner homicide and suicide**, so ask about interrogation rituals, surveillance, weapons and explicit threats.

ITEM	FIGURE	THE DISCRIMINATOR THE EXAMINER LISTENS FOR
Prevalence and subtypes	About 0.2 per cent lifetime, 1 to 2 per cent of psychiatric admissions; in 467 outpatients, persecutory 48 per cent, jealous 11 per cent, mixed 11 per cent, somatic 5 per cent	**Comorbid personality disorder in 42 per cent, mostly paranoid, and late-onset somatic delusions need dermatological and laboratory exclusion first**
Pimozide	Historical series across 209 patients: recovery over 50 per cent, partial 28 per cent, non-response 20 per cent	**A Cochrane review found no rigorous randomised evidence of superiority, and QT prolongation removes it from first line**
Aripiprazole	Average 11.1 mg daily across 21 cases, below the 15 mg defined daily dose	**Mean latency 5.7 weeks, so do not call it a failure early**
Other atypicals	Risperidone 1 to 3 mg, olanzapine 2.5 to 10 mg, quetiapine 150 mg daily	**Tolerability, not potency, drives the choice: side effects feed the persecutory system**
Response and therapy	Good response 33.6 per cent overall; a controlled trial of cognitive behavioural therapy in 24 patients over 24 weeks	**Therapy reduces conviction, distress and acting out rather than the delusion; psychodynamic work is contraindicated and electroconvulsive therapy has no antidelusional effect here**

THE INDIAN LAW ANSWER, GIVEN HONESTLY

Admission and treatment run under the Mental Healthcare Act 2017, and the substance is what you must state. Independent admission where the patient has capacity and asks for it.

Supported admission where the patient lacks capacity, has severe mental illness and presents substantial risk of harm to self or others or cannot care for basic needs, on application by the nominated representative with two medical examination reports of which at least one is by a psychiatrist, reported to the Mental Health Review Board, with a separate provision for continuation.

A nominated representative the patient may designate, and an advance directive made while capacity is intact. Electroconvulsive therapy only with muscle relaxants and anaesthesia, with restrictions in a minor. Limits on restraint and seclusion. Decriminalisation of attempted suicide, with a presumption of severe stress and a duty to treat.

Capacity is decision-specific, time-specific and context-specific, so a delusion about a spouse can coexist with intact financial capacity.

TRAP The examiner will press for the numbered section, the certification requirement and the permitted duration of each provision. The section numbers, the initial duration of supported admission, the emergency treatment time limit and the sections governing electroconvulsive therapy, restraint, advance directives and decriminalisation of attempted suicide are not carried by the sourced material used here.

Read them off the bare Act and verify before the exam. Naming the provision, the applicant and the two certifications, and flagging the number as one you will confirm, beats a half-remembered figure.

HOLD THIS

Duration is the spine of the psychosis differential. Under 1 month and remitting, acute and transient; at least 1 month, schizophrenia; psychosis for at least 2 weeks free of mood, schizoaffective; delusions alone past the threshold, delusional disorder. Then stratify the first prescription by aggression and maintain a first episode 7 to 12 months.

WHAT EARNS THE MARKS

- **Duration used as the spine of the differential**: the four exits sort on how long the psychosis has run and how it sits with mood.
- **The first prescription stratified by aggression**: it is the step candidates omit, and it changes the agent.
- **Resistance defined before clozapine is mentioned**: two adequate trials with adherence verified, not two prescriptions.
- **The law given as substance plus a declared gap**: name the provision, the applicant and the two certifications, and flag the section number as one you will confirm.

SEE ALSO 13 Possession and content 25 Differential hierarchy 26 For, against, comorbidity 31 Mania and bipolar depression

31 · Mania, and bipolar depression

LONG CASES · TOPIC 31

This patient is manic. What do you give, what do you argue about valproate, and who is maintained indefinitely?

LONG CASE, MANIA; VIVA TABLE ON MOOD STABILISERS

THE SHAPE OF THE ANSWER

1 **The entry criterion**	Increased activity or energy combined with mood, not mood alone
2 **Hypomania against mania**	Duration, function, psychosis, as a three-column contrast
3 **Acute treatment in steps**	Stop the antidepressant, verify adherence, then combine or monotherapy
4 **The valproate argument**	Effect sizes, suicidality, teratogenic liability, guideline placement
5 **Bipolar depression hierarchy**	First, second and third line, then the antidepressant figures

Two poles, two examinations. Mania is an emergency assessment plus a drug-choice argument; bipolar depression is a diagnostic argument plus a hierarchy you recite in order.

- **Key point.** Both cases **close on how long you continue and who is maintained indefinitely**.

1. State the criteria for a manic episode and how the current framing differs.
2. Separate hypomania from mania, and define marked impairment.
3. Give the differential, including borderline personality disorder.
4. Conduct the emergency assessment and place the admission in law.
5. Give first-line pharmacotherapy for acute mania.
6. Argue the evidence on valproate against lithium and the antipsychotics.
7. Manage a mixed episode and rapid cycling.
8. Set out refractory mania, clozapine and electroconvulsive therapy.
9. Transition to maintenance, and say who is maintained indefinitely.

The entry criterion is the discriminator. The current framing requires increased activity or energy *combined with* euphoric, irritable or expansive mood; the older framing permitted elevated mood alone. That change exists to stop the diagnosis resting on a subjective state, and it is the answer to "how does the new classification differ".

FEATURE	HYPOMANIA	MANIA	MANIA WITH PSYCHOSIS
Duration	At least 4 days in one manual, several days in the other	7 days, or shorter than 1 week if truncated by treatment	7 days, or shorter than 1 week if truncated by treatment
Function	**No marked impairment; efficiency may rise**	**Marked impairment of social, family or occupational roles**	**Marked to severe disruption**
Psychosis	Absent by definition	Absent	**Present, and its presence alone makes the episode manic**

RULE **Any delusion or hallucination makes the episode mania, however cheerful and cooperative the patient looks.** Hours against days settles the borderline differential: affective shifts there last a few hours and rarely exceed a few days, while hypomania runs at least 4 days and mania 7 days.

Key point. **Acute treatment, step one: stop any antidepressant.** If the patient is already on a mood stabiliser, verify adherence, check the level and optimise the dose.

Concept. For severe untreated mania, **first-line is a mood stabiliser (lithium or valproate) plus an atypical antipsychotic**, or monotherapy with lithium, valproate or a second-generation antipsychotic, with a benzodiazepine adjunctive and short-term.

Refractory mania is failure of at least 2 antimanic trials of adequate dose and duration from different classes. Escalate to combination, switch the antipsychotic class, consider clozapine, and use modified electroconvulsive therapy for life-threatening exhaustion, delirious mania, severe psychotic agitation or non-response.

A mixed manic episode is **full manic criteria with 3 or more concurrent depressive symptoms**, and it responds better to valproate or a second-generation antipsychotic than to lithium alone.

LITHIUM, THE NUMBERS YOU CAN DEFEND

Start 300 mg two to three times daily and adjust by 300 mg every 3 days against drug monitoring. Target trough in acute depression 0.8 to 1.2 mEq/L, reduced to 0.4 to 0.8 mEq/L in older adults; maintenance 0.6 to 1.0 mEq/L.

Draw the trough exactly 12 hours after the last dose and at least 5 days after any adjustment. Establish two consecutive in-range levels, then monitor every 3 to 6 months, with renal function, thyroid function and plasma calcium at baseline, at 6 months and at least annually.

TRAP The starting dose, titration step and target level for each antimanic agent, and the benzodiazepine ceilings, are not carried by the sourced material here for that indication.

Verify every antimanic dose against a primary dosing reference before the exam. Asked which malformation belongs to which older mood stabiliser: carbamazepine is linked to spina bifida and anomalies of the fingers, while the malformation specific to first-trimester lithium and a neural tube defect figure for carbamazepine are unsourced here.

Do not invent a percentage under pressure.

The valproate argument is a set piece that rewards precision. Approved for acute mania in 1994 after one positive trial, it then showed diminishing effect sizes across 4 further placebo-controlled trials, 2 of which failed to separate from placebo.

Its **standardised mean difference against placebo is 0.16**, where lithium, carbamazepine and several antipsychotics range from 0.37 to 0.56.

One national guideline places it third-line; another keeps it among 8 first-line options but below lithium and quetiapine.

Anticonvulsants carry a risk of suicidal behaviour twice that of placebo, where lithium has documented anti-suicidal efficacy.

- **Concept.** Valproate **produces more weight gain than lithium, olanzapine or quetiapine**, and carries hepatotoxicity, pancreatitis, thrombocytopenia and polycystic ovary syndrome.
- **Key point.** **Its teratogenic liability is the highest of any bipolar medication**, and it is declared unacceptable for women of childbearing potential.
- **Concept.** The same source **advises against carbamazepine for women who could become pregnant**, and names lithium and the second-generation antipsychotics, quetiapine in particular, instead.

Maintenance. Continue the full therapeutic dose for 6 to 9 months after remission, because tapering inside that window carries substantial relapse risk.

Lithium is the premier maintenance agent, preventing relapse at both poles with established reduction of suicide mortality, and quetiapine holds approval and robust long-term evidence.

Valproate showed maintenance effectiveness in a 2021 review of 13 reports and 9,240 participants, though **off-label for that purpose in some jurisdictions**.

Indefinite maintenance is indicated for multiple relapses on treatment or withdrawal, 2 or more affective episodes in 5 years, severe highly lethal attempts, residual symptoms, a strong family history with poor outcome, and concurrent substance dependence.

RED FLAGS FOR BIPOLARITY IN A DEPRESSED PATIENT

First episode before 25 years of age, with onset averaging 6 years earlier than unipolar depression. Five or more prior depressive episodes. A first-degree relative with bipolar disorder or who died by suicide. Psychotic features, psychomotor agitation, or postpartum onset of depression or psychosis. Atypical vegetative symptoms: hypersomnia, hyperphagia, leaden paralysis. Past suicide attempts.

Past antidepressant-induced hypomanic symptoms. A screening questionnaire aids case-finding but lacks the sensitivity and specificity to stand alone, so mood charting and collateral history settle the boundary.

The hierarchy, in order. First-line for acute bipolar I depression: quetiapine monotherapy; lurasidone with lithium or divalproex; lithium; lamotrigine; cariprazine; adjunctive lurasidone, with lamotrigine also usable adjunctively.

Second-line: divalproex; adjunctive selective serotonin reuptake inhibitor or bupropion; electroconvulsive therapy; olanzapine with fluoxetine; lumateperone at 42 mg.

Third-line is alphabetical, and the examinable part is that intravenous ketamine, repetitive transcranial magnetic stimulation, light therapy and modafinil sit there.

Not recommended: antidepressant monotherapy, aripiprazole monotherapy, lamotrigine with folic acid.

Switching is preferred over adding unless the first agent gave a meaningful partial response.

In bipolar II depression **quetiapine monotherapy is the only first-line option**, with lithium, lamotrigine, lumateperone, adjunctive bupropion, and sertraline or venlafaxine in pure non-mixed depression as second-line. Maintenance first-line is quetiapine, lithium and lamotrigine.

AGENT	DOSING YOU CAN QUOTE	THE DISCRIMINATOR
Quetiapine	Goal 300 mg daily; start 50 to 100 mg at bedtime, increase by 50 to 100 mg every 1 to 7 days up to 400 mg daily; maximum 800 mg per 24 hours	**Highest-ranked monotherapy and the only first-line monotherapy in bipolar II; weight monthly for 3 months then quarterly, glucose and lipids at 3 months and yearly**
Lurasidone	Monotherapy or added to lithium or divalproex	**Low metabolic liability and the only first-line agent in children and adolescents; it lacks anti-manic monotherapy efficacy**
Lamotrigine	25 mg daily for 2 weeks, 50 mg for 2 weeks, 100 mg for 1 week, target 200 mg daily; reduce by 50 per cent with valproate	**Titration over 6 or more weeks cannot rescue a crisis, and the valproate reduction prevents a severe cutaneous reaction**
Lithium	Acute trough 0.8 to 1.2 mEq/L, 0.4 to 0.8 mEq/L in older adults, maintenance 0.6 to 1.0 mEq/L	**The only agent effective across mania, depression and maintenance with an anti-suicide effect**

The antidepressant controversy, with the figures. Non-response is 1.6 times commoner in bipolar than unipolar depression, 51.3 per cent against 31.6 per cent, and modern agents do no better than tricyclics.

Switch to mania occurred in 48.8 per cent of bipolar patients against 0.0 per cent of unipolar patients, across classes: reuptake inhibitors 22.0 per cent, bupropion 20.7 per cent, tricyclics 13.0 per cent.

A concurrent mood stabiliser reduces switch, 18.8 per cent with against 81.3 per cent without.

It does not, however, prevent **cycle acceleration (25.6 per cent) or induced rapid cycling (32.1 per cent of those exposed for 52 or more weeks)**.

Late loss of response is 3.4 times commoner, and withdrawal relapse within 8 weeks of stopping is 4.7 times less frequent.

- Key point. So the position is: **adjunctive reuptake inhibitor or bupropion only, second-line, and only with a therapeutic mood stabiliser**.

- **Concept.** Avoid it in **mixed features, rapid cycling and past antidepressant-induced mania**, taper it if manic symptoms appear, and do not continue it long-term.

HOLD THIS

Increased activity plus mood is the entry criterion for mania; hours against days is the entry criterion for the borderline differential. In bipolar depression quetiapine leads, antidepressant monotherapy is out, and a mood stabiliser reduces switch without preventing cycle acceleration.

WHAT EARNS THE MARKS

- **The entry criterion quoted precisely**: activity or energy combined with mood is what the current framing requires.
- **Hours against days for the borderline differential**: affective shifts there last hours, hypomania at least 4 days.
- **The valproate effect size given as a number**: 0.16 against a comparator range of 0.37 to 0.56 settles the argument.
- **Doses declared as unverified where they are**: an invented starting dose costs more than a stated gap.

SEE ALSO 11 Mood and affect 29 Prognosis 30 Schizophrenia 32 Psychotic depression, OCD

32 · Psychotic depression, recurrent depression, and obsessive compulsive disorder

LONG CASES · TOPIC 32

This depression is psychotic. Combination or electroconvulsive therapy, and for how long at what dose?

LONG CASE, DEPRESSION; VIVA TABLE ON ELECTROCONVULSIVE THERAPY

THE SHAPE OF THE ANSWER

1	Separate the three psychoses	Timing of psychosis, delusional theme, inter-episode state
2	Name the trial	Combination beats either monotherapy, with the remission definition
3	Electroconvulsive therapy	Indications, the response comparison, then the statutory safeguards
4	Continuation and maintenance	Full acute dose, when the antipsychotic comes off, who is maintained
5	The obsessional ladder	Phenomena, insight, scale, then dose and duration

Two cases, one decision each. In psychotic depression **the decision is combination therapy or electroconvulsive therapy**, because neither class alone will do.

- **Key point.** In obsessive compulsive disorder the decision is **dose and duration**, because the commonest error is calling an adequate trial a failure at a depression dose.

1. Elicit the psychotic symptoms and separate them from schizophrenia and schizoaffective disorder.
2. Grade severity, and define recurrent depressive disorder.
3. Assess suicide risk and acute medical urgency.
4. State the trial evidence for combination against monotherapy.
5. Give the indications for electroconvulsive therapy and its comparative response rate.
6. State the statutory safeguards for electroconvulsive therapy.

7. Structure continuation, and say when the antipsychotic comes off.
8. Give the indications for maintenance and the dose you maintain.

FEATURE	SEVERE DEPRESSION WITH PSYCHOSIS	SCHIZOAFFECTIVE, DEPRESSIVE TYPE	SCHIZOPHRENIA WITH SECONDARY DEPRESSION
Timing of psychosis	**Only during the depressive episode**	**Persists in the absence of prominent mood symptoms**	**Chronic psychosis precedes the depression**
Delusional theme	Mood-congruent: guilt, poverty, somatic decay, deserved punishment	Often bizarre or mood-incongruent	Bizarre, persecutory or first-rank, unrelated to affect
Between episodes	Substantial or full recovery	Intermediate recovery	Persistent negative symptoms and cognitive decline

THE TRIAL TO NAME

The randomised double-blind trial of pharmacotherapy in psychotic depression enrolled 259 patients, half aged 60 and older, to olanzapine plus sertraline (n = 129) against olanzapine plus placebo (n = 130) for 12 weeks. Combination produced significantly higher remission at week 12, with comparable efficacy and tolerability in the older half.

Remission was a Hamilton Depression Rating Scale total below 17 with 2 consecutive ratings free of delusions. About half of all participants discontinued before week 12, a third of those withdrawn at week 5 under protocol rules.

Antidepressant monotherapy fails to clear the delusions and antipsychotic monotherapy fails to clear the affective syndrome, which is why the answer is both, or electroconvulsive therapy.

Electroconvulsive therapy. Indications, first half: severe psychotic depression with intense suicidality needing immediate containment; depressive stupor or catatonia; severe food and fluid refusal with dehydration.

Indications, second half: failure of adequate combination pharmacotherapy; high medical risk precluding high-dose drugs or requiring rapid response; and patient preference or documented past response.

Quote the comparison: favourable response in 86 per cent treated with electroconvulsive therapy against 42 per cent on a tricyclic and antipsychotic combination.

A meta-analysis of 44 physical treatment studies favoured it, with **bilateral over unilateral placement** and clear superiority over antidepressant monotherapy.

Statutory safeguards to state. Unmodified treatment is prohibited, so muscle relaxants and general anaesthesia are required.

Informed consent from the patient or nominated representative is needed and **emergency psychiatric treatment excludes it**. Its use in a minor requires the nominated representative's

consent with Mental Health Review Board permission.

Choosing the antipsychotic. Olanzapine has the randomised evidence with high metabolic liability. Quetiapine has randomised evidence and useful sedation but a warning added in 2011 on corrected QT prolongation with 12 co-prescribed drugs.

Ziprasidone is metabolically favourable but produced a mean corrected QT rise of 15 msec in an open trial of 19 patients. Aripiprazole preserves metabolic status, with 63 per cent response among 13 completers.

Continue the antidepressant at the full acute dose for 4 to 9 months, reassessing within 4 to 8 weeks of starting. Do not halve it, which is the classic wrong answer.

- **Concept.** **Taper the antipsychotic cautiously** after sustained euthymia and full resolution of psychosis.
- **Key point.** After electroconvulsive therapy, **relapse is rapid without prophylaxis**, and the best-supported regimen is lithium with nortriptyline.

Maintenance is indicated for 3 or more lifetime episodes, chronic depression, 2 severe episodes in 5 years, previous psychotic or highly suicidal episodes, and residual symptoms. It runs at the full acute dose for at least 1 year.

TRAP The numbered sections governing unmodified electroconvulsive therapy, its use in a minor and its exclusion from emergency treatment are not carried here, and neither is the minimum symptom-free interval that defines recurrent depressive disorder. **Read both off the classification and the bare Act and verify before the exam.** State the substance confidently and flag the number as one you will confirm.

Obsessive compulsive disorder. The ladder opens on phenomenology: elicit and define the phenomena, then separate them from ruminations, delusions and tics.

- **Concept.** Then **assess insight and give the specifiers**, and administer the Yale-Brown scale with its bands.
- **Concept.** Then **first-line pharmacotherapy and how the doses differ from depression**, and the definition of an adequate trial.
- **Concept.** The ladder closes on **exposure and response prevention, the augmentation algorithm, and maintenance duration with monitoring**.

PHENOMENON	WHAT IT IS	THE DISCRIMINATOR
Obsession	Recurrent intrusive idea, image or impulse	**Recognised as arising from the patient's own mind, ego-dystonic, resisted**
Depressive rumination	Mood-congruent theme of past failure or worthlessness	**Ego-syntonic reflection with no stereotyped neutralising act**
Delusion	Fixed unshakeable belief	**No internal struggle or resistance, often attributed to an external agency**
Motor tic	Sudden rapid recurrent movement or vocalisation	**Preceded by a premonitory urge, not goal-directed at averting a catastrophe**

- **Concept. One manual gives three insight specifiers** (good or fair, poor, absent with delusional beliefs) plus a tic-related specifier; the other gives two (fair to good, poor to absent).

The Yale-Brown scale and its children's version are semi-structured clinician-rated interviews. After a symptom checklist, 10 core items are scored, 5 for obsessions and 5 for compulsions.

The items cover **time, interference, distress, resistance and control**, each rated 0 to 4, giving a maximum total score 40.

- **RULE** **State the age scope when you quote the Indian figures.** The severity bands (mild 16 to 19, moderate 20 to 29, severe 30 to 40) and the Indian symptom-frequency and dose figures come from the children and adolescent guideline, scoped to people aged 16 years or younger. Adult Indian thresholds were not available in the sourced material, and saying so is stronger than presenting a paediatric cut-off as an adult one.

Dose and duration, the examinable core. Doses run 2 to 3 times higher than in depression: fluoxetine at least 40 to 60 mg daily, sertraline at least 200 to 300 mg daily, fluvoxamine at least 200 to 300 mg daily, paroxetine at least 40 to 60 mg daily.

An adequate trial is 8 to 12 weeks with at least 6 weeks at target dose. The Indian practice parameter holds the maximum tolerable dose for 12 weeks with stability at that peak for the preceding 3 weeks.

Declaring failure at 4 to 6 weeks on fluoxetine 20 mg or sertraline 50 mg is the error that exhausts first-line agents.

About 40 to 60 per cent respond to a first trial, **responders showing a 40 to 50 per cent fall in severity**.

INTERVENTION	RESPONSE AT 12 WEEKS	REMISSION AT 12 WEEKS	EFFECT SIZE
Exposure and response prevention alone	**70 per cent**	53 per cent	1.53 against waitlist, 0.93 against placebo
Combination with a reuptake inhibitor	66 per cent	49 per cent	**0.14 against therapy alone, not significant**
Reuptake inhibitor monotherapy	49 per cent	24 per cent	0.51 against placebo
Placebo	29 per cent	15 per cent	Reference
Waitlist	13 per cent	10 per cent	Reference

Exposure therapy has 9 components, from assessment and psychoeducation through hierarchy construction on a fear thermometer, graded exposure with strict response prevention, and work on family accommodation.

It runs **across 10 to 12 sessions of which 8 to 10 are active, each 50 to 60 minutes**. After a failed reuptake inhibitor, adding 17 sessions produced response in 56 per cent.

A low-dose dopamine D2 antagonist helps about one in three refractory patients: aripiprazole 5 to 10 mg daily, risperidone 1 to 3 mg daily, haloperidol 2.5 to 10 mg daily.

Assess at 4 weeks and stop if there is no response. Antipsychotics work better in moderate than extreme severity, olanzapine and quetiapine failed to separate from placebo as augmenters, and antipsychotic monotherapy can induce obsessions.

Maintain 12 months minimum at the remission dose, continuing a successful augmentation at least 1 year, since stopping at 3 months produces rapid relapse.

TRAP The usual last question is what follows when augmentation fails, and the expected answer is referral for neuromodulation or neurosurgery at a specialised centre after multidisciplinary review. The procedures, targets, response rates and selection criteria are not carried here. **Prepare them separately and verify before the exam**; do not improvise a target.

HOLD THIS

Psychotic depression takes two drugs or electroconvulsive therapy, at full dose, for 4 to 9 months. Obsessive compulsive disorder takes 2 to 3 times the depression dose for 8 to 12 weeks with at least 6 weeks high, and exposure therapy beats antipsychotic augmentation after a failed trial.

WHAT EARNS THE MARKS

- **Psychosis timed against the mood episode**: only during it, persisting without it, or preceding it, which names the three diagnoses.
- **Both drugs kept at full dose**: halving the antidepressant in continuation is the classic wrong answer.
- **The obsessional dose multiplier**: 2 to 3 times the depression dose, held 8 to 12 weeks with 6 weeks at target.
- **The age scope stated with the Indian severity bands**: they come from the child and adolescent guideline.

SEE ALSO 21 Suicide and violence 29 Prognosis 31 Mania and bipolar depression 33 Panic, anxiety, trauma

33 · Panic with agoraphobia, generalised anxiety, and the trauma disorders

LONG CASES · TOPIC 33

Take me through stepped care for this patient, with the review intervals and the maintenance duration.

LONG CASE, ANXIETY AND TRAUMA; VIVA TABLE ON STEPPED CARE

THE SHAPE OF THE ANSWER

1 Define and threshold	Worry for 6 months, attacks plus 1 month of anticipatory worry
2 Exclude the mimics	Coronary and respiratory emergency, phaeochromocytoma, substances
3 Stepped care in four steps	Identification, self-help, active treatment, specialist referral
4 Titration and review	Half the starting dose, then the interval schedule by disorder
5 The trauma contrast	Three clusters, then three self-organisation disturbances

Stepped care is the answer shape. Each of these cases is asked as **a ladder from recognition to specialist referral.**

- Key point. The examinable content is **the session arithmetic, the review intervals and the maintenance durations.**

1. Define the panic attack, panic disorder, agoraphobia and generalised anxiety disorder.
2. Give the duration thresholds and the screening instruments.
3. Exclude medical mimics and substances, and sequence comorbidity.
4. Manage an acute attack in casualty.
5. Give the cognitive model and explain interoceptive exposure.
6. Set out stepped care for each disorder.
7. Initiate and titrate the drug, and handle early activation.
8. State the position on benzodiazepines, beta-blockers and pregabalin.
9. Give review intervals, maintenance duration and the taper.

Generalised anxiety disorder is excessive uncontrollable worry across domains on most days for at least 6 months.

Panic disorder is recurrent unforeseen attacks followed by at least 1 month of anticipatory worry, fear of consequences or behavioural change, with at least 2 unexpected attacks.

- Concept. **Agoraphobia is fear and avoidance of situations from which escape might be difficult or help unavailable.**
- Key point. The discriminator against social anxiety disorder is **what drives the avoidance**: inability to escape or get help, rather than negative evaluation. Against phaeochromocytoma it is the anticipatory cognitive dread.
- Concept. In casualty, **exclude an acute coronary or respiratory emergency** and do not admit an uncomplicated attack.

STEP	GENERALISED ANXIETY DISORDER	PANIC DISORDER
1	Identification, assessment, psychoeducation, active monitoring	Recognition, comorbidity timeline, minimal investigation in casualty
2	**Non-facilitated self-help over at least 6 weeks with no more than 5 minutes of therapist contact; guided self-help across 5 to 7 sessions of 20 to 30 minutes; psychoeducational groups of 1 therapist to about 12 participants, 6 weekly sessions of 2 hours**	**Mild to moderate illness: facilitated or non-facilitated self-help, support groups, structured exercise**
3	**Individual cognitive behavioural therapy or applied relaxation, 12 to 15 weekly sessions of 1 hour; or a reuptake inhibitor, sertraline first on cost-effectiveness**	**Moderate to severe illness: cognitive behavioural therapy of 7 to 14 hours total, weekly sessions of 1 to 2 hours within a maximum of 4 months; or a licensed reuptake inhibitor**
4	Complex refractory illness with self-neglect or suicide risk: specialist teams, combination therapy or admission	Specialist referral if significant symptoms persist after 2 completed interventions, then home-based therapy where agoraphobia prevents attendance

Titration and review. Antidepressants are the only recommended long-term class for both disorders.

Start at half the usual starting dose to prevent the activation syndrome of transient agitation, worsened panic and insomnia, and warn the patient. Full response takes several weeks, up to 3 months.

Patients under 30 years carry raised risk of treatment-emergent suicidal ideation and are **reviewed within 1 week and weekly for the first month**.

Review panic disorder at 2, 4, 6 and 12 weeks, then at 8 to 12 week intervals. Review generalised anxiety disorder every 2 to 4 weeks for 3 months and every 3 months after.

Maintain at least 1 year in generalised anxiety disorder and at least 6 months after optimal response in panic disorder, then taper gradually.

With no improvement after a full 12-week course, **verify adherence and diagnosis, then switch within or across class**.

Benzodiazepines should not be prescribed in panic disorder, and in generalised anxiety disorder are restricted to crisis use not normally exceeding 2 to 4 weeks. Propranolol is not recommended for either.

- Key point. **Pregabalin is for intolerance of both reuptake inhibitor classes.** It works within the first week, and it carries abuse liability, withdrawal and a pregnancy malformation signal.

TRAP The symptom-count criterion for a formal panic attack, that is how many of how many listed symptoms are required, is not carried here, and neither are the starting doses for propranolol, pregabalin, imipramine, clomipramine and venlafaxine. **Confirm each in the manual or a primary dosing reference and verify before the exam** rather than guessing a count.

- **Concept. The current framing keeps three clusters and tightens each.** Re-experiencing *in the present* through vivid intrusions, flashbacks or nightmares with fear or horror; deliberate avoidance of internal and external reminders; and a persistent sense of current threat shown by hypervigilance or an accentuated startle.
- **Concept. The older framing also had three clusters**, named intrusive thoughts or images, avoidance and hyperarousal.
- **Key point. So the shift is threefold**: re-experiencing rather than recall, hyperarousal narrowed to current threat, and the affective, self-concept and relational disturbances moved into the new complex category.
- **Concept. Complex post-traumatic stress disorder needs the three core clusters plus three disturbances in self-organisation**: affect dysregulation, persistently negative self-concept, and difficulty sustaining relationships.
- **Key point.** Against borderline personality disorder the discriminators are **a mandatory trauma history, mandatory re-experiencing, a *stable* pervasive negative self-view rather than a shifting identity**, and consistent relational avoidance rather than chaotic idealisation and devaluation.

TWO ANSWERS THAT SEPARATE CANDIDATES

Do not accept that therapy is proven superior to medication. Only 4 head-to-head randomised trials exist, 2 at high risk of bias, and meta-analysis of the 2 low-risk trials found no significant difference.

The indirect comparisons that favour therapy are confounded by absent blinding, which inflates standardised mean differences by 0.37 for subjective outcomes, by wait-list controls which roughly double effect sizes, by median sample sizes of 74 against 169 with small studies inflating effect by about 0.21, and by contrasting completed therapy with discontinued medication.

Second, latent profile analysis of 581 patients found three roughly equal groups, one matching core post-traumatic stress disorder at 37 per cent, one matching the complex category at 36 per cent, and a low quality of life group at 27 per cent, so severe trauma does not inevitably produce either diagnosis.

HOLD THIS

Half the usual starting dose, review under 30 years within 1 week, maintain generalised anxiety disorder a year and panic disorder six months. In trauma, three clusters plus three self-organisation disturbances makes the complex diagnosis, and head-to-head trials show no proven superiority of therapy over a reuptake inhibitor.

WHAT EARNS THE MARKS

- **Half the usual starting dose, with the reason**: the activation syndrome is what the warning to the patient is about.
- **The under-30 review interval**: within 1 week and weekly for the first month, because of treatment-emergent ideation.
- **The benzodiazepine position stated plainly**: not for panic disorder, and crisis use only in generalised anxiety.
- **Therapy not claimed as superior to medication**: only 4 head-to-head trials exist, and the low-risk pair found no difference.

SEE ALSO 18 Vitals and systems 24 ICD-11 and DSM-5-TR 32 Psychotic depression, OCD 36 Dissociation and borderline

34 · Alcohol dependence with complications, and opioid dependence

LONG CASES · TOPIC 34

This patient stopped drinking 30 hours ago. What do you expect, and what do you prescribe?

LONG CASE, SUBSTANCE DEPENDENCE; VIVA TABLE ON WITHDRAWAL TIMELINES

THE SHAPE OF THE ANSWER

1 Diagnose dependence	3 of 6 features in a year, then quantify and screen
2 Decide the setting	Admission criteria, from unit count to comorbidity and support
3 Run the withdrawal clock	Four syndromes by onset, each with its discriminator
4 Prescribe and protect	Benzodiazepine choice and taper, then thiamine before glucose
5 Relapse prevention	Anticraving drugs with monitoring, then motivational work

Both substance cases are timelines. Dependence is a criterion count, the acute case runs against a withdrawal clock, and the long case ends on relapse prevention with its monitoring.

1. Diagnose dependence and separate it from harmful use.
2. Quantify intake and screen, then investigate.
3. Decide inpatient or outpatient detoxification.
4. Give the withdrawal timeline including seizures and delirium tremens.
5. Select, dose and taper the benzodiazepine, adjusting for liver disease.
6. Manage seizures and delirium tremens at the bedside.
7. Give thiamine dosing, routes and cofactors.
8. Give the anticraving drugs with their monitoring and contraindications.
9. Structure motivational work across the stages of change.

Dependence needs 3 or more of 6 features together at some time in the previous 1 year: craving, impaired control, a withdrawal state or use to relieve it, tolerance, progressive neglect of alternatives, and persistence despite clear harm. Harmful use is damage without those criteria.

The four-item CAGE takes 2 positive answers as a strong indication and 3 as evidence of dependence. On the ten-item AUDIT, 8 or more indicates hazardous or harmful use and 16 and above a high level of problems.

Indian figures. Alcohol is the primary substance other than tobacco in 21.4 per cent of those surveyed, and 17 to 26 per cent of drinkers are dependent, for a national prevalence near 4 per cent.

Alcohol accounts for **43.9 per cent of treatment seekers at government de-addiction centres**.

Admit for severe dependence, meaning over 30 units daily, or 15 to 30 units with comorbidity.

Admit also for **anticipated severe or complicated withdrawal, past seizures or delirium tremens, decompensated cirrhosis**, suicide risk, concurrent sedative or opioid dependence, poor support, past outpatient failure, old age or pregnancy.

SYNDROME	ONSET AFTER THE LAST DRINK	THE DISCRIMINATOR
Simple withdrawal	6 to 8 hours, peaking at 48 to 72 hours and subsiding by the fourth to fifth day	**Tremor, sweating, tachycardia, hypertension, insomnia, clear sensorium**
Alcoholic hallucinosis	8 to 12 hours	**Hallucinations, typically accusatory voices, with an entirely clear sensorium and intact orientation**
Withdrawal seizures	12 to 24 hours, recurring 3 to 6 hours after the first	**Generalised tonic-clonic in clusters; status epilepticus is relatively rare, and phenytoin does not prevent them**
Delirium tremens	2 to 5 days, and may emerge up to 7 days	**Clouded consciousness, coarse tremor, vivid visual hallucinations, autonomic storm; 3 to 5 per cent of dependent drinkers**

Benzodiazepine equivalents: diazepam 10 mg, chlordiazepoxide 25 mg, lorazepam 2 mg, oxazepam 30 mg.

Lorazepam and oxazepam escape phase-one hepatic oxidation and are preferred in liver disease, cognitive impairment and old age, with doses reduced by 50 per cent there.

Fixed schedule: chlordiazepoxide 15 mg to 50 mg four times daily (60 to 200 mg total daily), or diazepam 10 mg to 25 mg three times daily (30 to 75 mg total daily).

That schedule is **held for the first 2 days then tapered to zero over 7 to 10 days at 20 per cent daily**.

Symptom-triggered dosing runs off the CIWA-Ar: 10 items, 9 scored 0 to 7 and orientation 0 to 4, above 20 severe, giving 10 to 20 mg diazepam equivalent per trigger.

Symptom-triggered dosing is **unsuitable in community care or with past withdrawal seizures**.

For a seizure, intravenous lorazepam 2 mg at once, then a taper. Antiepileptics are for an independent seizure disorder.

For delirium tremens, intravenous diazepam 10 mg repeated at 5 to 20 minute intervals until calm, escalating to 20 mg per bolus after the first 2.

THIAMINE, AND THE GLUCOSE RULE

Prophylaxis in uncomplicated withdrawal: parenteral thiamine at least 250 mg once daily for the first 3 to 5 days, then oral thiamine for a minimum of 3 months. Suspected Wernicke encephalopathy: parenteral thiamine 500 mg daily for 3 to 5 days, then oral.

International regimens to name: 500 mg intravenously three times daily (1500 mg daily) for 3 days then 250 mg once daily for 5 days, or 200 mg intravenously three times daily (600 mg daily) until symptoms resolve, with oral maintenance of 50 mg to 100 mg daily.

Parenteral glucose is not given without concurrent thiamine, because glucose utilisation exhausts residual stores and precipitates acute Wernicke encephalopathy. Hypomagnesaemia produces thiamine-refractory disease, so replace magnesium 10 to 30 mEq, potassium 60 to 180 mEq and phosphate 10 to 40 mmol/L per day.

RULE **Three confusions, three discriminators.** Delirium tremens has an autonomic storm and coarse tremor; Wernicke encephalopathy has quiet confusion, nystagmus, abducens palsy and truncal ataxia without the storm; hepatic encephalopathy has asterixis, fetor hepaticus and jaundice, and benzodiazepines will worsen it.

ANTICRAVING DRUG	DOSE	MONITORING AND THE DISCRIMINATOR
Acamprosate	333 mg tablets; 1332 mg daily under 50 kg, 1998 mg daily above 50 kg	**Renally excreted, so safe in mild to moderate hepatic disease but contraindicated when serum creatinine exceeds 120 micromoles per litre**
Naltrexone	50 mg once daily orally	**Contraindicated in acute liver failure, caution when transaminases exceed 4 to 5 times normal; liver function at baseline, monthly for 3 months, then quarterly**
Disulfiram	250 mg once daily, raised to 500 mg or 750 mg if no reaction occurs and adherence is verified	**Written informed consent is mandatory, start at least 24 hours after the last drink, reaction risk persists 2 weeks after stopping, liver function every 2 weeks for 2 months then quarterly**
Baclofen	Individualised	**The off-label choice where cirrhosis and significant anxiety coexist**

Opioid dependence. Present the agent, route, daily quantity, duration, consequences and the time of the last dose.

Withdrawal begins 6 to 12 hours after a short-acting opioid and 24 to 72 hours after a long-acting one, with an acute syndrome of about 5 days for the former and a protracted course for the latter.

In Indian cohorts starting buprenorphine, **prescription opioids account for 53 per cent and illicit opioids 38 per cent**, with lifetime injecting in 35 per cent and psychiatric comorbidity in 32 per cent.

DECISION	FIGURE	THE DISCRIMINATOR
When to induce	Withdrawal scale 13 or more, or 6 or more with mydriasis and piloerection; 6 to 12 hours after a short-acting opioid, 24 to 72 hours after a long-acting one	**Objective signs, not the patient's distress**
Pharmacology	Half-life 37 hours, range 24 to 69 hours; agonist ceiling above 24 mg daily; naloxone co-formulated 4:1	**The ceiling limits respiratory depression, and sublingual naloxone deters injection only**
Induction	2 mg to 4 mg on the first day, reassess after 1 to 3 hours, up to 5.7 mg with 1.4 mg naloxone or 8 mg with 2 mg; up to 11.4 mg with 2.9 mg, or 16 mg with 4 mg, on the second day	**Precipitated withdrawal comes on within 1 to 3 hours of the dose and is not under-dosing**
Microdosing	0.5 mg twice daily under a continuing full agonist, titrated over 5 to 7 days to 12 mg	**For patients who cannot tolerate an abstinence window**
Maintenance in India	Mean 6.7 mg daily; adherent 7.7 mg against 4.3 mg; 6 mg or less gave 39 per cent non-adherence against 17 per cent, an odds ratio of 3	**Historical guidance of 4 mg to 8 mg under-doses; titrate above 6 mg**
Clonidine	Test dose 50 mcg, repeated after 1 hour if systolic pressure stays above 90 mm Hg; then 3 to 4 mcg/kg four times daily (200 to 250 mcg for 70 kg); daily range 0.5 mg to 1.4 mg	**Withhold below 90 mm Hg systolic; it does not touch bone aches, insomnia or craving, and outpatient supply is limited to 3 days**
Tramadol extended release	103 patients on a 7-day taper: completion 90 per cent with buprenorphine, 72 per cent with tramadol, 66 per cent with clonidine	**Comparable to buprenorphine where partial agonists are restricted**

TRAP The reliable follow-up on thiamine is the administration precaution: anaphylactoid reaction risk, infusion rate and dilution, and resuscitation facilities on standby. That is not carried here, and neither is the opioid-free interval required before naltrexone nor the naloxone challenge dose. **Quote a local protocol and verify before the exam** rather than a remembered infusion time.

HOLD THIS

Alcohol: 3 of 6 criteria in a year, thiamine before glucose, lorazepam or oxazepam in liver disease with doses halved. Opioids: induce on objective signs at 6 to 12 hours, and titrate above 6 mg of buprenorphine, because 6 mg or less triples the odds of dropping out.

WHAT EARNS THE MARKS

- **The withdrawal clock given by hours**: 6 to 8, 8 to 12, 12 to 24, then 2 to 5 days, each with its syndrome.
- **Thiamine before glucose**: glucose utilisation exhausts residual stores and precipitates acute Wernicke encephalopathy.
- **Lorazepam or oxazepam in liver disease**: they escape phase-one oxidation, and the dose is halved there.
- **Buprenorphine induced on objective signs**: a withdrawal score with mydriasis and piloerection, not the patient's distress.

SEE ALSO 08 Substance, sexual, marital 18 Vitals and systems 26 For, against, comorbidity 35 Delirium and dementia

35 · Delirium, and the dementia long case

LONG CASES · TOPIC 35

This elderly patient is confused. Is it delirium or dementia, and what do you do first?

LONG CASE, ORGANIC PSYCHIATRY; SHORT CASE AND VIVA ON COGNITIVE SCREENS

THE SHAPE OF THE ANSWER

1 Confirm delirium	Four features, with the rule for a positive diagnosis
2 Subtype and the three-way contrast	Hypoactive is missed, then delirium against dementia and depression
3 Treat the cause, not the behaviour	Multicomponent care first, then the haloperidol evidence
4 The dementia history	Informant, onset, course, functional decline, bedside battery
5 Subtype and manage	Course sorts the subtype, then behavioural symptoms and the law

One is an acute medical emergency dressed as psychiatry; the other is a history taken from somebody else.

1. Confirm delirium on the Confusion Assessment Method, then list predisposing and precipitating causes.
2. Identify the motoric subtype and say why the quiet one is missed.
3. Separate delirium from dementia and depression.
4. Give the workup, the non-pharmacological package and the haloperidol evidence.
5. Give geriatric doses, and say when a benzodiazepine is indicated.

6. Assess the informant, then establish onset, course and functional decline.
7. Administer a bedside battery with its cut-offs, and elicit the neurological signs.
8. Separate the four dementia subtypes and exclude reversible causes.
9. Manage behavioural symptoms, prescribe with monitoring, and cover carer burden and the law.

The Confusion Assessment Method takes under 5 minutes and has 4 features: acute onset with fluctuating course, inattention, disorganised thinking, and altered level of consciousness.

A positive diagnosis needs features 1 and 2 plus either 3 or 4. An intensive care version exists for intubated and non-verbal patients.

Delirium affects **10 to 30 per cent of hospitalised older adults**.

Thresholds worth carrying. Oral intake below 1200 mL per 24 hours is dehydration, and saturation below 90 per cent is hypoxia.

Post-void residual above 200 mL needs intermittent catheterisation at 8-hour intervals until it falls below 200 mL.

- **Trap.** Missing the hypoactive subtype, **misread as depression, dementia or ageing**. A quiet patient disrupts no ward routine, so the underlying urosepsis or hypoxaemia goes undetected.

FEATURE	DELIRIUM	DEMENTIA	DEPRESSION
Onset	**Hours to days**	Months to years	Weeks to months
Consciousness	**Altered and clouded**	Clear until late	Intact
Attention	**Markedly impaired and fluctuating**	Preserved until late	Reduced by low effort
Self-awareness	Fluctuating awareness of confusion	**Unaware, conceals deficits**	**Distressed, complains of memory loss**
Reversibility	**Usually reversible once the cause is treated**	Rarely reversible	Reversible with mood recovery

THE HALOPERIDOL ANSWER

A systematic review and meta-analysis of 6 randomised trials in 2863 intensive care patients found no significant reduction in short-term mortality (odds ratio 0.89, confidence interval 0.60 to 1.32) or long-term mortality (odds ratio 0.87, confidence interval 0.70 to 1.07), and no significant difference in delirium resolution.

It shortened intensive care stay by just over a day, with no effect on total hospital stay, and prophylactic haloperidol did not improve survival at 28 days in high-risk critically ill adults. Haloperidol stabilises behaviour in a crisis; it does not treat delirium.

Doses where behaviour poses immediate risk. Oral haloperidol 0.5 to 1 mg at 2 to 4 hour intervals to a maximum of 2.5 mg in the first 24 hours, or injectable 0.25 to 0.5 mg to a

maximum of 2 mg.

The alternatives: **risperidone 0.25 to 0.5 mg to a maximum of 2 mg; olanzapine 2.5 to 5 mg to a maximum of 10 mg; quetiapine 12.5 to 50 mg to a maximum of 100 mg.**

- Key point. In Parkinson disease and Lewy body dementia, **high-potency D2 antagonists are contraindicated and quetiapine is the oral agent of choice**.

Benzodiazepines worsen general medical delirium. The single exception is withdrawal delirium, where thiamine 100 mg daily for 3 days and folic acid 5 mg daily precede intravenous glucose.

- Concept. **Cholinesterase inhibitors are not used here.**
- Key point. **Non-pharmacological multicomponent care is first-line**: reorientation aids, spectacles and hearing aids, protected sleep, early mobilisation, family presence, and avoidance of restraints.

ITEM	RANGE AND CUT-OFF	THE DISCRIMINATOR
Mini-Mental State Examination	0 to 30 points, fewer than 24 indicates impairment, 5 to 10 minutes	**Sensitivity 71 to 92 per cent, specificity 56 to 96 per cent, and heavily education-dependent, so false positives in illiterate patients and false negatives in highly educated ones**
Montreal Cognitive Assessment	10 to 15 minutes	**Higher sensitivity for executive and visuospatial deficits, so better early and in mild cognitive impairment**
Blessed Information Memory Concentration	0 to 28 errors, more than 10 errors indicates impairment	**Sensitivity 90 per cent**
Short Test of Mental Status	Total score 38, a score of 29 or lower indicates impairment	**Tests calculation, copying and abstraction**
Functional Activities Questionnaire	10 activities scored 0 to 30, cut-off 9	**Informant-rated, so it measures dependency rather than test performance**

Subtypes by share. Alzheimer disease is 50 to 60 per cent of dementias, vascular 15 to 20 per cent, mixed 10 to 20 per cent, other causes under 10 per cent.

- Key point. **Course sorts them.** Insidious and smoothly progressive with early episodic memory loss and medial temporal atrophy is Alzheimer disease.
- Concept. **Stepwise with executive deficits, focal signs, white matter hyperintensities and lacunar infarcts is vascular.**
- Concept. **Fluctuating attention over hours with well-formed visual hallucinations**, rapid eye movement sleep behaviour disorder and spontaneous parkinsonism is Lewy body disease.

Early disinhibition, apathy and language breakdown between 45 and 64 years with asymmetric frontal and anterior temporal atrophy is **frontotemporal dementia, the one misdiagnosed as late-onset mania**.

The 1-year rule separates Lewy body disease from Parkinson disease dementia. In Lewy body disease cognition fails before, with or within 1 year of parkinsonian signs; in Parkinson disease

dementia it follows motor disease by at least 1 year.

Up to 50 per cent of Lewy body patients show **catastrophic neuroleptic sensitivity**.

Behavioural symptoms affect 60 to 80 per cent at any time, with cumulative lifetime risk above 90 per cent.

- Key point. **Non-pharmacological management is first-line**: the PAIN screen for physical illness, activity-related friction, iatrogenic drug effects and sensory overload, and the antecedent-behaviour-consequence framework.
- Concept. **Prescribe only for severe distress or immediate risk, and start low.**

Cholinesterase inhibitors in mild to moderate disease: donepezil 5 to 10 mg daily with pulse monitoring for bradycardia, rivastigmine 3 to 12 mg daily divided, galantamine 8 to 24 mg daily. Memantine 5 to 20 mg daily in moderate to severe disease.

Risperidone 0.25 to 2 mg daily is the first-line antipsychotic for severe aggression or psychosis with risk of harm, and quetiapine 12.5 to 300 mg daily is first-line in Lewy body and Parkinson disease.

Atypical antipsychotics carry **roughly a threefold increase in stroke risk**.

Review any antipsychotic at least every 6 weeks, withdraw over 4 weeks or more if there is no benefit, re-justify continuation beyond 3 months, and treat maintenance beyond 12 months as carrying cumulative risk.

THE INDIAN FRAME FOR A DEMENTIA CASE

Name three statutes and what each gives you. The Mental Healthcare Act provides for an advance directive and a nominated representative while capacity is intact. The Rights of Persons with Disabilities Act recognises chronic neurological and intellectual impairment, and certification at the benchmark of 40 per cent disability opens welfare concessions and travel and tax benefits.

The Maintenance and Welfare of Parents and Senior Citizens Act gives a summary mechanism through maintenance tribunals to secure maintenance, lodging and care from children or legal heirs, and permits invalidation of property transfers where elderly parents are neglected.

Then add what decides outcome here: institutional care is scarce and stigmatised, so caregiving rests on spouses and adult children, and the plan must screen the carer for burnout and depression and cover wander prevention, identity bracelets, grab bars and non-slip flooring.

TRAP The numeric stage anchors of the global severity instrument, and the Global Deterioration Scale or Reisberg stages, are not carried here, nor are the years of enactment of those three Acts, nor the expected electroencephalogram findings in delirium, nor the formal diagnostic criteria for delirium as distinct from the screening algorithm. **Verify each before the exam.**

HOLD THIS

Features 1 and 2 plus either 3 or 4 makes delirium, and haloperidol shortens the intensive care stay without changing mortality or delirium duration. In dementia, course sorts the subtype, the 1-year rule sorts Lewy body from Parkinson disease dementia, and neuroleptic sensitivity makes quetiapine the safe choice there.

WHAT EARNS THE MARKS

- **The diagnostic rule stated, not the four features alone**: features 1 and 2 plus either 3 or 4 is what makes it positive.
- **The haloperidol evidence given honestly**: it shortens intensive care stay without changing mortality or delirium resolution.
- **Course used to sort the dementia subtype**: smooth, stepwise, fluctuating, or frontal-onset, each with its imaging correlate.
- **Three Indian statutes named with what each gives**: the directive and representative, disability certification, and the maintenance tribunal.

SEE ALSO 15 Cognitive examination 19 Neurological screen 27 Investigations 37 Eating, disability, postpartum

36 · Dissociation, bodily distress, and the borderline pattern

LONG CASES · TOPIC 36

These convulsions are not epileptic. How do you say so, and what do you do instead of prescribing?

LONG CASE, DISSOCIATION AND PERSONALITY; VIVA TABLE ON FUNCTIONAL SIGNS

THE SHAPE OF THE ANSWER

1 Place dissociation	Trance and possession across the classifications, agency as the axis
2 Separate from epilepsy	Video electroencephalography, prolactin as adjunct, interictal spikes
3 Map the gains, then treat	Primary, secondary, tertiary, then the acute and long-term plan
4 Bodily distress disorder	Rule-in diagnosis, six essential features, three acronym traps
5 The borderline pattern	Grade severity, name trait domains, stratify therapy by severity

Three cases that punish the candidate who reaches for a drug first.

1. Define dissociation and place trance and possession in each classification.
2. Give the Indian epidemiology, then separate dissociative convulsions from epilepsy.
3. State the role of video electroencephalography and post-ictal prolactin.
4. Separate pathological possession from culturally sanctioned trance, and explain shared trance.
5. Map primary, secondary and tertiary gain, then give the acute and long-term plan.
6. State the conceptual shift to bodily distress disorder and its essential features.
7. Separate it from hypochondriasis, dissociative neurological symptom disorder and somatic passivity.
8. Give the modern communication strategy and the stepped management.
9. Grade personality dysfunction, give the trait domains, and stratify therapy by severity.

- **Concept. Trance and possession sits at F44.3 in one classification** and within the dissociative disorders in another, while the third subsumes it under dissociative identity disorder in possession form.
- **Key point. Possession is a disruption of *agency***, the experience of owning and controlling one's own thoughts and actions. That separates it from thought insertion, where alien thoughts enter a preserved identity.
- **Concept. Pathological trance is involuntary, distressing, contextually inappropriate and impairing.** Sanctioned ritual trance resolving without distress is not pathological.
- **Concept. Shared trance spreads by modelling from an index case**, and it is amplified when adults validate paranormal explanations.
- **Concept. The gains.** Primary keeps intolerable affect out of awareness, secondary is escape plus attention, tertiary is the family benefit.
- **Key point. Acute management is a quiet room and protection from injury without aggressive restraint.** No intravenous antiepileptic bolus once the diagnosis is clinically made, and clustered cases are separated.
- **Concept. Frontal lobe epilepsy is separated by stereotypy, brief duration, onset from sleep and absent emotional triggers.**

ITEM	FIGURE	THE DISCRIMINATOR
Indian service data over 10 years	893 patients, 591 outpatients (66 per cent) and 302 inpatients (34 per cent); 1.5 to 15.0 per 1,000 outpatients	**Motor disorder leads at 43.3 per cent of outpatients and 37.7 per cent of inpatients, then convulsions at 23.0 and 27.8 per cent**
Possession in West Karnataka	91 per cent found the possessing entity troublesome and distressing	**Clinical possession is unwanted suffering, and multiple-personality presentations are rarely diagnosed here**
Post-ictal prolactin at 10 to 20 minutes	Above 500 IU/mL in 90 per cent of generalised tonic-clonic and 60 per cent of focal impaired awareness seizures, specificity 73 per cent; capillary sensitivity 67.3, 33.9 and 11.1 per cent, median specificity 99.1 per cent	**An adjunct, not a decider: focal aware seizures and prolonged status leave it normal, and rises follow dissociative events and syncope**
Interictal electroencephalogram	Spike-and-wave discharges appeared in 3 siblings with shared possession trance	**Without ictal concordance it is not epilepsy, and starting an antiepileptic on it is the error**
Psychological course	6 sessions, resolution within 1 week, stability at 3-month follow-up; clonazepam 0.25 mg for acute containment only	**Separation of clustered cases plus a gain-reduction model and grounding**

Bodily distress disorder. The conceptual shift is the answer. The older framework required symptoms to be medically unexplained, which enforced dualism and asked clinicians to prove a negative, leaving somatisation disorder at a reported primary care prevalence of about 0.1 per cent.

The current framework makes it a rule-in diagnosis at code 6C20 with 6 essential features. Distressing bodily symptoms; excessive attention, shown as preoccupation or as healthcare visits far beyond what is indicated; persistence of that attention despite appropriate examination and reassurance.

The remaining features: **symptoms on most days for at least 3 months; functional impairment; and no better explanation in another disorder.** Coexisting physical disease is permitted where the attention is clearly excessive.

The comparison instrument requires **persistence beyond 6 months at a prevalence of 5 to 7 per cent**.

- **Key point.** Against hypochondriasis at 6B23 the discriminator is **what the patient wants, reassurance against relief**.
- **Concept.** Against dissociative neurological symptom disorder at 6B60 it is **positive signs of internal inconsistency such as the Hoover sign**. Against somatic passivity it is intact reality testing.
- **Trap.** Forcing reattribution. **Validate the suffering, refuse the dichotomy, and explain hypervigilance as an amplifier on genuine signals**, then renegotiate toward functional recovery with one designated physician and fixed-interval visits.

- **RULE** **Three acronym traps, three answers.** Bodily distress *disorder* is the diagnosis at 6C20 and needs positive psychobehavioural features. Bodily distress *syndrome* is a research construct grouping functional somatic syndromes into 4 clusters and requires medical inexplicability. Bodily *stress* syndrome is a primary care proposal needing 3 unexplained symptoms with impairment. Body dysmorphic disorder, which shares the initials, is coded 6B21 under the obsessive-compulsive group.

- **Key point.** **The borderline pattern.** The current classification abandons the categorical types for a single dimensional severity judgement, retaining one qualifier, the borderline pattern, to preserve continuity of care and referral to psychotherapy.
- **Concept.** **Formulate in four steps.** Confirm the general requirements; grade severity; identify prominent trait domains among negative affectivity, detachment, disinhibition, dissociality and anankastia; then apply the qualifier.

The qualifier's 4 components are **affective instability, unstable self-concept with chronic emptiness, frantic efforts to avoid abandonment, and impulsive or self-harming regulation**.

- **Concept.** **Anankastia replaced psychoticism** because odd beliefs and perceptual aberration belong to the schizophrenia spectrum.
- **Key point.** On drugs, say plainly that **no agent is established to alter the core pathology**, that prescribing is adjunctive and secondary to structured psychotherapy, and that the sourced material here carries no agent or dose to quote.
- **Trap.** The long admission. **Keep admission brief and structured around a safety plan**, because long unstructured admissions produce regression, dependency and contagion.

ITEM	FIGURE	THE DISCRIMINATOR
Severity tiers	5 tiers: no impairment, personality difficulty, then mild, moderate and severe disorder	**The first 2 tiers are not mental disorders, and severity rather than type predicts impairment and mortality**
Prevalence	12.16 per cent Western and 4.1 per cent Asian community samples; borderline personality disorder 0.7 to 2.7 per cent; 46 to 58 per cent in clinics	**In emergency departments it accounts for 35 per cent of suicide attempts and 22 per cent of self-harm presentations**
Adolescence	Permitted at any age if the pattern has persisted 2 years; cumulative prevalence 25.7 per cent by age 22	**Only 1 per cent of youth in primary services receive it, against 11 to 22 per cent of outpatients and 33 to 49 per cent of inpatients**
Severity instrument	14 items, cut-offs 12 mild, 16 moderate, 19 severe	**Below 12 is sub-clinical difficulty**
Dialectical behaviour therapy	4 modes: individual therapy, skills group, telephone coaching, consultation team	**Mild disorder can take skills plus consultation team; moderate and severe need all 4, because the target is life-threatening behaviour**
Alternatives	A Cochrane update across 1804 participants in 28 studies	**Mentalisation-based treatment, transference-focused therapy, schema-focused therapy and a 20-week group programme involving carers; none has a very robust evidence base**

TRAP Two examiner-favourite discriminators here are not carried by the sourced material: factitious disorder and malingering against bodily distress disorder, and the culture-specific somatic idioms including Dhat syndrome. **Source both from a classification text and verify before the exam.**

HOLD THIS

Agency distinguishes possession from thought insertion; prolactin is an adjunct and not a decider; and an interictal spike is not epilepsy without ictal concordance. Bodily distress disorder is rule-in at 3 months, and in the borderline pattern severity sets the intensity of therapy while pharmacotherapy stays adjunctive.

WHAT EARNS THE MARKS

- **Agency named as the axis in possession**: it is what separates possession from thought insertion.
- **Prolactin offered as an adjunct**: it rises after dissociative events and syncope, and focal aware seizures leave it normal.
- **Bodily distress disorder framed as rule-in**: six positive features, so no one is asked to prove a negative.
- **Severity used to set therapy intensity**: mild disorder can take skills plus consultation team, moderate and severe need all four modes.

SEE ALSO 14 Perception 19 Neurological screen 24 ICD-11 and DSM-5-TR 33 Panic, anxiety, trauma

37 · Eating disorders, intellectual disability, and the postpartum long case

LONG CASES · TOPIC 37

This patient is severely underweight and about to be fed. What are you watching for, and how do you prevent it?

LONG CASE, EATING AND PERINATAL PSYCHIATRY; SHORT CASE ON INSTABILITY SIGNS

THE SHAPE OF THE ANSWER

1 Diagnose and grade	Anorexia against bulimia, then the shared core psychopathology
2 Find instability at the bedside	Temperature, pulse, posture, wasting, parotids, calluses, oedema
3 Refeeding syndrome	Definition, mechanism, timing, risk stratification, prevention
4 Intellectual disability	Cut-off, strata, overshadowing, functional analysis, certification
5 The postpartum case	Exclude organic causes, assess infant risk, then the stepwise algorithm

Three cases where the physical examination decides the answer.

1. Distinguish anorexia from bulimia and give the shared core psychopathology.
2. Give the bedside signs of instability and the criteria for emergency admission.
3. Define refeeding syndrome, its mechanism and timing, and stratify the risk.
4. Set out family-based treatment and enhanced cognitive behaviour therapy.
5. Define intellectual disability, its developmental cut-off and severity strata.
6. Explain diagnostic overshadowing, then conduct a functional assessment.
7. Give the evidence for positive behavioural support and for drugs.
8. Cover disability certification and guardianship.
9. Classify the postpartum disorders, assess infanticide and suicide risk, and give the algorithm and lactation prescribing.

Anorexia nervosa is restriction producing significantly low weight, an adult body mass index below 18.5 kg/m2 or the corresponding centile, with intense fear of weight gain or behaviour interfering with gain, and body image disturbance.

Bulimia nervosa is recurrent binge eating with loss of control plus compensatory behaviour at normal weight or above. Where both occur at significantly low weight the hierarchy makes it anorexia nervosa, binge-eating and purging subtype.

- Key point. **The shared core is overvaluation of shape and weight as the basis of self-worth.**

Markers of instability: hypothermia, bradycardia below 60 beats per minute, postural hypotension or tachycardia above 100.

- Concept. The rest of the instability set: **severe wasting, painless parotid swelling, dorsal hand calluses, oedema, delirium, ataxia and seizures**.

REFEEDING SYNDROME, THE EXAMINABLE CORE

The consensus definition is acute electrolyte deficiency and metabolic shift on reintroducing nutrition to a malnourished person, and isolated hypophosphataemia after refeeding is sufficient for the diagnosis.

In a cohort of 69 hospitalised adolescents, moderately severe refeeding syndrome occurred in 6 per cent and mild in 22 per cent, and it typically appears within the first 5 days.

Mechanism: fasting exhausts glycogen, the metabolic rate falls by as much as 20 per cent, and intracellular phosphate, magnesium and potassium deplete while serum values look falsely normal; carbohydrate then drives insulin, which pulls them into cells.

Hypophosphataemia gives diaphragmatic weakness, respiratory failure and seizures, hypokalaemia gives arrhythmia and ileus, hypomagnesaemia gives arrhythmia and seizures, thiamine is consumed and precipitates Wernicke encephalopathy, and fluid retention gives pulmonary oedema.

High risk is little or no intake for more than 7 days to 10 days, weight loss above 10 to 15 per cent over 3 to 6 months, baseline electrolyte depletion, and a past episode.

Prevention: baseline biochemistry and an electrocardiogram, thiamine and a multivitamin before the first meal, cautious caloric initiation, fluid matched to output, and daily electrolytes for the initial 5 days.

THERAPY	STRUCTURE	THE DISCRIMINATOR
Family-based treatment	About 9 months, no more than 20 sessions of 50 to 60 minutes, the family meal in session 2 up to 90 minutes; phase I months 1 to 4 weekly, phase II every 2 to 3 weeks, phase III every 3 to 4 weeks from 95 per cent of median body mass index	**Parents take direct control of refeeding and the illness is externalised from the adolescent**
Its outcomes	Across six trials, full remission under 40 per cent and response near 75 per cent; in bulimia nervosa remission 39 against 18 per cent, and 29 against 10 per cent at 6 months	**Response is not remission, and quoting response as remission is the error**
Enhanced cognitive behaviour therapy	Above the 25th centile, 20 sessions of 50 minutes over 20 weeks; between the 3rd and 25th centile, 30 to 40 sessions over 30 to 40 weeks; parents attend a 1-hour assessment within 2 weeks then 15 to 20 minute joint sessions	**The patient is the agent of change, and it is preferred when family therapy is refused, contraindicated or ineffective**

TRAP Not carried by the sourced material here: the precise haemodynamic, temperature, body mass index and biochemical admission thresholds; the caloric starting range; the licensed high dose of fluoxetine in bulimia nervosa; the contraindicated antidepressant, which you should still be ready to name for its seizure risk; and the joint medical and psychiatric risk framework the examiner expects, MARSIPAN and its junior version. **Verify each before the exam.**

Intellectual disability with behavioural problems. The developmental period originates before age 22 years, which is the trap for candidates who quote 18.

Significant limitation is an IQ around 70 or as high as 75, with limitation across conceptual, social and practical adaptive skills.

Strata: mild IQ 52 to 69, moderate 36 to 51, severe 20 to 35, profound 19 or below.

Challenging behaviour occurs in 6.1 per cent of community adults and up to 40 per cent in long-stay institutions, with self-injury 4.9 per cent and aggression 9.8 per cent.

Those figures rise to **82 per cent and 45 per cent in profound disability with multiple impairments**.

- **Concept. Aetiology is biopsychosocial**: behavioural phenotypes such as Prader-Willi and Lesch-Nyhan syndromes, epilepsy and antiepileptic effects, and physical discomfort from constipation, dental abscess, reflux or urinary infection.
- **Concept.** The aetiology list closes on **comorbid mood and anxiety disorder, and communication barriers**.
- **Trap. Diagnostic overshadowing**, attributing the whole disturbance to the disability and missing treatable illness.
- **Key point.** Where communication is impaired, **read the rhythms of sleep, appetite and energy**, and expect obsessive phenomena as repetitive self-injury and depression as aggression.

Functional analysis has 5 steps: an unambiguous description of the behaviour; antecedents that predict it; consequences that maintain it; a hypothesis about its function, whether demand avoidance, tangible seeking or sensory stimulation; and direct observation to corroborate, recorded on an antecedent-behaviour-consequence chart.

Positive behavioural support is first-line. Across 109 articles, 52 per cent of interventions achieved at least a 90 per cent reduction and 68 per cent at least an 80 per cent reduction, with gains maintained 1 to 24 months in about two-thirds.

On drugs, be honest. A trial in 86 individuals found neither haloperidol nor risperidone superior to placebo at 4 weeks, while a trial in 77 found risperidone superior and maintained over 48 weeks. The mood stabiliser evidence disappeared once high-risk-of-bias studies were excluded.

CERTIFICATION, AND THE SCALE THAT DOES NOT APPLY

The Indian Disability Evaluation and Assessment Scale was developed in 2002 by the Rehabilitation Committee of the Indian Psychiatric Society to quantify disability from chronic mental illness, and its eligible diagnoses are strictly schizophrenia, bipolar disorder, dementia and obsessive compulsive disorder, with illness duration of at least 2 years.

It scores 4 items, self-care, interpersonal activities, communication and understanding, and work, and adds a chronicity score for months of illness in the last 2 years: 1 for under 6 months, 2 for 7 to 12 months, 3 for 13 to 18 months, 4 for more than 18 months.

The Global Disability Score is the item total plus that chronicity score, ranging 1 to 20. Bands: 1 to 7 is under 40 per cent and ineligible; 8 and above crosses the 40 per cent benchmark, with moderate 8 to 13, severe 14 to 19 and profound 20. Reassessment every 2 years.

The trap is saying this scale certifies intellectual disability. It does not: that is certified on standardised IQ and adaptive behaviour assessment by a board with a psychiatrist, a clinical psychologist and a paediatrician or physician.

TRAP Quoting a per-item maximum of 2 on that scale caps the global score at 12, contradicting its stated range of 1 to 20 and its profound band at 20, so the per-item maximum consistent with the arithmetic is 4. The published source text stops at the moderate anchor, so **check the severe and profound item anchors in a complete copy before the exam**, along with the guardianship provisions for an adult, including the National Trust Act and limited guardianship, and the conversion table from IQ to disability percentage, none of which is carried here.

The postpartum long case. Postpartum psychosis has an incidence of 0.89 to 2.6 in 1,000 births, with onset in the first 2 weeks in most cases and within 4 weeks in 90 per cent.

- **Key point. Exclude the organic causes first**: postpartum thyroiditis, Sheehan syndrome, sepsis, eclampsia, cerebral venous sinus thrombosis, autoimmune encephalitis, urea cycle disorders and deficiencies of thiamine, vitamin B12 or folate.
- **Concept. Admit to a mother and baby unit where one exists**, and otherwise room in with a family carer.

ITEM	FIGURE	THE DISCRIMINATOR
Risk	Infanticide 4 per cent, maternal suicide 5 per cent, recurrence 50 per cent	**Motives are altruistic delusion or command hallucination, and perinatal suicide uses violent methods, so no unsupervised infant contact while the episode is active**
Postpartum obsessive compulsive disorder	2.07 per cent in pregnancy, 2.43 per cent postpartum, cumulative 9.0 per cent by 6 months	**Harm thoughts are ego-dystonic and resisted with horror, insight is preserved, and there is no evidence of raised risk of infant harm**

ITEM	FIGURE	THE DISCRIMINATOR
Bipolar disorder	45 per cent risk of a postpartum episode, 20 per cent psychosis and 25 per cent depression; 74 per cent with a positive family history	**Sleep disruption in labour triggers the manic switch, so protect sleep and hand night feeds to the family**
The stepwise algorithm	Bedtime lorazepam 0.5 to 1 mg up to three times daily for 3 days remitted 6.3 per cent; adding an antipsychotic for 2 weeks a further 18.8 per cent; adding lithium a further 73.4 per cent, cumulative 98 per cent; electroconvulsive therapy at 12 weeks	**Lithium is what remits most patients, and with agitation, catatonia or imminent infanticide risk all three start together; acute target 0.8 to 1.0 mmol/L**
Electroconvulsive therapy	87 per cent remission against 73.5 per cent in matched non-postpartum controls	**No chemical excretion into milk, so it is compatible with breastfeeding**
Prescribing in lactation	Olanzapine 10 to 20 mg daily; quetiapine up to 400 mg; haloperidol under 10 mg daily; risperidone up to 6 mg; aripiprazole up to 15 mg	**Olanzapine is first-line; monitor the infant for sedation, poor feeding and weight gain, and note aripiprazole can suppress lactation**
Lithium in lactation and pregnancy	Infant and maternal levels on the 10th day after initiation, with infant renal function and thyroid-stimulating hormone; Ebstein anomaly 1 in 20,000 background against early estimates of 1 in 1,000; scans at 12 weeks and 20 weeks; monthly levels, weekly from 36 weeks	**Not absolutely contraindicated with structured monitoring; infant toxicity shows as hypotonia, lethargy, poor feeding and hypothermia**
Maintenance and prophylaxis	Lithium for 9 months postpartum; prophylaxis continued through pregnancy gives 23 per cent relapse against 66 per cent when stopped; taper over at least 4 weeks; restart within 24 hours of delivery	**Antipsychotic monotherapy without lithium relapses more often in the first postpartum year**

TRAP The onset window defined by the peripartum specifier is not carried here, and the figure of one year that circulates in this literature is a review's own inclusion window rather than a diagnostic criterion, which contradicts the observed onset data in the same source. The classification category and code used in Indian case sheets is unsourced here, as is the diagnostic cut-off on the postnatal depression screening scale. **Read all three off the manual and the classification and verify before the exam.**

HOLD THIS

Isolated hypophosphataemia after refeeding is refeeding syndrome, and it arrives within the first 5 days. The Indian disability scale covers four mental illnesses and does not certify intellectual disability. Postpartum psychosis begins within 2 weeks in most cases, carries 4 per cent infanticide and 5 per cent suicide risk, and lithium is what remits it.

WHAT EARNS THE MARKS

- **Isolated hypophosphataemia named as sufficient**: it is the diagnosis of refeeding syndrome on its own, inside the first 5 days.
- **Response separated from remission**: family-based treatment reaches response near 75 percent with full remission under 40 percent.
- **The developmental cut-off given as 22 years**: quoting 18 is the trap the examiner is fishing for.
- **Infant contact addressed explicitly**: no unsupervised contact while the episode is active, with the motives named.

SEE ALSO 06 Personal history 17 Why you examine 31 Mania and bipolar depression 35 Delirium and dementia

The whole long case in one table	
IDENTIFYING DATA	Ends on advance directive and nominated representative. Capacity, the directive and the representative sit in Sections 4, 5 and 14 of the Mental Healthcare Act 2017.
INFORMANT	Consistency, confirmability, credibility, constancy, adequacy. Name the observation window.
HISTORY OF PRESENTING ILLNESS	ABCDE per symptom; abrupt onset within 48 hours, acute within 2 weeks; relapse is the same episode before recovery, recurrence a new one after it.
NEGATIVE HISTORY	Organic, substance, competing polarity, risk. Anything unmentioned in the formulation is presumed absent.
GENOGRAM	Three generations, proband marked, offspring in birth order, consanguinity a double line, legend in the corner. First-cousin risk 3 to 5 percent against a 2 to 3 percent baseline.
PREMORBID	Temperament for childhood onset, personality for adult onset; six descriptive headings, each with an anecdote.
SUBSTANCE USE	Dependence is impaired control plus salience plus physiology. Organ damage alone is harmful use.
APPEARANCE AND SPEECH	Attitude is towards you, rapport forms between you. Speech in eight parameters; relevance and coherence are independent axes.
MOOD AND AFFECT	Climate against weather. Restricted narrows range, blunted lowers intensity, flat abolishes display. Inappropriate looks outward, incongruent looks inward.
THOUGHT	Circumstantiality reaches the goal, tangentiality does not; flight of ideas keeps understandable links, derailment loses them. Possession asks whose thought, content asks what it says.
PERCEPTION	Illusion has a stimulus, hallucination has none, delusional perception has a normal stimulus with abnormal meaning. Space separates true from pseudo.
COGNITION	Attention first or nothing counts. Cueing separates retrieval from storage failure. Isolated disorientation to person is functional, not focal.
INSIGHT AND JUDGEMENT	Insight graded 1 to 6 and three-dimensional; judgement intact or impaired, with ward behaviour overriding the hypothetical.
PHYSICAL EXAMINATION	Yours to do: mortality 2 to 4 times higher, 6 to 20 percent of physical disease misattributed. Glasgow Coma Scale 3 to 15 at admission; a 17-step screen in about 3 minutes.
RISK	Foreseeable, not predictable. Static, stable, dynamic, future; then the six formulation questions, then the plan.
FORMULATION	Summary condenses, formulation explains, in 10 to 20 sentences with no negative history. Perpetuating factors are the treatment targets.

The whole long case in one table	
CLASSIFICATION	ICD-11 dimensional for personality, categorical for dependence; DSM-5-TR the reverse. Gender incongruence sits outside the mental disorders chapter.
DIFFERENTIALS	Organic, substance, psychotic, affective, neurotic, personality. Bizarre delusions and thought disorder favour schizophrenia; suicidal ideation and detoxification history favour substance-induced psychosis.
INVESTIGATIONS	History 15.6 percent, examination 14.9 percent, routine screening 1.1 percent. Yield rises in the old, the agitated and the disoriented.
MANAGEMENT AND PROGNOSIS	Setting, first 24 hours, master plan on three axes, follow-up within 7 days. Prognosis on three axes, symptomatic and functional stated separately.

HOLD THIS

The long case rewards order, elicitation and explanation. Present in the proforma sequence, name the question that produced each phenomenon, and end with a formulation that explains why this person broke down now, followed by a risk statement and a prognosis on three axes.

Every discriminator in the case ladders, in one table	
SCHIZOPHRENIA	At least 1 month of symptoms; under 1 month is acute schizophrenia-like psychosis. Stratify the first drug by aggression: olanzapine then clozapine. Maintain a first episode 7 to 12 months. Resistance is 2 failed adequate trials.
ACUTE AND TRANSIENT PSYCHOSES	Onset within 2 weeks; remission ceiling 1 month for schizophrenia-like forms, 3 months for polymorphic and delusional ones. Transition to schizophrenia spectrum 36.14 per cent at 8 years.
SCHIZOAFFECTIVE AND DELUSIONAL DISORDER	Psychosis for at least 2 weeks free of mood is the dropped criterion; delusional disorder needs 1 month in one manual and 3 months in the other.
MANIA	Increased activity plus mood is the entry criterion; hypomania 4 days, mania 7 days, any psychosis makes it mania. Valproate's effect size is 0.16 against 0.37 to 0.56.
BIPOLAR DEPRESSION	Quetiapine leads, and is the only first-line monotherapy in bipolar II. Switch on an antidepressant 48.8 against 0.0 per cent. Lithium trough 0.8 to 1.2 mEq/L acute, 0.6 to 1.0 maintenance, drawn at 12 hours.
PSYCHOTIC AND RECURRENT DEPRESSION	Combination or electroconvulsive therapy, not monotherapy; response 86 against 42 per cent. Full dose for 4 to 9 months. Maintenance for 3 or more lifetime episodes.
OBSESSIVE COMPULSIVE DISORDER	2 to 3 times the depression dose, 8 to 12 weeks with 6 weeks high. Exposure therapy after a failed trial gives 56 per cent. Judge augmentation at 4 weeks, continue a success 1 year.

Every discriminator in the case ladders, in one table	
PANIC AND GENERALISED ANXIETY	Half the usual starting dose; review under 30 years within 1 week. Maintain 1 year and 6 months respectively. Benzodiazepines are not for panic disorder.
TRAUMA DISORDERS	Three tightened clusters, re-experiencing in the present, plus three self-organisation disturbances for the complex category. No proven superiority of therapy over a reuptake inhibitor; response about 55 per cent.
ALCOHOL AND OPIOID DEPENDENCE	3 of 6 criteria in a year; thiamine before glucose, at least 250 mg parenterally for 3 to 5 days; lorazepam in liver disease with doses halved. Induce buprenorphine on objective signs at 6 to 12 hours and titrate above 6 mg.
DELIRIUM AND DEMENTIA	Acute fluctuating onset plus inattention, plus disorganised thinking or altered consciousness. Haloperidol shortens intensive care stay only. Course sorts the dementia subtype; the 1-year rule sorts Lewy body disease; up to 50 per cent show neuroleptic sensitivity.
DISSOCIATION AND BODILY DISTRESS	Agency, not thought, is displaced in possession. Prolactin is an adjunct at 10 to 20 minutes, and an interictal spike is not epilepsy. Bodily distress disorder is rule-in at 3 months.
BORDERLINE PATTERN	Severity sets the intensity of therapy, diagnosis needs 2 years of the pattern at any age, and pharmacotherapy stays adjunctive.
EATING DISORDERS	Isolated hypophosphataemia after refeeding is the diagnosis, within the first 5 days. Parents refeed in family-based treatment; the patient leads in enhanced cognitive behaviour therapy.
INTELLECTUAL DISABILITY	Developmental period before age 22 years. Positive behavioural support first: 52 per cent of interventions achieved at least a 90 per cent reduction. The Indian scale does not certify intellectual disability; the benchmark is 40 per cent.
POSTPARTUM DISORDERS	Onset within 2 weeks in most cases and 4 weeks in 90 per cent; infanticide 4 per cent, suicide 5 per cent, recurrence 50 per cent. Lithium remits the majority, maintained 9 months.

HOLD THIS

Every case on this list is asked as the same ladder: summarise, diagnose, defend the diagnosis, decide the setting in law, prescribe, say for how long, say what you monitor, and say what you do when it fails. Where a number is not in your sources, say so and say where you will check it.

An examiner forgives a declared gap and remembers an invented figure.

PART III

The Psychiatry Short Case

Twenty minutes and one focused question: the short-case schema and the general-hospital cases it is built for.

01 The twenty-minute schema

02 Consultation-liaison cases

03 Substance and emergency cases

04 Neurodevelopmental cases

05 Quick review

The Psychiatry Short Case

Four sections · 19 topics · one complete notes document

The short case is not a compressed long case. It is a different instrument, and candidates lose marks by answering the wrong question. **Syndrome, risk, organicity, disposition.** That is the whole arc, and it is the arc an examiner listens for while you talk.

The pages that follow build the schema first, then walk the cases a general hospital actually sends: the catatonic patient on a medical ward, the febrile rigid patient, the patient refusing treatment, the patient in withdrawal, the patient whose movements came from the prescription, and the neurodevelopmental presentations that arrive with a parent and a form.

HOW TO USE THESE NOTES

Every topic runs in the same five beats, so you do not have to hunt for the part you want.

A **question band** gives the examiner's opening question and the station it belongs to. A **skeleton** numbers the sections of the answer in the order you say them, one cue line each.

Then the body in short blocks: concept lines, key-number lines, trap lines, the ladder of questions examiners work through, and the grids that separate the near neighbours with the one discriminator marked.

Then **what earns the marks**, which is the examiner's tick list rather than a summary of the topic. Then links to the other topics that feed this one.

Read a topic in three passes. First the band and the skeleton, which is the answer you could give with no notes at all. Then the key-number and trap lines. Then the grids and the held line. Where a figure is commonly quoted but could not be traced to a verified source, it appears as a trap to check rather than as a fact to recite.

The twenty-minute schema

1 · Short case against long case: what is being tested

TWENTY-MINUTE SCHEMA · TOPIC 01

This is a short case. How does your approach here differ from a long case?

SHORT CASE, OPENING QUESTION

THE SHAPE OF THE ANSWER

1 **Name the instrument**	Syndromic triage under time pressure, not a life history.
2 **Declare the pruning**	Say what you left out, and give the reason.
3 **Cross-sectional state**	Signs you elicited, positives first, then pertinent negatives.
4 **One-line formulation**	Fixed order, ending in the provisional syndrome.
5 **Disposition**	Risk, organicity, and what happens in the next hour.

The long case asks who this person is; the short case asks what is wrong now and what you will do. The examiner is watching for clinical reflex, efficiency of technique, and semiological precision under time pressure.

The commonest failure is a candidate who has prepared only long cases and recites a shortened one. **Prune deliberately, and prune out loud.**

- **Concept.** The mark sits in what you did at the bedside, not in how much you collected beforehand.
- **Concept.** Four beats carry the whole case: syndrome, risk, organicity, disposition.
- **Trap.** A shortened long case reads as unprepared, not as thorough.

1. How does your approach here differ from a long case?
2. Which parts of the history did you deliberately omit, and why?
3. Present your cross-sectional psychopathology.
4. Give me your one-line formulation.
5. What would make you think this is organic?
6. What is your disposition, and under what provision?

DIMENSION	LONG CASE	SHORT CASE
Objective	Complete biographical and developmental life chart	**Syndromic identification and risk triage**
Pedigree	Extended, traits across generations	First-degree relatives: illness, suicide, substance use
Development	Antenatal to adulthood milestones	Asked only if neurodevelopmental deficits emerge
Premorbid personality	Exhaustive social and moral inquiry	Functional baseline and coping pattern
Occupation	Full chronological job history	Current capacity and recent decline
Negatives	Systematic and complete	Pertinent only: category, organicity, risk
Mental state	Granular inventory of phenomena	**Focused evaluation of core functions**

Table: the pruning examiners expect, with the two cells that carry the mark.

TRAP Quoting a fixed number of minutes for either format, or a fixed presentation length, as a national standard. The figures in circulation could not be traced to a verified source. Verify your own institution's allocation before the exam and answer with the structure, not the clock.

RULE **Say what you compressed.** "No detailed developmental history: adult affective presentation, no suggestion of intellectual disability" scores. A silent gap does not.

HOLD THIS

Syndrome, risk, organicity, disposition. Actionable cross-sectional pathology beats historical narrative.

WHAT EARNS THE MARKS

- **Naming the instrument**: saying this is syndromic triage shows you know which question is being asked.
- **Pruning said out loud**: the omission plus its reason, in one clause, scores where silence does not.
- **Pertinent negatives only**: category, organicity, risk, and nothing further.
- **Structure instead of a clock**: asked how long you would take, answer with the arc.

SEE ALSO 02 The focused mental state examination 03 Organicity, safety, and the one-line formulation

2 · The focused mental state examination

TWENTY-MINUTE SCHEMA · TOPIC 02

Which core functions must every focused mental state examination account for?

SHORT CASE, BEDSIDE EXAMINATION

THE SHAPE OF THE ANSWER

1 What it is	A sign-gathering instrument, the psychiatric physical examination.
2 The seven functions	Name them, then the threefold structure of thought.
3 How you tested	Digit span, months backwards, registration, delayed recall.
4 The summary	Seven lines, positives first, then preserved functions.

The interview is a narrative method. **The mental state examination is an objective sign-gathering instrument, equivalent to the physical examination in general medicine.**

Historical surveys show its subdivision has ranged from 6 to 18 distinct items, but descriptive psychopathology settles on seven core functions, with thought evaluated across a threefold structure of stream, form, and content.

- **Concept.** A sign is what you elicited. A symptom is what was recounted to you.
- **Concept.** Thought splits three ways: stream, form, content. Stream and form are how it moves, content is what it carries.
- **Trap.** Presenting derailment, circumstantiality and delusional ideation as one undifferentiated list.

1. Which core functions must every focused examination account for?
2. How did you test attention and memory at the bedside?
3. Why is thought assessed in three dimensions?
4. Give me your psychopathological summary.

FUNCTION	BEDSIDE ELICITATION	PATHOLOGY SOUGHT
Consciousness	Alertness, responsiveness, environmental tracking	**Clouding, fluctuation, stupor, torpor**

FUNCTION	BEDSIDE ELICITATION	PATHOLOGY SOUGHT
Orientation	Time, place, person	Disorientation pointing to organicity or dissociation
Attention	Digit span, months backwards	Distractibility, impersistence
Perception	Observation plus direct query	Illusions, true against pseudohallucination, somatic passivity
Thought stream and form	Speech velocity, continuity, goal-direction	Flight of ideas, retardation, loosening, blocking
Thought content	Prevailing worries, overvalued ideas, delusional inquiry	Persecutory, referential, somatic, grandiose delusions
Memory	Registration, recent recall, remote landmarks	Amnesia, paramnesia, confabulation
Volition and psychomotricity	Spontaneous activity, drive, goal execution	Abulia, posturing, stereotypy, agitation

THE SUMMARY, IN SEVEN LINES

Appearance and behaviour; sensorium and cognition; affective life with subjective mood, objective affect, and congruence; thought across stream, form and content; perception; conation and volition; insight and judgement. Positive findings first, then the pertinent preserved functions. That order lets an examiner follow without interrupting.

TRAP The anamnesis fallacy. "Memory is impaired because he complains of forgetfulness" reports a symptom and calls it a sign. Give digit span, registration, and delayed reproduction instead.

TRAP Conflating thought dimensions. Derailment, circumstantiality, and neologism are stream and form; delusional ideation is content. Presenting them as one list invites the topic to be taken apart. Structured schedules such as the SCID, MINI, and SCAN rely on closed binary questioning, and that style flattens semiological depth at the bedside.

HOLD THIS

Seven functions, thought in three dimensions. Consciousness, perception, thought, memory, attention, orientation, volition.

WHAT EARNS THE MARKS

- **Signs with their elicitation**: digit span and delayed reproduction reported as findings, not as complaints.
- **Thought kept in three dimensions**: stream and form separated from content without being prompted.
- **An ordered summary**: seven lines, positives first, so the examiner does not interrupt.
- **Pertinent preserved functions**: naming what was intact shows the examination was systematic.

SEE ALSO 01 Short case against long case 03 Organicity, safety, and the one-line formulation 04 Catatonia: the signs, Bush Francis, the lorazepam challenge

3 · Organicity, safety, and the one-line formulation

TWENTY-MINUTE SCHEMA · TOPIC 03

What at the bedside makes you think this presentation has an organic driver?

SHORT CASE, PRESENTATION AND DISPOSITION

THE SHAPE OF THE ANSWER

1 **Organicity screen**	Consciousness, perception, pupils, focal signs, nocturnal worsening.
2 **Safety**	Intent, plan, lethality, command hallucinations, harm to others.
3 **Formulation**	Seven beats in one sentence, ending in the syndrome.
4 **Tiered differential**	Most likely, the organic possibility, one psychiatric alternative.

Two things are settled before you present: is this biologically driven, and is anyone in danger now. **A single documented alteration of consciousness turns a psychiatric short case into a medical one.**

- **Concept.** Organicity screen: fluctuating consciousness, visual or tactile hallucinations, sudden nocturnal worsening, pupillary abnormality, focal neurological deficit, disorientation in time or place.
- **Concept.** A definite alteration in the level of consciousness should raise strong suspicion of delirium or acute encephalopathy and prompt urgent organic workup rather than a psychiatric label.
- **Trap.** Presenting a rehearsed investigation panel as though it were a national standard.

1. What at the bedside suggests an organic driver?
2. What baseline investigations would you order?
3. Quantify the risk for me.
4. Give your formulation in one sentence.
5. Present a tiered differential.

Safety. Quantify intent, plan, and lethality for suicide and self-harm, and ask directly about command hallucinations and homicidal ideation.

Catatonic signs reorder the whole case. They move the patient into the pathway in topic 4 and take antipsychotics off the immediate table.

Formulation. One sentence, fixed order: age and sex, marital and occupational background, premorbid baseline, onset profile as acute, subacute or insidious, duration, the key psychopathological cluster, presence or absence of stressors and organic triggers, degree of socio-occupational decline and behavioural risk, then the provisional syndromic diagnosis.

MNEMONIC

ABCDEFG

Age and background, **B**aseline premorbid function, **C**ourse and onset, **D**uration, **E**vidence of the cluster, **F**actors present or absent, **G**rade of decline and risk, then the diagnosis. Seven beats, one sentence.

TRAP Reciting a first-line investigation panel as a standard. Examiners do ask for the baseline battery here, and the panel circulating in candidate notes could not be traced to a verified source, so it carries no cut-offs. Verify your unit's protocol before the exam and name it as local practice.

RULE **Tier the differential.** Most likely, then the organic possibility and how you excluded it, then the one psychiatric alternative you could be pressed on. Vagueness about drug class, target dose, titration speed and monitoring loses the management marks.

HOLD THIS

Consciousness first, risk second, formulation third. Sign, not symptom: what you elicited, not what was recounted.

WHAT EARNS THE MARKS

- **Consciousness addressed first**: the examiner is waiting to hear whether this is a medical case.
- **Risk quantified**: intent, plan and lethality as separate statements, plus harm to others.
- **One sentence that holds seven beats**: the formulation delivered without restarting.
- **A differential in tiers**: the organic possibility named with how you excluded it.

SEE ALSO 02 The focused mental state examination 04 Catatonia: the signs, Bush Francis, the lorazepam challenge 14 After deliberate self-harm: lethality, intent, safety planning

Consultation-liaison cases

4 · Catatonia: the signs, Bush Francis, the lorazepam challenge

CONSULTATION-LIAISON · TOPIC 04

This patient on a medical ward is mute and immobile. Define catatonia and give me the threshold.

SHORT CASE, MEDICAL WARD REFERRAL

THE SHAPE OF THE ANSWER

1 Define and count	The picture is dominated by a minimum number of listed features.
2 Elicit the signs	The volitional signs need your hands, not observation.
3 Rate it	Screening instrument against the full rating scale.
4 Challenge and treat	Lorazepam, reassess by route, then scheduled dosing.
5 Cause and cover	Medical workup, plus the complications of prolonged stupor.

On a general hospital ward the cause is at least as often medical as psychiatric. It is under-recognised because the volitional signs must be physically elicited.

- **Key number.** Incidence is approximately 10 per 100,000 person-years, and it affects roughly 1 in 10 mental health inpatients.
- **Key number.** DSM-5-TR requires the picture to be dominated by at least 3 of 12 features: stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerism, stereotypy, agitation, grimacing, echolalia, echopraxia.
- **Trap.** Scoring from observation alone, which leaves the volitional signs unexamined.

1. Define it and give the threshold.
2. Elicit the signs at the bedside.
3. Screening instrument against full scale.
4. Conduct and interpret a lorazepam challenge.
5. Why lorazepam, and why are antipsychotics risky?
6. Which medical causes will you investigate?

INSTRUMENT	ITEMS	THRESHOLD	OPERATING CHARACTERISTICS
Screening instrument	First 14 items	**At least 2 signs**	Sensitivity 100 per cent, specificity 96.2 per cent, area under the curve 0.98
Full rating scale	All 23 items, scored 0 to 3	**At least 2 signs**	Sensitivity 100 per cent, specificity 90.7 per cent, area under the curve 0.95

Table: the threshold is identical; what changes is what the longer scale can see.

In an Indian tertiary hospital study of 300 patients assessed within 24 hours of admission, **80 patients (26.7 per cent) showed at least one sign.**

The full 23-item scale found an additional 4 per cent of cases over the 14-item screen, raising detected prevalence from 16.3 per cent to 20.3 per cent.

Factor analysis gives 3 dimensions accounting for 65.48 per cent of variance, retarded, excited and aberrant volitional, and 55.7 per cent of patients show signs from more than one dimension.

RULE **The volitional signs live at the back of the scale.** Apart from waxy flexibility, item 13, they sit in supplementary items 15 to 23, which is why a passive screen misses them. Mitgehen: the limb rises to light fingertip pressure despite instruction to resist. Gegenhalten: resistance scaling with the force applied. Ambitendency: stuck in conflicting half-movements. Each is scored present or absent.

Fig 3.1 The lorazepam challenge as a two-branch ladder: baseline rating and vital signs, lorazepam by the chosen route, reassessment at the route-specific interval, then either confirmation with scheduled dosing or a repeat dose and formal escalation.

The challenge. Record the score and vital signs, then lorazepam 1 to 2 mg intravenously, intramuscularly or orally. Reassess at 5 minutes intravenous, 15 minutes intramuscular, 30 minutes oral.

- **Key number.** Positive is a fall of at least 50 per cent in signs, seen in approximately 8 in 10 patients.
- **Key number.** With no sedation and an equivocal response, repeat the 1 to 2 mg dose, then titrate scheduled dosing over several days to at least 16 mg per 24 hours in divided doses before declaring failure.

- **Trap.** Stopping at anxiolytic doses of 2 to 4 mg per 24 hours is the standard error. Taper gradually, since abrupt cessation causes recurrence.

RULE **Lorazepam is a GABA receptor agonist, and its reliability here, alongside catatonia in anti-NMDA receptor encephalitis, implicates GABAergic and glutamatergic dysregulation.** The mirror is the hazard: dopamine receptor blocking agents can precipitate neuroleptic malignant syndrome, so antipsychotics are avoided without a psychotic disorder and used cautiously with one. Where benzodiazepines fail and electroconvulsive therapy is unavailable, amantadine and memantine carry the strongest remaining evidence; if abrupt clozapine withdrawal was the trigger, reinstate clozapine.

Workup. Imaging for a first episode; anti-NMDA receptor and other neuronal surface antibodies in serum and cerebrospinal fluid, remembering a normal video electroencephalogram does not exclude that encephalitis; electroencephalography for nonconvulsive status.

Then counts, metabolic, renal and liver panels, inflammatory markers, lupus serology, thyroid profile, HIV testing; urine drug screen; slit-lamp for Kayser-Fleischer rings.

- **Concept.** The complications of stupor are their own answer: thromboprophylaxis for pulmonary embolism, fluids and early nasogastric feeding, pressure-area care.
- **Concept.** Stay alert to rhabdomyolysis and aspiration, which are what kill an immobile patient.

INDIAN PRACTICE NOTE

Electroconvulsive therapy is indicated when benzodiazepine trials fail, in malignant catatonia, or where medical deterioration demands speed: bilateral placement, a minimum of 2 times weekly, titrated to response. Unmodified treatment is prohibited under the Mental Healthcare Act 2017, every course runs under general anaesthesia with muscle relaxation, and treatment in a minor needs the nominated representative's consent and prior Mental Health Review Board permission. Admission of a minor is governed by Section 87.

TRAP Quoting section numbers for the electroconvulsive therapy provisions, the emergency treatment provision or its maximum duration, or the total score ranges of either Bush Francis instrument. None could be traced in this material. State the substantive rule and verify the numbers against the bare Act and the scale manual before the exam.

HOLD THIS

Two signs on either instrument is the threshold; items 15 to 23 hold the volitional signs. A fall of 50 per cent after 1 to 2 mg of lorazepam confirms the diagnosis and starts the treatment.

WHAT EARNS THE MARKS

- **The same threshold on both instruments**: saying so shows you know the screen and the full scale.
- **Volitional signs elicited**: mitgehen, gegenhalten and ambitendency demonstrated rather than listed.
- **A challenge carried to a real dose**: titration before failure is declared, not an anxiolytic dose abandoned.
- **Antipsychotics held back**: the malignant syndrome hazard named before the examiner raises it.

SEE ALSO 03 Organicity, safety, and the one-line formulation

05 Rigid and febrile: NMS, serotonin syndrome, malignant catatonia 17 Autism: DSM-5, the ISAA, and disability certification

5 · Rigid and febrile: NMS, serotonin syndrome, malignant catatonia

CONSULTATION-LIAISON · TOPIC 05

This patient on an antipsychotic is rigid and febrile. Work through the differential for me.

SHORT CASE, EMERGENCY AND CL

THE SHAPE OF THE ANSWER

1 **The tetrad, in order**	Mental state, rigidity, autonomic dysregulation, fever.
2 **Separate the three**	Tone, reflexes, pupils, bowel sounds, drug history.
3 **Criteria and enzymes**	Which criteria sets, and what creatine kinase can prove.
4 **The first hour**	Stop the drug, fluids, cooling, monitored bed, lorazepam.
5 **Rechallenge**	Defer, admit, and move to a low D2 affinity agent.

The drug history, the tone, the pupils, the bowel sounds and the reflexes separate them.

Neuroleptic malignant syndrome is **a diagnosis of exclusion**, so malignant hyperthermia, heat stroke, pheochromocytoma, thyrotoxicosis, tetanus, parkinsonian hyperthermia after abrupt dopamine agonist withdrawal, sedative withdrawal and anticholinergic poisoning belong in the same breath.

- **Key number.** Core temperature must exceed 38.0 degrees Celsius (100.4 degrees Fahrenheit) on at least 2 occasions orally.
- **Key number.** Hyperthermia appears in 88 per cent of cases and rigidity in 86 per cent.

1. Give the tetrad and its order.
2. Separate the three at the bedside.
3. Which criteria sets, and what are their limits?
4. What does creatine kinase tell you?
5. Drugs and doses.
6. When is electroconvulsive therapy first-line?
7. How will you rechallenge?

RULE **Mental state, then rigidity, then autonomic dysregulation, then fever.** In 96 per cent of cases onset falls within the first 4 weeks of starting or escalating a dopamine antagonist, though it can emerge within 24 to 72 hours or after prolonged stable treatment. Blood pressure lability is a diastolic shift of at least 20 mmHg or systolic shift of at least 25 mmHg within a 24-hour period, and hypermetabolism a heart rate rise of at least 25 per cent with a respiratory rate rise of at least 50 per cent above baseline.

FEATURE	NEUROLEPTIC MALIGNANT SYNDROME	SEROTONIN SYNDROME	MALIGNANT CATATONIA
Trigger	D2 antagonists, antiemetics, dopamine agonist withdrawal	SSRIs, SNRIs, MAOIs, tricyclics, lithium, tramadol, triptans	Mood disorder or schizophrenia; no neuroleptic needed

FEATURE	NEUROLEPTIC MALIGNANT SYNDROME	SEROTONIN SYNDROME	MALIGNANT CATATONIA
Tempo	Days to weeks; 96 per cent within 4 weeks	Hours to 24 hours; within 24 to 72 hours	Subacute, after excited catatonia
Tone	**Hypokinetic lead-pipe rigidity, bradykinesia**	**Hyperkinetic: spontaneous, inducible or ocular clonus, tremor**	Posturing, waxy flexibility, catalepsy, negativism
Reflexes and pupils	Hyporeflexia; normal pupils with staring	**Hyperreflexia worse in the legs; mydriasis**	Normal or brisk; fixed unblinking gaze
Bowel sounds	Normal or hypoactive, no diarrhoea	**Hyperactive, with cramping and diarrhoea**	Normal or decreased
Creatine kinase	Above 1000 U/L or at least 4 times the upper limit of normal	Mild or normal unless prolonged seizures	Mild to moderate, from posturing
Resolution and mortality	7 to 10 days after stopping, alternatively given as 9 to 14 days, longer with depot; 5.6 per cent to 10 per cent, up to 50 per cent with renal failure	24 to 72 hours after cessation; fatal cases rare	Rapid with benzodiazepines or electroconvulsive therapy; 10 per cent estimated mortality

Table: tone, pupils and bowel sounds carry the discrimination; the drug history sets the prior.

Criteria. The two used sets are DSM-5 and Levenson. The secure Levenson threshold is creatine kinase above 1000 U/L; the Gurrera consensus criteria use at least 4 times the upper limit and state explicitly that no threshold score has been defined and validated.

Serotonin syndrome: Sternbach needs a new or increased serotonergic agent plus at least 3 of 10 features; the Hunter criteria are superior in accuracy and specificity, satisfied by any one of spontaneous clonus, inducible or ocular clonus with agitation or diaphoresis, tremor with hyperreflexia, or hypertonia with temperature above 38.0 degrees Celsius plus clonus.

- **Key number.** Creatine kinase is raised in up to 70 per cent of febrile patients on antipsychotics who do not have the syndrome, and up to 30 per cent of febrile patients on no psychotropic.
- **Concept.** So the enzyme supports the diagnosis and grades the muscle injury. It does not establish the diagnosis on its own.

AGENT	DOSE	THE LINE TO SAY
Lorazepam	1 to 2 mg orally or intravenously every 4 to 6 hours	**First-line at every severity, but not by the intramuscular route, which corrupts creatine kinase**
Bromocriptine	2.5 mg 2 to 3 times daily, titrated by 2.5 mg every 24 hours to a maximum of 45 mg per 24 hours	**Overcomes central D2 blockade; continue 10 days after control, 2 to 3 weeks after a depot**
Dantrolene	1 to 2.5 mg/kg intravenous bolus, then 1 mg/kg every 6 hours to a maximum of 10 mg/kg per 24 hours; oral 50 to 200 mg daily for 10 days	**Response within minutes; hepatotoxicity governs duration**

The first hour. Stop every antipsychotic and dopamine-blocking antiemetic; dialysis does not clear them.

Wide-bore access, aggressive saline to protect renal tubules against myoglobin, active surface cooling, no physical restraint, and transfer to a monitored bed with heparin prophylaxis and correction of acidosis and electrolytes.

- **Key number.** Recurrence risk on re-exposure is 30 per cent, so defer rechallenge at least 2 weeks after complete recovery, as an inpatient, switching to a low D2 affinity agent.
- **Key number.** Clozapine is safest, with favourable outcomes in 79 per cent of rechallenges after non-clozapine cases and 92 per cent where the index episode was on clozapine, and no reported mortality; quetiapine is the secondary option.

RULE **Electroconvulsive therapy is second-line after 48 hours of failed intensive treatment, and first-line in three situations:** ambiguity between neuroleptic malignant syndrome and malignant catatonia, an underlying psychotic depression, or catatonic features dominating the picture.

TRAP Reciting the itemised Levenson major and minor criteria with their counts, or the DSM-5 list with the number of associated features required, or a cyproheptadine dose. None could be traced here. Assuming atypicals cannot do this is the clinical trap: they can, often with less rigidity and lower creatine kinase, which delays diagnosis.

HOLD THIS

Hypokinetic with hyporeflexia is neuroleptic malignant syndrome; clonus, mydriasis and loud bowels is serotonin syndrome; rigid and febrile in a neuroleptic-naive patient after excited catatonia is malignant catatonia, and that one needs electroconvulsive therapy now.

WHAT EARNS THE MARKS

- **The tetrad in sequence**: mental state change precedes fever, and candidates invert it.
- **Three bedside discriminators as a set**: tone, reflexes and bowel sounds, said together.
- **Creatine kinase placed in context**: it supports and grades, and does not establish the diagnosis.
- **Electroconvulsive therapy positioned**: second-line generally, first-line in the three named situations.

SEE ALSO 04 Catatonia: the signs, Bush Francis, the lorazepam challenge

08 The medical mimics: steroid, thyroid, adrenal, porphyria, autoimmune

11 Two iatrogenic cases: tardive syndromes and lithium toxicity

6 · Capacity and the supported-admission ladder

CONSULTATION-LIAISON · TOPIC 06

This patient refuses admission and you believe he lacks capacity. Where is capacity defined, and what follows?

SHORT CASE, VIVA ON THE ACT

THE SHAPE OF THE ANSWER

1 The presumption	Capacity is presumed; the standard is the balance of probabilities.
2 Three limbs	Understand, appreciate, communicate, applied together.
3 Pick the tier	Recent behaviour against behaviour consistent over time.
4 Certifiers and days	Count the certifiers, then give the permitted duration.
5 Safeguards	Board notice, capacity review, advance directive, discharge planning.

Capacity is decision-specific and time-specific, and Indian law presumes you have it.

Under Section 4(1) of the Mental Healthcare Act 2017, every person, including a person with mental illness, is deemed to have capacity to decide about their mental healthcare or treatment unless proved otherwise.

- **Concept.** A patient may lack capacity for a complex treatment decision while retaining it for food or clothing, and regain it on remission.
- **Concept.** The standard of proof is the balance of probabilities, so say which way the balance falls and why.
- **Trap.** Treating a diagnosis, or a decision you disagree with, as incapacity.

1. Where is capacity defined, and what is the presumption?
2. Give the functional abilities under Section 4(1).
3. How does that differ from the United Kingdom test?
4. He lacks capacity and needs admission. Which provision?
5. He is still unwell at the end of that period.
6. The family want the tablets hidden in his food.

- **Concept.** Understand, 4(1)(a): the condition and the nature, purpose, risks and benefits of what is proposed, probed by asking the patient to say what they understand about the illness and why the treatment is suggested.
- **Concept.** Appreciate, 4(1)(b): applying it to oneself and foreseeing the reasonably foreseeable consequences of deciding or not deciding, probed by asking what will happen if they take it and if they refuse.
- **Concept.** Communicate, 4(1)(c): a stable choice by speech, writing, signing or gesture.

Section 4(2) requires the information in simple language the person understands, or in sign language, visual aids or any other means enabling understanding.

THE DRAFTING POINT EXAMINERS REWARD

The enacted text joins sub-clauses (a), (b) and (c) with "or" rather than "and", and commentary treats that as a drafting error, because a disjunctive reading would let a single retained ability establish capacity. Give the statutory wording, then say the limbs are applied together. The United Kingdom Mental Capacity Act 2005 applies from age 16 years with a two-stage test and four abilities, understand, retain, use or weigh, communicate; Indian law needs no separate diagnostic hurdle, and retaining is subsumed within understanding and appreciating long enough to choose.

STEP	INDEPENDENT, SECTION 86	SUPPORTED, SECTION 89	EXTENDED, SECTION 90
Capacity	**Decides without or with minimal support; treatment on informed consent**	**Lacks capacity, very high support from the nominated representative**	**Continues to lack capacity**
Severity criterion	Not applicable	Recent threats or attempts of harm to self, violence or causing fear of harm to others, or severe inability for self-care	The same behaviours, occurring consistently over time
Certifiers	The person's written consent	One psychiatrist plus one mental health professional or medical practitioner, examining on admission or within the preceding 7 days	2 psychiatrists examining independently within the preceding 7 days
Setting and duration	Hospital or community based; open, discharge on request	Hospital based only; limited to 30 days	Hospital based only; up to ninety days first, then one hundred and twenty days, thereafter one hundred and eighty days each time
Board notice and approval	Not applicable	Within 3 days for a woman or minor, within 7 days for any other person; on a requested review the Board gives binding findings within 7 days	Within 7 days of admission or readmission; the Board permits or orders discharge within 21 days of the last admission
Capacity review	No routine check unless capacity appears to be failing	Every 7 days, with the representative's recorded consent	Every fortnight

Table: capacity decides the route, the severity wording decides the tier, and the certifier count is the fastest discriminator between the supported tiers.

THREE PROVISIONS WORTH NAMING UNPROMPTED

An independent patient requesting discharge is discharged immediately under Section 88(1), with one exception: a mental health professional may prevent discharge for a period of twenty-four hours, and only to allow the assessment needed for a supported admission under Section 89. Readmission within seven days of a Section 89 discharge also falls under Section 90. Discharge planning under Section 98 is mandatory for every discharge, consulting the person, the nominated representative, the resident family member or care-giver, and the incoming psychiatrist.

TRAP Treating an unwise decision as incapacity, or a diagnosis as incapacity. Section 4(3) says a decision others perceive as inappropriate or wrong does not by itself mean the person lacks capacity, and Section 3(5) says a determination of mental illness does not by itself mean the person is of unsound mind unless a competent court has so declared. Cite the Indian provisions, not the United Kingdom principle. Offering proverb interpretation or the letter-and-fire judgement items as a capacity assessment is the third version: those probe abstract reasoning, not the statutory limbs.

Covert medication. Permitted only for an inpatient who lacks capacity, not for anyone who has it, with written informed consent from the nominated representative, restricted to essential medication to restore health or prevent severe harm.

It is not permissible for staff convenience, ward management or research. Check the advance directive; an application to review, alter, modify or cancel one goes to the Board under Section 11(1).

- **Concept.** Verify that crushing does not alter bioavailability, review at least weekly, and stop immediately if capacity returns.
- **Key number.** Scheduled rapid tranquillisation is reviewed every 24 hours, and each depot injection in a non-consenting incapacitated inpatient needs a capacity assessment within the preceding 7 days.
- **Concept.** Document the specific decision, the information given, the three limbs with the patient's own quotes, your opinion on the balance of probabilities, and the date of reassessment.

HOLD THIS

Understand, appreciate, communicate, presumed until proved otherwise. Recently equals Section 89 and one psychiatrist plus one professional; consistently over time equals Section 90 and 2 psychiatrists.

WHAT EARNS THE MARKS

- **The statutory wording**: the three limbs given with their sub-clause letters, then applied together.
- **The severity wording**: recent behaviour against behaviour consistent over time is what selects the tier.
- **The certifier count**: the quickest way to show you have the two supported tiers straight.
- **Indian provisions cited**: an unwise decision separated from incapacity on the Indian text, not the United Kingdom principle.

SEE ALSO 03 Organicity, safety, and the one-line formulation

14 After deliberate self-harm: lethality, intent, safety planning 19 Eating disorders and the SCOFF

7 · Epilepsy and psychosis, including forced normalisation

CONSULTATION-LIAISON · TOPIC 07

This patient with epilepsy has become psychotic. Classify the psychoses of epilepsy for me.

SHORT CASE, NEUROPSYCHIATRY

THE SHAPE OF THE ANSWER

1 **Classify by timing**	Ictal, postictal, interictal, and alternative psychosis.
2 **The lucid interval**	Full alertness between the seizures and the psychosis.
3 **Against schizophrenia**	Later onset, warm affect, few negative symptoms.
4 **Forced normalisation**	Reciprocal with seizure control; give the criteria.
5 **Manage each**	Short antipsychotic course, or let the seizures back.

Classify by timing relative to the seizure, and management follows.

- **Concept.** Ictal psychosis occurs during the discharge, characteristically in nonconvulsive status epilepticus.
- **Concept.** Postictal psychosis follows a lucid interval. Interictal psychosis runs between seizures as a chronic illness.
- **Concept.** Alternative psychosis, or forced normalisation, appears inversely with seizure suppression.
- **Key number.** Prevalence in epilepsy is 6.0 per cent against 0.7 per cent in the general population, with incidence 2.5 times higher; in temporal lobe epilepsy it is 7.0 per cent.

1. Classify the psychoses of epilepsy.
2. Ictal psychosis against nonconvulsive status epilepticus.
3. Describe the lucid interval.
4. Separate interictal psychosis from schizophrenia.
5. Give the Krishnamoorthy and Trimble criteria.
6. Manage each, and name the antipsychotic you avoid.

CATEGORY	TIMING	LATENCY FROM ONSET	DURATION	DOMINANT FEATURES
Ictal	During the discharge	Variable	Minutes to hours	**Confusion, automatisms, bizarre behaviour, amnesia**
Postictal	**After a lucid interval of 8 to 72 hours**	10 to 20 years	Days to weeks, rarely beyond 2 weeks	Pleomorphic, religious and grandiose delusions, affective flux
Interictal	Independent of seizure timing	10 to 15 years	Months to years	**Paranoid delusions with preserved affect and no negative deficit**
Forced normalisation	**Reciprocal with seizure control**	Longstanding, mean 15.2 years	Days to weeks	Paranoid, mood symptoms, twilight states

The lucid interval. Postictal psychosis follows a cluster of focal to bilateral tonic-clonic or complex partial seizures, with bilateral independent foci and past status epilepticus as predisposing factors.

During the lucid interval the sensorium clears completely, and the first warning sign is frequently severe insomnia.

Interictal psychosis, the schizophrenia-like psychosis of epilepsy described by Slater and Beard, carries risk from temporal lobe epilepsy especially left-sided and bitemporal foci, early onset and hippocampal sclerosis.

- **Concept.** It separates from nuclear schizophrenia by later onset and preserved affective warmth with little personality deterioration.
- **Concept.** Positive symptoms predominate, command hallucinations are rare, and negative symptoms are absent or mild.

- **RULE** **Postictal delirium has no lucid interval; postictal psychosis is defined by one.** Delirium follows the seizure immediately with clouded consciousness and autonomic fluctuation; psychosis emerges only after 8 to 72 hours of full alertness. Forced normalisation is the electroencephalographic diagnosis, alternative psychosis its clinical counterpart without a recording.

FORCED NORMALISATION: THE NAMES AND THE CRITERIA

In 1953 Heinrich Landolt described patients whose surface recordings normalised during psychotic episodes and coined forced normalisation; in 1965 Tellenbach introduced alternative psychosis; Wolf termed the process paradoxical normalisation. Trimble proposed dopaminergic kindling, Rayport, Ferguson and Wieser neocortical voltage suppression with running deep discharges, and kappa opioid overactivation is a third proposal. Krishnamoorthy and Trimble require criterion 1, established epilepsy on history, electroencephalography and imaging; criterion 2, acute or subacute behavioural disturbance with psychosis, significant mood change, anxiety with depersonalisation, or hysteria; and either criterion 3A, over 50 per cent reduction in total spike count on a 60-minute waking recording with a 16-channel machine using standard 10-20 placement against a behavioural baseline, or criterion 3B, complete cessation of seizures for at least 1 week corroborated by a carer, plus one supportive criterion such as a regimen change within 30 days.

The drugs. This occurs across essentially every antiepileptic class and tracks the rapidity of seizure suppression rather than one toxic property; resective surgery and vagus nerve stimulation have both induced it, and slow titration reduces risk.

- **Key number.** In 1,412 patients on levetiracetam for more than 1 month, 111 (7.8 per cent) developed severe psychiatric symptoms, 38 per cent of those within 1 to 4 weeks and most within the first 3 months, and 64 per cent had a pre-existing psychiatric history.
- **Key number.** Discontinuation alone gave complete recovery in 57 of 57 patients within 1 to 3 weeks, and dose reduction alone improved 5 of 11 within 2 to 3 weeks.
- **Key number.** Where seizure recurrence is unacceptable, low-dose haloperidol or risperidone produced remission within 1 to 2 weeks.

Postictal psychosis is self-limiting, and an early atypical antipsychotic at the first sign of postictal insomnia can abort a decompensation.

TRAP Quoting electrographic criteria for nonconvulsive status epilepticus, or a starting dose for postictal risperidone. Neither could be traced here; the sources say only that a discharge pattern time-locked to the clinical state is required. Verify before the exam. The clinical trap is clozapine, uniquely epileptogenic among the atypicals and generally avoided in active epilepsy.

HOLD THIS

Too many seizures gives postictal psychosis; too few gives forced normalisation. One takes a short antipsychotic course, the other takes letting the seizures back.

WHAT EARNS THE MARKS

- **Timing as the axis**: the classification is chronological, and management falls straight out of it.
- **The lucid interval named**: it is what separates postictal psychosis from postictal delirium.
- **Insomnia treated as the warning**: an early atypical at that point can abort the episode.
- **Clozapine avoided unprompted**: the epileptogenic atypical, named before the examiner asks.

SEE ALSO 04 Catatonia: the signs, Bush Francis, the lorazepam challenge

08 The medical mimics: steroid, thyroid, adrenal, porphyria, autoimmune

11 Two iatrogenic cases: tardive syndromes and lithium toxicity

8 · The medical mimics: steroid, thyroid, adrenal, porphyria, autoimmune

CONSULTATION-LIAISON · TOPIC 08

This patient with lupus on high-dose steroids is psychotic. Is this the drug or the disease?

SHORT CASE, MEDICAL WARD REFERRAL

THE SHAPE OF THE ANSWER

1 **Which way it moves**	More steroid improves the lupus and worsens the drug effect.
2 **Dose and latency**	Risk rises above a threshold dose; onset within days.
3 **Steroid must continue**	Taper below the threshold, then treat, then monitor.
4 **Thyroid and adrenal**	Match each psychiatric picture to its physical clue.
5 **The rarer mimics**	Porphyria, anti-NMDA encephalitis, neurosyphilis, carcinoid.

Steroid psychosis against lupus cerebritis turns on what the rest of the body is doing.

- **Key number.** Reported incidence of corticosteroid-induced psychosis ranges between 1.8 per cent and 62 per cent, and on prolonged high-dose therapy 1 in 2 to 3 patients develops symptoms: mania or hypomania 35 per cent, depression 28 per cent, frank psychosis 24 per cent.
- **Key number.** Risk rises sharply above 40 mg daily of oral prednisone or equivalent, and particularly at 80 mg daily or greater, equivalent to 12 mg oral dexamethasone.
- **Concept.** Dose does not predict symptom type, severity or duration, so the size of the dose sets the risk and not the picture.

1. Separate steroid psychiatry from neuropsychiatric lupus.
2. Give the dose threshold and onset latency.
3. The steroid cannot be stopped. What now?
4. Which pictures does thyroid disease give?
5. Acute intermittent porphyria: confirmation and safe drugs.
6. What else must be excluded in atypical psychosis or catatonia?

PARAMETER	CORTICOSTEROID-INDUCED	LUPUS CEREBRITIS
Onset	Within 3 to 4 days, or the first 5 days, of starting or escalating	With a systemic flare, independent of dose change
Systemic activity	**Renal function, joints and serologies stabilising**	**Active: fever, rash, arthritis, nephritis, cytopenias**
Serology and fluid	Complement reflects control; fluid unremarkable	Positive anti-double-stranded DNA; antinuclear antibody titre 1:80 or greater; raised protein or pleocytosis
More steroid	**Worsens or fails to remit**	**Improves**
Taper	92 per cent achieve full resolution	Flare and worsening deficits without immunosuppression

Table: what the systemic disease is doing, and which way the patient moves when the dose changes.

Where the steroid must continue, **taper to below 40 mg daily of prednisone**, dexamethasone equivalent 6 mg orally, then treat: risperidone 1 to 4 mg daily, olanzapine 2.5 to 20 mg daily with maintenance often 10 to 15 mg after starting at 2.5 mg, or quetiapine 25 mg daily with 12.5 mg as needed.

Monitor weight monthly for the first 3 months then quarterly, with blood pressure and fasting glucose at 3 months and annually.

- **Key number.** Lithium reduced corticotropin-induced symptoms from 62 per cent in controls to 38 per cent at levels of 0.8 to 1.2 mEq/L, with no patient developing frank psychosis.
- **Key number.** Fluoxetine 10 to 40 mg daily showed response within 7 days for depressive presentations.

TRAP Relying on psychiatric history to predict this, and reaching for a tricyclic. Healthy individuals with no prior illness develop acute steroid psychosis, and the sources disagree openly on whether past illness predicts recurrence, so say the disagreement out loud; female sex and older age are the factors both support. Tricyclics exacerbate steroid-induced symptoms, so sertraline or citalopram are preferred for minimal cytochrome interaction.

STATE	PSYCHIATRIC PICTURE	THE PHYSICAL CLUE
Thyrotoxicosis	Anxiety, irritability, lability, insomnia, restlessness	**Tremor, tachycardia, sweating, heat intolerance, weight loss, diarrhoea, goitre, eye changes**
Hypothyroidism	Depression, tearfulness, lethargy, memory lapses, concentration deficit	**Cold intolerance, weight gain, constipation, dry skin, hair loss, menstrual irregularity**
Cushing syndrome	Anxiety, euphoria, depression, mania, insomnia, psychosis	**Central obesity, hypertension, hyperglycaemia, diaphoresis**
Addison disease	Fatigue, reduced appetite, weight loss, apathy resembling neurovegetative depression	**Inadequate cortisol, with weight loss rather than gain**

Beta-blockers ease adrenergic tremor in thyrotoxicosis but can themselves cause fatigue or low mood, and in hypothyroidism mood recovery lags behind laboratory normalisation.

In a UK Biobank cohort of 349,993 participants followed over 1 to 13 years, baseline anxiety and depression predicted subsequent thyroid disorder, incidence 2.82 per cent, hazard ratios 1.23 to 2.01, and men with severe symptoms had a threefold higher hazard of hyperthyroidism, hazard ratio 3.02, 95 per cent confidence interval 1.64 to 5.54. **The arrow runs both ways.**

SAFE IN ACUTE INTERMITTENT PORPHYRIA	PORPHYRINOGENIC, TO BE AVOIDED
Amitriptyline, fluoxetine, lithium, lorazepam	**Chlordiazepoxide, clonazepam**
Chlorpromazine, fluphenazine, haloperidol	**Quetiapine, ziprasidone**
Gabapentin, diphenhydramine	Venlafaxine, phenelzine, carbamazepine, topiramate, trazodone, ramelteon

Table: the marked cells are the ones candidates reverse, because two ordinary benzodiazepines and two ordinary atypicals sit on the unsafe side.

Porphyria. Porphobilinogen deaminase deficiency gives severe colicky abdominal pain without peritoneal signs, neuropsychiatric symptoms, and neurological signs including motor neuropathy, seizures and quadriparesis, triggered by cytochrome inducers, fasting and hormonal cycling.

Confirm on urinary porphobilinogen greater than 6 mg/L during an attack, and treat with intravenous haem and carbohydrate loading.

Anti-NMDA receptor encephalitis. Young women, often with ovarian teratoma, a viral prodrome to agitation, catatonia and psychosis, then autonomic instability, dyskinesias and seizures, confirmed on cerebrospinal fluid because serum is less sensitive.

Neurosyphilis. A chancre 10 to 90 days after exposure, a secondary rash on palms and soles in 50 per cent, tertiary general paresis in 30 per cent of untreated individuals, screened on a titre greater than 1:4 and treated with intravenous crystalline penicillin G for 10 to 14 days.

Carcinoid. It diverts tryptophan to serotonin and produces pellagra, and resection of a neuroendocrine tumour often resolves the psychiatric symptoms within 8 weeks.

TRAP Misreading the Jarisch-Herxheimer reaction as penicillin allergy: fever, rigors and tachycardia within 24 hours of starting penicillin for syphilis is self-limiting. The statutory trap is the emergency treatment provision and its duration, untraced here. The secure position: emergency psychiatric treatment runs under the Mental Healthcare Act 2017, while the concurrent physical emergency such as an adrenal crisis runs under ordinary medical consent and necessity, and continued psychiatric care where capacity is lacking runs under Section 89 for up to 30 days.

HOLD THIS

Steroid psychiatry appears within days of a dose change while the systemic disease settles; lupus cerebritis appears with the flare. If more steroid helps, it was the lupus.

WHAT EARNS THE MARKS

- **The direction of change**: improvement on more steroid points to the lupus, and that sentence is the answer.
- **Systemic activity read separately**: rash, nephritis and cytopenias, not the psychiatric picture alone.
- **A plan with the steroid running**: taper below the threshold dose, then treat, then monitor on a schedule.
- **The porphyria lists straight**: two ordinary benzodiazepines and two ordinary atypicals sit on the unsafe side.

SEE ALSO 04 Catatonia: the signs, Bush Francis, the lorazepam challenge

06 Capacity and the supported-admission ladder 09 HIV neuropsychiatry: brain injury, mania, ART interactions

9 · HIV neuropsychiatry: brain injury, mania, ART interactions

CONSULTATION-LIAISON · TOPIC 09

How is cognitive impairment in HIV classified now, and why did the older criteria fall?

SHORT CASE, CL AND VIVA

THE SHAPE OF THE ANSWER

1 The nosology moved	Frascati categories, then the brain injury framing that replaced them.
2 Why they fell	Healthy people misclassified; local norms differ from North American ones.
3 Legacy or active	Static under suppression is legacy; new decline is not.
4 Depression and mania	Secondary mania is irritable and slowed, not expansive.
5 Interactions	Ritonavir, valproate, carbamazepine, and the safe benzodiazepines.

The nosology moved, and knowing it moved is the first mark.

In 2007 a National Institute of Mental Health working group set the Frascati criteria, with 3 categories on testing across at least 5 domains.

- **Key number.** Asymptomatic neurocognitive impairment: at least 1 standard deviation below the normative mean in at least 2 domains without functional impairment.
- **Key number.** Mild neurocognitive disorder: the same deficit with mild interference. HIV-associated dementia: marked impairment with severe restriction.
- **Key number.** In 2023 the International HIV-Cognition Working Group, 20 members of whom 9, that is 45 per cent, were from low- and middle-income countries, issued 6 consensus recommendations replacing that framing.

1. Give the Frascati categories and their limitations.
2. What is HIV-associated brain injury?
3. Legacy against active injury, and cerebrospinal fluid escape.
4. How does depression present, and how do you treat it?
5. What is HIV secondary mania?
6. Which interactions will you avoid?

Why the criteria fell. Approximately 16 per cent of results fall more than 1 standard deviation below the mean on any single test, so requiring that across 2 domains falsely classifies over 20 per cent, and up to 70 per cent depending on battery composition, of healthy HIV-negative individuals as impaired.

In healthy HIV-negative adults from a low-income Cape Town community the mean Montreal Cognitive Assessment score was 21.7 out of 30 against a North American cut-off of 26 to 30.

The criteria classify 20 per cent to 60 per cent of people living with HIV as impaired, up to 90 per cent in some cohorts, and 20 different classification methods applied to one South African cohort gave rates from 20 per cent to 97 per cent.

The new framework separates injury caused directly by HIV from the total burden of comorbidity, ageing and medication, and requires abnormalities in at least 2 of 3 assessment areas.

FEATURE	LEGACY BRAIN INJURY	ACTIVE BRAIN INJURY
Timing	Before effective therapy, advanced immunosuppression, or perinatal acquisition	During treatment interruption, failure or compartmentalisation
Pathology	Quiescent neuronal loss and synaptodendritic pruning, lowering reserve	Active replication, neuroinflammation, microglial activation
Course	**Static; does not progress on suppressive therapy**	**Progressive decline or evolving white matter change**
Cerebrospinal fluid	Plasma and fluid HIV RNA suppressed below 50 copies/mL	Viral escape, intrathecal inflammation, or raised neurofilament light chain
Reversibility	Irreversible; unresponsive to treatment change	Treatable by optimising penetration or changing regimen

- **RULE** **New decline under plasma suppression is not legacy damage.** It demands evaluation for cerebrospinal fluid escape, CD8 encephalitis, immune reconstitution inflammatory syndrome, systemic comorbidity or major depression. Escape is detectable virus in the fluid despite plasma below 50 copies/mL, presenting as subacute decline with motor symptoms and diffuse white matter hyperintensities, managed by changing the regimen on fluid resistance testing. A nadir CD4 count below 200 cells per microlitre is a risk factor for the neurocognitive disorder.

Depression. Estimates of major depression in HIV infection and AIDS range from 15 per cent to 40 per cent. Sertraline, citalopram and escitalopram have little effect on the major cytochrome isoforms.

Sourced doses are fluoxetine 20 to 60 mg daily and citalopram 20 to 60 mg daily with a halved 10 mg start, amitriptyline 25 to 150 mg at night where pain or insomnia coexist, and venlafaxine from 37.5 mg daily to a maximum of 225 mg daily.

- **Concept.** Secondary mania differs in phenomenology: prominent irritability with psychomotor slowing, cognitive deficits and confusion rather than expansive grandiosity.
- **Concept.** It differs in stage, occurring with advanced immunosuppression, and in signs, with frequent focal or frontal release signs.
- **Concept.** That reflects direct basal ganglia and frontal injury, viral escape or opportunistic infection.
- **Trap.** Treating it as primary mania. Exclude toxoplasmosis, cryptococcal meningitis and neurosyphilis first.

THE INTERACTIONS THAT MATTER

Antipsychotics: ritonavir raises risperidone levels with reported extrapyramidal symptoms and neuroleptic malignant syndrome, but induces CYP1A2 and glucuronidation and can lower olanzapine exposure, so a higher olanzapine dose is sometimes needed. Where intramuscular sedation is unavoidable, haloperidol 5 mg intramuscularly is the last drug considered, because acute dystonia is frequent; one series of 19 patients found favourable outcomes with antipsychotics, 17 on haloperidol, at low doses. **Mood stabilisers:** sodium valproate is generally first-line, with pre-treatment transaminases and platelets and an additive fatty liver risk with didanosine, abacavir, lamivudine, stavudine and zidovudine; it may inhibit zidovudine glucuronidation, with a report of severe anaemia. Lithium avoids hepatic metabolism but is avoided in dehydration and renal impairment, with levels about 5 days after a dose change, then monthly, then 3 to 6 monthly. Carbamazepine is avoided for interactions with ritonavir, nevirapine and efavirenz, neutropenia risk and reported virological failure. **Benzodiazepines:** alprazolam, midazolam and triazolam depend on CYP3A4 and accumulate dangerously with ritonavir, so use glucuronidated lorazepam, oxazepam or temazepam; lorazepam 1 to 2 mg orally, maximum 12 mg daily, is the sourced range. **Efavirenz:** vivid dreams, insomnia, depersonalisation and suicidal ideation, with depression 13 per cent to 16 per cent and anxiety 8 per cent to 11 per cent, most prominent at initiation, eased by bedtime dosing about three hours before retiring.

TRAP Quoting numbers this material cannot support: a CD4 cut-off specific to HIV mania, the deficit threshold for HIV-associated dementia, the weeks to peak for efavirenz symptoms, the enactment year and sections of the HIV and AIDS Prevention and Control Act, or the dolutegravir dose in the first-line combination. The substantive rules are secure: explicit informed consent for testing, treatment and research, no disclosure of status without written consent, and no denial of mental health treatment on the basis of status. Verify the numbers from the Act and current national guidelines.

HOLD THIS

Brain injury, not disorder, and progression is the question. Static decline on suppression is legacy; new decline means escape, CD8 encephalitis, immune reconstitution or depression.

WHAT EARNS THE MARKS

- **Knowing the framing changed**: the first mark is the nosology, not a recital of deficits.
- **New decline investigated**: fluid escape, CD8 encephalitis, immune reconstitution, depression, named as a set.
- **Secondary mania described**: irritability with slowing and confusion, in advanced disease.
- **Interactions given by class**: glucuronidated benzodiazepines, valproate first, carbamazepine avoided.

SEE ALSO 07 Epilepsy and psychosis, including forced normalisation

08 The medical mimics: steroid, thyroid, adrenal, porphyria, autoimmune

11 Two iatrogenic cases: tardive syndromes and lithium toxicity

10 · Functional neurological disorder: positive signs and the explanation

CONSULTATION-LIAISON · TOPIC 10

This patient has weakness with no structural cause. Show me a positive sign and then explain the diagnosis to her.

SHORT CASE, NEUROLOGY BEDSIDE AND OSCE

THE SHAPE OF THE ANSWER

1 **Rule in, not out**	Positive evidence of internal inconsistency is the criterion.
2 **Hoover sign**	Two phases; power returns on contralateral hip flexion.
3 **Tremor entrainment**	Pace the good hand, watch the tremor follow or stop.
4 **The explanation**	Validate, name it, show the sign, frame cause broadly.
5 **Treatment rules**	Physiotherapy and psychological therapy; avoid anti-seizure drugs and opioids.

This is a rule-in diagnosis, not a diagnosis of exclusion.

DSM-5, in 2013, removed the requirement for an antecedent psychological stressor and requires positive clinical evidence of incompatibility between the symptoms and recognised neuro-anatomical or medical conditions; ICD-11 calls it dissociative neurological symptom disorder.

- **Key number.** It accounts for approximately one-third of outpatient neurology attendances, and functional movement disorders form 2 per cent to 20 per cent of movement disorder referrals.
- **Key number.** Mean onset is 37 to 50 years, and coexisting structural disease occurs in 22 per cent to around one-quarter of patients.
- **Concept.** So structural disease and functional disorder sit together often enough that finding one does not exclude the other.

1. How has the diagnostic concept shifted?
2. Demonstrate Hoover sign.
3. Elicit tremor entrainment.
4. Which other positive signs do you know?
5. Explain the diagnosis to this patient.
6. What are the pharmacological rules?

Hoover sign. Supine and relaxed, both your hands under the heels. In the voluntary phase the patient presses the paretic leg down or lifts it against resistance, and you feel weak or absent power.

In the synergistic phase you keep one hand under the paretic heel and the other over the anterior thigh of the good limb, then ask for hip flexion of the good leg against firm resistance.

In functional paresis you feel an immediate strong downward thrust under the paretic heel. That return of power shows the corticospinal pathway and peripheral motor apparatus are intact.

Tremor entrainment. Note baseline frequency, amplitude and distribution, then have the unaffected hand tap to an externally set rhythm you demonstrate, changing from slow to fast.

- **Concept.** Positive findings are entrainment of the tremulous limb to the tapping frequency, suppression or marked irregularity of the tremor, or failure to maintain the paced rhythm because the tremor is preserved instead.
- **Concept.** Contralateral ballistic movements produce a brief pause.
- **Key number.** An electrophysiological battery evaluating entrainment, frequency shift, ballistic pauses and tonic coactivation gave 89.5 per cent sensitivity and 95.9 per cent specificity.

DOMAIN	SIGN	POSITIVE FINDING
Tremor onset	Tonic coactivation	**Palpable agonist and antagonist co-contraction immediately before the tremor appears**
Fixed dystonia	Swivel chair test	**Plantarflexed or inverted foot posture normalises while the patient propels the chair**
Cranial dystonia	Facial spasm pattern	Unilateral downward lip pulling with ipsilateral jaw deviation and platysma contraction
Gait	Incongruous pattern	Knee buckling without falls; leg dragged behind rather than circumducted
Functional seizures	Closed eyes sign	**Active eye closure resisting passive opening, head shaking, pelvic thrusting**
Functional seizures	Duration and awareness	Prolonged episodes of many minutes with preserved awareness despite bilateral convulsions

THE EXPLANATION, IN SIX STEPS

Validate: the symptoms are genuine, real and disabling, not imagined or feigned. Name it. Use the hardware and software analogy: the wiring is intact, the program sending messages between brain and body is not running properly. Show the positive sign you elicited, and say it proves the pathways are undamaged and recovery is possible. Frame cause multifactorially rather than asserting trauma, describing an overwhelmed brain. Give a care pathway and verified resources. Mean diagnostic delay is 2 years, and transparent communication improves outcomes.

- **RULE** **Anti-seizure medication does not treat functional seizures and can worsen them; opioids worsen functional symptoms and cognition and should be avoided or deprescribed.** Specialised physiotherapy retraining automatic motor programmes, cognitive behavioural therapy, and treatment of comorbid depression, anxiety or PTSD carry the evidence.

TRAP Leaning on la belle indifference, or on primary and secondary gain, as discriminators. Indian examiners probe all three and none could be traced here, so no figure or claim is offered. The sourced position is that diagnosis rests on positive signs of internal inconsistency and incongruence, that patients do not consciously produce their symptoms, and that functional imaging shows distinct patterns during involuntary symptoms compared with intentional mimicry. Forcing a trauma formulation is the companion error: fewer than half of patients present with an identifiable trigger.

HOLD THIS

Positive signs, not normal scans. Hoover and entrainment are shown to the patient, because the demonstration is the treatment's first step.

WHAT EARNS THE MARKS

- **A sign demonstrated**: Hoover or entrainment performed on the patient, not recited from memory.
- **The explanation delivered**: hardware intact, software failing, with the sign shown as the proof.
- **Drugs positioned**: anti-seizure medication does not treat functional seizures, and opioids are deprescribed.
- **No trauma forced**: an identifiable trigger is absent in a large share of patients.

SEE ALSO 04 Catatonia: the signs, Bush Francis, the lorazepam challenge
07 Epilepsy and psychosis, including forced normalisation 18 Tics and Tourette syndrome

11 · Two iatrogenic cases: tardive syndromes and lithium toxicity

CONSULTATION-LIAISON · TOPIC 11

This patient on long-term antipsychotics has orofacial movements. Examine him, then tell me what you do with the drug.

SHORT CASE, MOVEMENT EXAMINATION

THE SHAPE OF THE ANSWER

1 Examine	Cranial to caudal, at rest, on activation, then walking.
2 Separate by latency	Acute dystonia in days; tardive syndromes after months to years.
3 The anticholinergic test	Classic dyskinesia worsens; tardive dystonia may improve.
4 Act on the drug	Do not raise the dose; switch, then add a VMAT2 inhibitor.
5 Lithium beside it	Grade by chronicity and neurology, not by the concentration.

Both ask the same thing: the drug did this, so what do you do with the drug.

- **Key number.** Tardive dyskinesia under DSM-5 is involuntary athetoid or choreiform movement lasting at least a few weeks, on a dopamine receptor blocking agent given at least a few months, or at least 1 month in a person aged 60 years or older.
- **Key number.** It persists at least 1 month after the drug stops, typically emerges after 1 to 2 years, and rarely before 3 months.

1. Examine this patient and conduct a movement scale assessment.
2. Differentiate tardive from acute drug-induced disorders.
3. Why is raising the dose a trap?
4. Give the VMAT2 inhibitor evidence.

5. Lithium: range, sampling, and severity grading.
6. Give the dialysis criteria.

Bedside movement examination. Observe at rest, on activation and walking, inspecting cranial to caudal: facial expression, lips and perioral region, jaw, tongue in the mouth and on protrusion, upper limbs, trunk, lower limbs.

Activation matters because the movements attenuate at rest or under voluntary suppression: tongue protrusion for impersistence or darting, rapid finger-to-thumb tapping to unmask orofacial or contralateral choreoathetosis, and walking for arm swing and axial posturing.

The formal scale rates seven objective anatomical items, items 1 to 7, and a baseline total of 6 or more is the research cutoff used in the ARM-TD and AIM-TD trials. **Document dentition, because loose dentures mimic dyskinesia.**

FEATURE	ACUTE DYSTONIC REACTION	AKATHISIA AND PARKINSONISM	TARDIVE SYNDROMES
Onset	**50 per cent within 48 hours; 90 per cent within 5 days**	Days to weeks	**1 to 2 years; rarely before 3 months**
Core signs	Painful sustained spasms: oculogyric crisis, torticollis, retrocollis, trismus	Subjective restlessness with pacing; or bradykinesia, cogwheel rigidity, resting tremor	Oral-buccal-lingual stereotypy, chorea, dystonia, tardive akathisia, tics
On stopping	Resolves within 1 month	Resolves within 1 month	**Persists at least 1 month; up to 87 per cent unresolved**
Anticholinergic	Rapid dramatic relief	Modest for akathisia, significant for parkinsonism	**Classic dyskinesia worsens; tardive dystonia may improve**

Table: latency and the anticholinergic response separate these; the anticholinergic row is the one candidates reverse.

Cumulative incidence rises by approximately 5 per cent per year over the first 5 years, reaching 49 per cent at 10 years and 68 per cent at 25 years.

Meta-analytic prevalence is 25.3 per cent across 41 studies of 11,493 patients, 32.4 per cent for first-generation and 13.1 per cent for second-generation agents.

Only approximately 2 per cent remit on cessation alone while 33 per cent to 55 per cent worsen acutely, and formal evidence assessment classifies withdrawal or switching as Level U, insufficient evidence.

Raising the dose suppresses the movements by intensifying D2 blockade but worsens receptor upregulation and accelerates progression, so switching to clozapine is the guideline-supported move.

Deutetrabenazine starts at 6 mg twice daily, that is 12 mg per 24 hours, titrating to 24, 36 or 48 mg per 24 hours.

In ARM-TD the mean dose was 38.8 mg with a scale change of -3.0 against -1.6 for placebo, p=0.019. In AIM-TD across 75 centres the 36 mg arm gave -3.3 points, p=0.001, and the 24 mg arm -3.2 points, p=0.003, while 12 mg was not significant at p=0.217 against placebo -1.4.

Valbenazine is once daily from 40 mg per 24 hours, studied up to 75 mg per 24 hours. Both are Level A; tetrabenazine sits at Level C, clonazepam and ginkgo biloba at Level B, and bilateral pallidal deep brain stimulation achieves a 50 per cent reduction at 6 months sustained to 11 years.

TRAP Naming ARM-TD or AIM-TD as tetrabenazine trials; both are deutetrabenazine trials. Prescribing trihexyphenidyl for classic oral-buccal-lingual stereotypy, which it worsens while genuinely helping tardive dystonia, is the clinical error. The movement scale severity anchors, the Schooler and Kane thresholds, the valbenazine titration step and the vitamin E evidence could not be traced here; verify before the exam.

Lithium. Target 0.8 to 1.2 mmol/L in acute mania and 0.5 to 0.8 mmol/L in maintenance. Draw at a trough of exactly 12 hours after the previous night dose; steady state comes after 4 to 5 days on a constant dose.

Check weekly after a change until stable, then at intervals of at least 3 months with renal and thyroid function. Clearance is 15 to 20 mL/min with a half-life of 18 to 36 hours, and more than 95 per cent is cleared unchanged by the kidneys.

- **Key number.** Mild toxicity above 1.5 mmol/L gives fine tremor, nausea, vomiting, anorexia, weakness and drowsiness.
- **Key number.** Moderate at 1.5 to 2.5 mmol/L gives coarse tremor, hyperreflexia, myoclonus, cerebellar dysarthria, ataxia and nystagmus, and confusion.
- **Key number.** Severe above 2.5 to 4.0 mmol/L gives stupor, rigidity, hypotension, generalised seizures, coma and cardiopulmonary collapse.

RULE **Chronicity governs severity, not the number.** In acute overdose brain distribution takes up to 24 hours, so an alarming level can sit alongside an intact examination; chronic toxicity presents at a modest median 1.6 mmol/L yet caused permanent damage at presentation concentrations as low as 1.8 to 3.2 mmol/L where eGFR was 21 to 54 mL/min/1.73 m2. Haemodialysis is recommended at Grade 1D for severe poisoning, for eGFR below 45 mL/min/1.73 m2 with a level above 4.0 mEq/L, and for reduced consciousness with a Glasgow Coma Scale below 15, seizures or life-threatening dysrhythmia regardless of level; it is suggested at Grade 2D above 5.0 mEq/L, for confusion, or where the expected time to fall below 1.0 mEq/L exceeds 36 hours.

TRAP The phlebotomy tube and the charcoal reflex. Green-top tubes containing lithium heparin produce factitiously high readings, so use plain red-top or gel separator yellow-top tubes. Charcoal does not bind lithium ions and only adds aspiration risk. Volume repletion is the essential intervention, often 4 to 6 litres of crystalloid initially then 200 to 250 mL/hr, with levels every 2 to 4 hours until the peak and then every 6 to 12 hours; thiazides raise levels by an average of 25 per cent and up to 4-fold, and non-steroidal anti-inflammatory drugs by 10 per cent to 25 per cent, with paracetamol and aspirin the safe analgesics. Rapid lowering of a high chronic level is itself hazardous.

HOLD THIS

Do not raise the antipsychotic to hide the movements, and do not read a lithium level without asking how long the exposure has run. Clozapine plus a VMAT2 inhibitor; grade lithium by chronicity and neurology.

WHAT EARNS THE MARKS

- **Activation manoeuvres used**: tapping and walking unmask what rest conceals.
- **The anticholinergic row straight**: classic dyskinesia worsens, tardive dystonia may improve.
- **The dose not raised**: suppression by more blockade accelerates the underlying process.
- **Lithium graded clinically**: duration of exposure and neurology decide, and not the concentration alone.

SEE ALSO 04 Catatonia: the signs, Bush Francis, the lorazepam challenge

05 Rigid and febrile: NMS, serotonin syndrome, malignant catatonia 18 Tics and Tourette syndrome

Substance and emergency cases

12 · Alcohol withdrawal: CIWA-Ar, benzodiazepines, seizures, delirium tremens

SUBSTANCE AND EMERGENCY · TOPIC 12

This patient stopped drinking yesterday and is tremulous and sweating. Stage the withdrawal and choose the regimen.

SHORT CASE, EMERGENCY AND DETOXIFICATION WARD

THE SHAPE OF THE ANSWER

1 Stage by the clock	Hours since the last drink set the expected stage.
2 Score it	What the scale contains, its thresholds, and its limits.
3 Choose the drug	The liver decides which benzodiazepine you reach for.
4 Seizures	Benzodiazepine, capillary glucose, and no phenytoin.
5 Delirium tremens	Monitored bed, electrolytes, then the escalation agents.

Stage it by the clock from the last drink, then choose the regimen by the liver.

- **Key number.** Signs can appear as early as 6 hours after the last drink.
- **Key number.** Withdrawal seizures fall between 12 and 48 hours, generalised tonic-clonic, brief and self-terminating.
- **Key number.** More than 3 seizures or status epilepticus is rare and mandates investigation for another cause.
- **Key number.** Delirium tremens develops in approximately 5 per cent of withdrawing patients and peaks around 96 hours.

1. Give the timeline and staging.
2. What does the CIWA-Ar contain, and what are its thresholds and limits?
3. Fixed-schedule against symptom-triggered dosing.
4. Which benzodiazepine in liver disease, and why?
5. Manage a withdrawal seizure.

6. What is kindling?
7. Manage refractory delirium tremens.

The scale. The CIWA-Ar has 10 items, nine rated 0 to 7 and orientation rated 0 to 4, giving a maximum of 67.

A score of 8 or greater triggers pharmacotherapy, 15 or greater triggers a PRN rescue dose in a patient on a scheduled regimen, and greater than 11 indicates a significant inpatient detoxification regimen.

If the initial score is 8 or greater, assess hourly for 8 hours, then 2 hourly for 8 hours, then 4 hourly; if below 8, assess 4 hourly for 72 hours and stop scoring if it stays below 8 for 72 hours. Response is assessed 30 to 60 minutes after a dose.

DRUG	METABOLISM	USE	CEILING
Chlordiazepoxide	Hepatic oxidation	Oral inpatient taper, normal liver function	**Maximum 200 mg per 24 hours, regular plus PRN**
Diazepam	Hepatic oxidation; long-acting active metabolites give a smooth self-taper	Rapid parenteral control: 5 mg intravenous bolus, then 5 to 20 mg every 5 to 15 minutes to light sedation	**Maximum 100 mg/h or 250 mg in 8 hours**
Lorazepam	Glucuronidation, no active metabolites	**Liver disease, elderly, low body weight, withdrawal fits: 1 to 4 mg every 5 to 15 minutes**	**Maximum 20 mg/h or 50 mg in 8 hours**
Oxazepam	Glucuronidation	Mild to moderate withdrawal with liver impairment	Tapered over 1 week

Table: the metabolic route is the whole answer in liver disease; rapid intravenous lorazepam also carries propylene glycol toxicity from its vehicle.

Fixed schedule. Regimens taper over 5 to 7 days, or up to 2 weeks in severe dependence, and can be guided by the Severity of Alcohol Dependence Questionnaire, 20 items scored out of 60.

- **Key number.** A score of 31 or higher takes chlordiazepoxide 30 to 40 mg four times daily, 16 to 30 takes 20 to 30 mg four times daily, and below 16 does not routinely need benzodiazepines.
- **Key number.** Breakthrough symptoms take PRN chlordiazepoxide 10 mg orally every 2 to 4 hours, and 3 or more PRN doses within 24 hours means the regimen needs review.
- **Concept.** Symptom-triggered dosing lowers total benzodiazepine use and shortens stay, but needs trained nursing for hourly monitoring and is unsuited to established delirium tremens.

RULE **Phenytoin should not be offered for alcohol withdrawal seizures.** The mechanism is transient GABA-A hypofunction with NMDA upregulation, which answers to a benzodiazepine, not a sodium channel blocker. Without intravenous access, buccal midazolam 10 mg, repeatable once after 10 minutes to a maximum of 20 mg, with emergency transfer if convulsions continue. Flumazenil can precipitate intractable seizures here. Check capillary glucose immediately after any seizure.

KINDLING, AND THE FOURTH DETOXIFICATION

Prolonged heavy use upregulates NMDA receptors and downregulates GABA-A receptors, so abrupt cessation produces a high glutamatergic, low GABAergic state. Repeated withdrawal episodes become progressively more severe through increased neuronal excitability and sensitivity, the kindling effect, so the number and recency of prior episodes inform the level of care. Seizures are more likely where there is a previous history of fitting, past withdrawal seizures or past delirium tremens mandate parenteral thiamine and closer observation, and apparent benzodiazepine resistance may itself reflect kindling.

Refractory delirium tremens. Monitored high-dependency bed, quiet low-stimulus room, restraint only until chemical sedation works, vigorous correction of hypokalaemia and hypomagnesaemia, and high-dose intravenous benzodiazepines as the primary modality.

Where symptoms fail to settle, add phenobarbital or pentobarbital boluses at distinct GABA-A binding sites; propofol infusion, which needs mechanical ventilation; ketamine at a 0.3 mg/kg load then 0.15 to 0.30 mg/kg/h; or dexmedetomidine for sympatholysis without respiratory depression, though it does not reduce ventilation rates or length of stay.

Haloperidol 5 mg intravenously or intramuscularly, repeatable in 30 to 60 minutes, may be added for uncontrolled psychotic agitation but not as monotherapy, because neuroleptics lower the seizure threshold and prolong the QTc interval.

TRAP Scoring a delirious patient with the CIWA-Ar, which needs intact comprehension and speech and is not validated for rapid emergency triage: use the Richmond Agitation-Sedation Scale instead. The other error is a discharge benzodiazepine prescription, because resumed drinking alongside one risks fatal respiratory depression; step down over 3 to 4 days, or 5 to 7 days, monitoring respiratory rate, saturation and conscious level.

HOLD THIS

A score of 8 starts treatment, 15 triggers rescue, seizures fall 12 to 48 hours, delirium tremens peaks around 96 hours. Glucuronidated benzodiazepine if the liver is failing, and no phenytoin for these fits.

WHAT EARNS THE MARKS

- **The clock given first**: staging from the last drink is what the examiner is waiting to hear.
- **The metabolic route named**: glucuronidation is the whole answer in liver disease.
- **Phenytoin refused with a reason**: the receptor mechanism explains why it does not work here.
- **Kindling applied**: the count and recency of prior withdrawals set the level of care.

SEE ALSO 05 Rigid and febrile: NMS, serotonin syndrome, malignant catatonia

13 · Thiamine, Wernicke encephalopathy, and the dextrose rule

SUBSTANCE AND EMERGENCY · TOPIC 13

Why will oral thiamine not do in a patient you suspect has Wernicke encephalopathy?

SHORT CASE, EMERGENCY AND DETOXIFICATION WARD

THE SHAPE OF THE ANSWER

1 **The absorption ceiling**	Active transport saturates, so oral dosing cannot reach treatment levels.
2 **Prophylaxis**	Parenteral for a few days in the at-risk patient, then oral.
3 **Treatment**	High-dose parenteral, then step down on documented improvement.
4 **The dextrose rule**	Thiamine first or together, and treat hypoglycaemia immediately.
5 **Magnesium**	A cofactor, so the refractory case is a magnesium problem.

The triad is confusion, ataxia and ophthalmoplegia, incomplete presentations are frequent, and most cases are identified only post mortem.

Inadequate treatment leads to Korsakoff syndrome, an irreversible amnestic state with anterograde amnesia and confabulation and relatively preserved other intellectual functions, conferring a 10 per cent to 20 per cent increased mortality risk.

1. Why is oral thiamine inadequate here?
2. Give the prophylactic regimen.
3. Give the treatment regimen.
4. Why must thiamine precede dextrose, and what is the exception?
5. Why does magnesium matter?
6. Separate Wernicke from hepatic encephalopathy.

- **Key number.** Oral bioavailability is 3.7 per cent to 5.3 per cent.
- **Key number.** Active intestinal transport saturates at approximately 8.3 mg from a single oral dose, with minimal urinary excretion above 2.5 mg.
- **Concept.** Alcohol-induced mucosal injury suppresses absorption further, so the ceiling in a drinking patient is lower still.

Prophylaxis. In severe dependence, or in mild to moderate dependence with risk factors such as malnutrition, previous severe withdrawal, current withdrawal, past Wernicke encephalopathy or delirium tremens, recurrent vomiting or peripheral neuropathy, give 1 pair of parenteral ampoules daily for 3 to 5 days.

Then oral thiamine 100 mg three times daily with a daily multivitamin. Mild to moderate dependence without risk factors takes oral thiamine 100 mg three times daily and 1 multivitamin tablet daily.

Treatment. For suspected or established Wernicke encephalopathy the high-dose regimen is thiamine 500 mg intravenously three times daily for 2 to 3 days, infused over 30 minutes.

If improvement is documented continue 250 mg parenterally once daily for 5 days or as long as improvement continues, and if there is no improvement after 2 to 3 days the high-dose course stops.

The traditional alternative is 100 mg intravenously or intramuscularly daily for 3 to 7 days then indefinite oral maintenance, with delayed responses reported up to 30 days after starting.

Anaphylaxis risk is estimated at 1 per 5 million pairs of intramuscular ampoules, with zero cases in a series of over 300,000 patients; observe for 30 minutes after an intramuscular dose with resuscitation equipment available.

RULE **Thiamine before or with the dextrose, but do not withhold glucose from a hypoglycaemic patient.** Intravenous glucose in a thiamine-depleted patient accelerates glycolysis and consumes remaining thiamine pyrophosphate, precipitating acute Wernicke encephalopathy or irreversible brainstem necrosis. But neuroglycopenia kills neurons within minutes while thiamine crosses the blood-brain barrier over several hours, so if bedside testing shows hypoglycaemia give dextrose 50 per cent in water, 25 to 50 mL intravenously, immediately, with concurrent thiamine 100 mg intravenously.

MAGNESIUM, THE STEP CANDIDATES SKIP

Magnesium is an essential cofactor for transketolase and for thiamine pyrophosphokinase, the enzyme converting thiamine into thiamine pyrophosphate. In hypomagnesaemia patients are clinically refractory to thiamine replacement, so serum magnesium and potassium must be measured and actively normalised. Saying this unprompted answers the question before it is asked.

FEATURE	WERNICKE ENCEPHALOPATHY	HEPATIC ENCEPHALOPATHY
Eye signs	**Nystagmus, lateral rectus palsy**	Absent
Motor signs	**Cerebellar ataxia**	**Asterixis, constructional apraxia**
Systemic clues	Malnutrition, recent poor intake	Fetor hepaticus, signs of portal hypertension
Response	**Parenteral thiamine**	Directed at the hepatic driver

TRAP Two things circulate here that could not be traced: the numeric ammonia threshold and the named treatments for hepatic encephalopathy, and the interictal electroencephalographic description used to separate withdrawal seizures from idiopathic epilepsy. Verify both rather than quoting a figure. Post-detoxification, continue oral thiamine 100 mg three times daily and a daily multivitamin for at least 1 month, or indefinitely with ongoing consumption or chronic nutritional deficiency.

HOLD THIS

Oral thiamine cannot treat acute Wernicke encephalopathy: absorption ceilings at approximately 8.3 mg per dose with 3.7 per cent to 5.3 per cent bioavailability. Parenteral, high dose, and correct the magnesium or the thiamine will not work.

WHAT EARNS THE MARKS

- **The absorption ceiling quoted**: it is the reason oral thiamine cannot treat the acute state.
- **The dextrose rule given both ways**: thiamine first, and glucose still given to a hypoglycaemic patient.
- **Magnesium raised unprompted**: it answers the refractory case before the question arrives.
- **The consequence named**: undertreatment leaves an irreversible amnestic state.

SEE ALSO 12 Alcohol withdrawal: CIWA-Ar, benzodiazepines, seizures, delirium tremens

04 Catatonia: the signs, Bush Francis, the lorazepam challenge

08 The medical mimics: steroid, thyroid, adrenal, porphyria, autoimmune

14 · After deliberate self-harm: lethality, intent, safety planning

SUBSTANCE AND EMERGENCY · TOPIC 14

This patient has been medically cleared after an overdose. How do you assess lethality and intent?

SHORT CASE, EMERGENCY AND LIAISON

THE SHAPE OF THE ANSWER

1	After clearance	Exclude delirium and intoxication, then interview in three phases.
2	Two dimensions	Medical lethality and suicidal intent, separately rated.
3	The scale	Composition, banding, and what it cannot be used for.
4	Factor structure	Planning predicts damage; lethal intent tracks hopelessness.
5	Safety plan	Six steps, ending in restriction of lethal means.

Lethality and intent are separate dimensions with surprisingly low correlation.

Assessment begins only after resuscitation, with delirium, toxic encephalopathy and intoxication excluded. Where consciousness fluctuates, cognitive screening precedes psychometric work.

- **Concept.** Pre-attempt: precipitants, entrapment and cognitive rigidity, acquisition of means and planning.
- **Concept.** Intra-attempt: isolation, timing, and precautions taken against discovery.
- **Concept.** Post-attempt: how discovery happened, whether help was sought, and the reaction to survival.

1. Structure the evaluation after medical clearance.
2. Separate medical lethality from suicidal intent.
3. Give the composition and banding of Beck's Suicide Intent Scale.
4. What does factor analysis show?
5. Can the score clear this patient for discharge?
6. Separate non-suicidal self-injury from a low-intent attempt.

7. Construct a safety plan.

WHERE THE TWO DIMENSIONS COME APART

High intent, low lethality: a patient swallows over-the-counter vitamin tablets believing any overdose causes irreversible cardiac arrest. **Low intent, high lethality:** an adolescent impulsively swallows an agricultural organophosphate to communicate despair, believing lavage is an assured antidote. Regression shows actual physical damage is predicted by objective planning behaviours, $t = 2.76$, $p = 0.007$, whereas subjective lethal intent does not independently predict damage, and clinician-rated danger to life does not reliably match measured intent.

The scale. Beck developed the Suicide Intent Scale in 1978, with earlier validation in 1974. It is a 20-item clinician-administered semi-structured interview, not a self-report checklist.

Three sections: objective circumstances, items 1 to 8; self-report, items 9 to 15, where premeditation is coded 0 for impulsive acts, 1 for contemplation of 3 hours or less and 2 for longer; and other aspects, items 16 to 20, whose 5 items are excluded from the total.

Each scored item is rated 0 to 2 on a 3-point scale, an inapplicable item is coded -8 and excluded, and the total across items 1 to 15 ranges 0 to 30, banded in clinical templates as 0 to 10 low risk, 11 to 19 medium and 20 to 30 high.

Factor structure. Polychoric factor analysis on inpatients, mean age 28 plus or minus 10 years, 62.2 per cent female, 93 per cent with depressive disorders, identified 2 factors.

- **Key number.** Lethal Intent covers items 9 to 14, Cronbach alpha 0.90, correlating with the Beck Hopelessness Scale, $r = 0.27$, $p = 0.02$.
- **Key number.** Planning covers items 1 to 7 and 15, Cronbach alpha 0.74, with no correlation with hopelessness, $r = -0.06$, $p = 0.59$.
- **Key number.** Neither correlates with depressive severity on the 24-item Hamilton scale. The 15-item total has Cronbach alpha 0.81 and inter-rater reliability $r = 0.95$ in 45 attempters.
- **Trap.** Reading advance communication as high intent. Item 8 loaded 0.30 on the first factor and -0.14 on the second with a negative item-total correlation, $r = -0.10$, $p = 0.35$: communication is an idiosyncratic behavioural style, not a core dimension of intent.

RULE **No scale predicts which individual will die by suicide.** After self-harm the risk of completed suicide is 0.5 per cent to 2 per cent at 1 year and exceeds 5 per cent after 9 years, with mortality 3.3 times expected rates, and index methods of hanging, deep cutting, gas or traffic trauma confer a 1.8 to 5-fold increase; a score of 19 or higher, age above 55 years and mood disorders significantly predict subsequent completed suicide. Meta-analyses establish that no scale has sufficient sensitivity or positive predictive value to serve as a triage tool, because the low base rate keeps false positives high. Scales structure the inquiry; formulation governs disposition.

Non-suicidal self-injury. Its function is affect regulation, relief of intolerable tension, self-punishment, anti-dissociation or communication, accompanied by an urge to relieve distress while continuing to live, and typically enacted as repetitive superficial cutting, scratching or minor burning.

A suicide attempt functions to escape, to end consciousness, or to resolve insoluble agony, accompanied by perceived burdensomeness, thwarted belongingness and defeat, using methods perceived as fatal.

- **Concept.** The link between them is acquired capability: repetitive self-injury habituates the person to pain and fear of death, progressively raising vulnerability to high-intent acts.

THE STANLEY-BROWN SAFETY PLAN, SIX STEPS

Warning signs. Internal coping strategies used alone. Social contacts and settings for distraction, without disclosing the crisis. Family members or trusted friends approached with explicit disclosure and a request for help. Professionals and crisis agencies. Lethal means restriction. The Tele-MANAS helpline is 14416 and national emergency services is 112. An optional seventh step records reasons for living. Low-lethality items may be limited by the patient, but high-lethality means must be delegated to responsible family members, caregivers or law enforcement, and in Indian practice that restriction must name toxic agrochemicals, not only medications and sharp implements.

TRAP Offering a no-suicide contract: it has no empirical validation and no legal protection, offers no coping alternative, and induces guilt when urges resurface. The statutory trap is quoting the section decriminalising attempted suicide, the penal provision it displaces, or the supported-admission duration attached to this material, none of which could be traced. The secure substance is that a person who attempts suicide is presumed to have been under severe stress and is to receive medical care, psychosocial support and rehabilitation rather than prosecution, and that supported admission where capacity is lacking runs under Section 89 for 30 days. The Indian method distribution and its toxicological management are likewise untraced: what is secure is that mental state assessment waits until the toxidrome is controlled and delirium excluded.

HOLD THIS

Planning predicts damage; lethal intent predicts hopelessness. Item 8, advance communication, loads on neither factor and is not a marker of high intent.

WHAT EARNS THE MARKS

- **The two dimensions separated**: high intent can sit with a harmless method, and the reverse.
- **The scale positioned honestly**: it structures the inquiry, and formulation governs disposition.
- **Means restriction that fits India**: agrochemicals named alongside medicines and sharp implements.
- **The no-suicide contract refused**: no validation, no legal protection, and it induces guilt.

SEE ALSO 03 Organicity, safety, and the one-line formulation 06 Capacity and the supported-admission ladder 19 Eating disorders and the SCOFF

15 · Cannabis-induced psychosis and acute intoxication

SUBSTANCE AND EMERGENCY · TOPIC 15

This young man is psychotic and uses cannabis heavily. Is this a cannabis-induced disorder or schizophrenia?

SHORT CASE, EMERGENCY AND ADDICTION

THE SHAPE OF THE ANSWER

1 **The criteria**	DSM-5 timing, and the excess-of-dose threshold ICD adds.
2 **Intoxication or disorder**	Reality testing is the line between them.
3 **Against schizophrenia**	Visual hallucinations, preserved insight, few negative symptoms.
4 **Conversion**	Highest of the substances, and mostly in the early years.
5 **Acute management**	Unknown substance means no benzodiazepine.

Intoxication leaves reality testing intact; the disorder does not.

DSM-5 needs delusions or hallucinations developing during, or within 1 month of, intoxication or withdrawal, not better explained by an independent disorder, not exclusively during delirium, and causing significant distress or impairment.

ICD-10 and ICD-11 add a threshold DSM-5 does not: the symptoms must be in clear excess of what that dose would produce, which is why ICD-based studies select more severe presentations.

1. Give the criteria and the divergence from ICD.
2. Separate intoxication from the psychotic disorder.
3. Separate the disorder from schizophrenia with comorbid misuse.
4. Give the conversion rates.
5. The substance is unknown and the patient is agitated.
6. The urine is positive. Does that settle Criterion B?

In simple intoxication people recognise that the temporal distortion, euphoria, anxiety, depersonalisation and mild paranoid ideas come from the drug. **In the disorder reality testing is compromised, delusions become fixed and hallucinations dominate.**

- **Key number.** Global Drug Survey data show 56 per cent of respondents had cannabis-associated psychosis resolve within 24 hours, while 21 per cent had symptoms lasting at least a few weeks.
- **Concept.** Symptoms persisting beyond a few weeks are likely driven by an independent disorder.
- **Key number.** Between 7 per cent and 25 per cent of first-episode presentations in specialist services are substance or medication induced.
- **Key number.** In an emergency cohort of 386 patients with early psychosis and regular substance use, 169, that is 44 per cent, had substance-induced psychotic disorder, with polysubstance use in 40 per cent.
- **Key number.** Population incidence of cannabis-induced psychotic disorder is approximately 2.7 per 100,000 person-years.

DOMAIN	CANNABIS-INDUCED PSYCHOTIC DISORDER	SCHIZOPHRENIA WITH COMORBID MISUSE
Hallucinations	**Visual significantly more frequent; non-verbal auditory**	**Verbal auditory: running commentary, third person**
Delusions	Paranoid, grandiose or religious, with hostility and agitation	Persecutory, passivity, thought insertion, bizarre
Negative symptoms	**Less prominent, with significantly lower scores**	**Pervasive flattening, alogia, avolition, anhedonia**
Affective profile	High rates of depression, hypomania, somatisation, phobic anxiety, panic	Blunted or incongruous affect
Insight	Relatively preserved reality testing and better insight	Marked loss of reality testing, poor insight
Consumption	Heavier: 80 per cent smoke more than 8 joints per day	Variable or lower; dependence less severe
Family history	Higher rates of parental substance abuse	Higher rates of nonaffective psychosis in first-degree relatives
Sensorimotor gating	Pre-pulse inhibition deficit at the 30 ms preattentive level, intact at higher levels	Deficits across preattentive and higher attentional gating

Table: visual hallucinations, absent negative symptoms and preserved insight point away from schizophrenia.

RULE **Cannabis unmasks, it does not create.** Conversion to schizophrenia spectrum disorders is highest for cannabis at 40 per cent to 45 per cent, reaching 46 per cent in Finnish data, against 30 per cent to 35 per cent for methamphetamine and 4.7 per cent to 22 per cent for alcohol, with an 18 per cent cumulative hazard for narrow schizophrenia in Swedish data, and most conversions within the first 3 years. In 7,606 individuals followed an average of 7 years, the 11 per cent who converted had familial risk scores for nonaffective psychosis identical to patients with primary schizophrenia and higher than the 89 per cent who did not, while familial risk for alcohol and drug abuse did not differ. Younger age, male sex, self-harm after the episode, poor premorbid adjustment and absent insight predict transition.

RULE **When the ingested substance is unknown, avoid benzodiazepines.** Co-ingested sedatives, opioids or alcohol compound respiratory depression, so low-potency first-generation antipsychotics are given instead for calming without compromising respiratory drive. Where cannabis or a stimulant is confirmed, symptom-guided benzodiazepines and second-generation antipsychotics are used, though only 2 clinical trials have formally evaluated pharmacological treatment here, so practice remains empirical.

TRAP Treating a positive urine cannabinoid screen as proof of temporal causation. It confirms exposure, not timing, and Criterion B asks when the symptoms developed. Metabolites persist well past acute intoxication, with prolonged urinary detection reported up to 102 days of abstinence in a case report. The routine detection window in heavy users could not be traced here, so quote no figure: argue timing from the pattern and recency of use, the mental state, and collateral informants who can say whether there has been a month or more of abstinence.

INDIAN PRACTICE NOTE

Antipsychotics are commonly started immediately despite the theoretical value of a drug-free observation period, because distress and behavioural risk demand it; where symptoms remit rapidly after substance clearance, taper slowly over several months while watching for re-emergence, since a brief or uncertain drug-induced episode may not need long-term maintenance. Because substance relapse triggers psychotic recurrence and raises conversion risk, integrated dual-diagnosis care with relapse prevention, family education, vocational support and safe housing is indicated. The Narcotic Drugs and Psychotropic Substances Act distinguishes bhang from ganja and charas, but its enactment year and statutory definitions could not be traced here. Continuing care runs through the District Mental Health Programme and Tele-MANAS.

HOLD THIS

Visual hallucinations, preserved insight, few negative symptoms, heavy use. A positive urine screen proves exposure, not timing.

WHAT EARNS THE MARKS

- **The ICD threshold named**: symptoms in clear excess of the dose, which DSM-5 does not require.
- **Reality testing as the divide**: it separates simple intoxication from the psychotic disorder.
- **Conversion quoted with its meaning**: familial risk is unmasked rather than created.
- **The urine screen read correctly**: it proves exposure, and Criterion B asks about timing.

SEE ALSO 07 Epilepsy and psychosis, including forced normalisation

12 Alcohol withdrawal: CIWA-Ar, benzodiazepines, seizures, delirium tremens

16 Adult ADHD: assessment, ASRS, treatment in India

Neurodevelopmental cases

16 · Adult ADHD: assessment, ASRS, treatment in India

NEURODEVELOPMENTAL · TOPIC 16

This adult describes lifelong inattention. Give me the DSM-5 thresholds for adult ADHD.

SHORT CASE, ADULT NEURODEVELOPMENTAL STATION

THE SHAPE OF THE ANSWER

1	Thresholds	Symptom count, age of onset, settings, duration, impairment.
2	Screen and score	Which part, how many boxes, and what a positive means.
3	Differential	Course, plus the quality of the attention deficit.
4	Prescribe in India	What exists here, and the rules that bind it.
5	Monitor	Cardiovascular checks, review intervals, annual review of need.

The diagnosis is clinical, longitudinal and cross-situational; no test confirms it.

- **Key number.** It affects approximately 4 per cent to 5 per cent of adults, and assessment usually needs at least 2 visits.

- **Key number.** DSM-5 asks for at least 5 symptoms in either domain persisting at least 6 months, several present before age 12, impairment in at least 2 settings, and clear interference with functioning.
- **Concept.** Corroborate from partners, parents or school records wherever feasible, because the history is the instrument.

1. Give the DSM-5 thresholds for adults.
2. Score and interpret the self-report scale.
3. Differentiate from mood, anxiety and personality disorder.
4. What can you prescribe in India, and under what rules?
5. What monitoring is required?
6. How does stimulant withdrawal present?

The self-report scale. Version 1.1 of the Adult ADHD Self-Report Scale has 18 items, with Part A the 6 most predictive questions and Part B the remaining 12, taking approximately 5 minutes over the past 6 months.

4 or more marks in the shaded boxes of Part A is a positive screen.

The DSM-5 version is a 6-question screener for adults aged 18 and older with intellectual ability in the normal range, scored across 5 frequency anchors from 0 to 4 for a total of 0 to 24, with 14 as the cut-off.

- **Key number.** In general population surveys weighted to an 8.2 per cent prevalence, sensitivity is 91.4 per cent, specificity 96.0 per cent, area under the curve 0.94 and positive predictive value 67.3 per cent.
- **Key number.** In clinical specialty samples at 57.7 per cent prevalence, sensitivity is 91.9 per cent, specificity 74.0 per cent, area under the curve 0.83 and positive predictive value 82.8 per cent.
- **Concept.** So the same instrument performs differently by setting, which is the point to make when asked about its value.

ENTITY	COURSE	QUALITY OF THE ATTENTION DEFICIT	IMPULSIVITY
Adult ADHD	**Chronic, continuous, onset before age 12**	Executive dysfunction, worst on boring repetitive tasks	Impatience, interrupting, verbal blurting
Bipolar disorder	**Episodic, with distinct episodes and baseline recovery**	Impaired during depressive or manic episodes	Linked to grandiosity; reduced need for sleep
Borderline personality disorder	Pervasive instability from early adulthood	Disrupted by interpersonal crises and dissociation	**Self-harm and reckless spending triggered by fears of abandonment**
Generalised anxiety disorder	Fluctuating with life stress	**Driven by worry; normalises when anxiety subsides**	Low; agitation is somatic tension
Autism spectrum disorder	Neurodevelopmental, early childhood	Hyperfocus on special interests, resistance to disruption	Rigidity and insistence on sameness rather than disinhibition

WHAT IS ACTUALLY PRESCRIBABLE

Methylphenidate is manufactured in India in immediate-release and sustained or extended-release forms, and the release systems and brand lines are not interchangeable; immediate-release acts within roughly 1 hour and wears off across 3 to 4 hours, while atomoxetine gives modest gains by 2 weeks but needs 4 to 8 weeks for full effect. Clonidine is available but licensed for hypertension, so its use here is off-label and specialist-led. Mixed amphetamine salts and lisdexamfetamine are not verified Indian retail options, and guanfacine and viloxazine have no established domestic supply. Methylphenidate falls under Schedule X of the Drugs Rules and the Narcotic Drugs and Psychotropic Substances framework, prescriptions are written in duplicate with the pharmacy retaining 1 copy for 2 years, and it sits on the prohibited list under the Telemedicine Practice Guidelines, so both initial and repeat prescriptions require an in-person consultation. Atomoxetine is not on that list.

Monitoring. Blood pressure and pulse before and after each dose adjustment and at least every 6 months; weight every 6 months; no routine electrocardiogram unless there is cardiac history or physical signs.

Review within 30 days of initiation, monthly until stable, then every 3 to 6 months, with a formal review of continued need at least once a year.

Cardiac referral and a pre-treatment electrocardiogram are indicated for unexplained sudden cardiac death in a first-degree relative under age 40, exertional syncope or chest pain, or a pathological murmur. There are 4 FDA-approved non-stimulants: atomoxetine, clonidine, guanfacine and viloxazine.

Withdrawal. A crash over 1 to 2 days with prolonged sleep, depressed mood, irritability, overeating and mild cravings.

Then persistent dysphoria over days to weeks with at least 2 of insomnia or hypersomnia, fatigue, vivid unpleasant dreams, psychomotor agitation or retardation, and cravings; psychotic symptoms or severe agitation may emerge in the first 1 to 2 weeks.

Support with hydration of at least 2 to 3 litres daily plus B-complex and vitamin C, and for severe agitation diazepam 5 to 10 mg orally four times daily as needed or olanzapine 2.5 to 5 mg orally twice daily as needed, strictly for a maximum of 7 to 10 days.

TRAP Using paediatric thresholds in an adult: the adult cut-off is 5 symptoms, and a positive Part A screen of 4 or more shaded boxes warrants assessment rather than confirming a diagnosis. The companion errors are promising immediate benefit from atomoxetine, prescribing methylphenidate over teleconsultation, writing a single rather than duplicate Schedule X prescription, and ordering blanket electrocardiograms. Whether adult ADHD is a notified benchmark disability under the Rights of Persons with Disabilities Act 2016, and any certification pathway, could not be traced here; read the statute directly and quote no percentage.

HOLD THIS

Five symptoms, before age 12, in 2 settings, for 6 months. A screen of 4 shaded boxes opens the assessment; methylphenidate needs an in-person consultation and a duplicate Schedule X prescription.

WHAT EARNS THE MARKS

- **Adult thresholds rather than paediatric**: the symptom count drops in adults, and that is the discriminating detail.
- **A screen kept as a screen**: a positive Part A opens the assessment and confirms nothing.
- **The prescribing rules**: duplicate Schedule X and an in-person consultation, stated unprompted.
- **Targeted cardiac testing**: an electrocardiogram on indication, not as a blanket order.

SEE ALSO 15 Cannabis-induced psychosis and acute intoxication 17 Autism: DSM-5, the ISAA, and disability certification 18 Tics and Tourette syndrome

17 · Autism: DSM-5, the ISAA, and disability certification

NEURODEVELOPMENTAL · TOPIC 17

This child has social communication difficulty. Which instrument is mandated for certification in India, and how is it scored?

SHORT CASE, CHILD AND CERTIFICATION STATION

THE SHAPE OF THE ANSWER

1 Two sources	Caregiver developmental history plus direct observation.
2 The instrument	Items, domains, administration, and the frequency anchors.
3 Threshold and bands	The diagnostic cut-off, then the certification ladder.
4 Its limits	Validated for classification, weak for staging severity.
5 DSM-5 counting	All three in A, two of four in B, severity rated separately.

Two sources of information and one statutory instrument. Diagnosis rests on caregiver developmental history and direct observation.

- **Key number.** In Indian community studies prevalence in children aged 2 to 9 years is 0.8 per cent to 1.3 per cent, comparable to international estimates of 0.67 per cent to 1.2 per cent.
- **Key number.** 87 per cent of clinic-referred children show comorbid intellectual disability or global developmental delay.

1. Which instrument is mandated for certification in India?
2. Give its structure and scoring.
3. Give the diagnostic threshold and disability bands.
4. What are its psychometric limits?
5. Give the DSM-5 counting rule and severity levels.
6. Differentiate from social communication disorder and intellectual disability.

The instrument. The Indian Scale for Assessment of Autism was developed by the National Institute for the Mentally Handicapped under the Ministry of Social Justice and Empowerment

with the Ministry of Health and Family Welfare and the National Trust, and is the designated instrument for certifying autism-related disability.

It has 40 items across 6 domains: social relationship and reciprocity, 9 items; emotional responsiveness, 5; speech, language and communication, 9; behaviour patterns, 7; sensory aspects, 6; cognitive component, 4.

Administration combines observation, caregiver interview and direct testing, takes 20 to 30 minutes per the manual with an average of 17.4 minutes in practice, and uses a kit of 24 testing materials.

Each item is rated on a 5-point scale anchored by frequency: rarely, up to 20 per cent of the time; sometimes, 21 per cent to 40 per cent; frequently, 41 per cent to 60 per cent; mostly, 61 per cent to 80 per cent; extreme frequency, 81 per cent to 100 per cent. Raw totals run 40 to 200.

RAW SCORE	CLINICAL BAND	CERTIFIED DISABILITY
Below 70	**No autism**	Not applicable
70	**Mild autism begins**	**40 per cent, the statutory benchmark**
71 to 88	Mild	50 per cent
89 to 105	Mild	60 per cent
106 to 123	Moderate band runs 107 to 153	70 per cent
124 to 140	Moderate	80 per cent
141 to 158	Severe above 153	90 per cent
Above 158	Severe	100 per cent

Table: the score bands and the certification ladder; the marked rows carry the discrimination candidates reverse.

TRAP Confusing the cut-off score with the disability percentage. A raw score of 70 is the diagnostic boundary, set by receiver operating characteristic analysis with area under the curve 0.931, and corresponds to 40 per cent disability, the statutory threshold for benefits, not to 70 per cent.

Psychometrics, honestly. Standardisation across 1124 participants aged 3 to 22 years gave Cronbach alpha 0.93, inter-rater reliability 0.62 to 0.81 and 3-month test-retest reliability 0.83.

- **Key number.** An independent accuracy study in children aged 2 to 9 years gave sensitivity 92.3 per cent, specificity 97.4 per cent, positive likelihood ratio 35.5, negative likelihood ratio 0.08, positive predictive value 85.7 per cent and negative predictive value 98.7 per cent.
- **Key number.** But agreement with the Childhood Autism Rating Scale severity ratings was low, Kappa 0.14, and construct validity was sub-optimal at correlation of 0.5 or less in 4 of 6 domains, reaching 0.8 to 0.89 only in the social and emotional domains.
- **Concept.** It is validated for binary classification and certification, not for staging severity, and needs caution in children aged 2 to 3 years where age-typical behaviour is penalised as deviant.

DSM-5, THE COUNTING RULE

Criterion A requires all 3 areas of social communication deficit: social-emotional reciprocity, nonverbal communicative behaviour, and developing, maintaining and understanding relationships. Criterion B requires at least 2 of 4 areas of restricted repetitive behaviour: stereotyped or repetitive movements, use of objects or speech; insistence on sameness and ritualised patterns; highly restricted fixated interests abnormal in intensity or focus; and hyper- or hyporeactivity to sensory input. Criterion C places onset in the early developmental period, D requires clinically significant impairment, E requires that intellectual disability or global developmental delay does not better explain it. Severity is rated separately for A and B across 3 levels: requiring support, requiring substantial support, requiring very substantial support. Specify accompanying intellectual and language impairment, association with a known medical or genetic condition, association with another disorder, and catatonia.

RULE **Sensory reactivity is criterion B.4, not a separate entity, and A is all three while B is two of four.** Social communication disorder is diagnosed only where Criterion B has not been met at any time, so any repetitive stereotypy, insistence on sameness, fixated interest or sensory atypicality rules it out. In isolated intellectual disability social reciprocity and nonverbal skills track mental age; in autism they are markedly depressed relative to general developmental capacity.

THE NATIONAL TRUST SCHEMES

Statutory welfare runs under the National Trust Act of 1999, administering 10 schemes. Disha provides early intervention and school readiness for children aged 0 to 10 years, 4 to 6 hours daily, batch size 20. Vikaas covers those above 10 years of age for 6 hours daily, batch size 30. Samarth is respite residential care, batch size 30. Gharaunda is lifelong group-home care for adults over 18 years, batch size 20. Niramaya is health insurance up to Rs 1,00,000 per person per year with no prior health check. Sahyogi trains care associates through primary courses of 3 months and advanced courses of 6 months. Sambhav places assistive-device demonstration centres in cities with populations over 5 million. Gyan Prabha supports education, Prerna assists marketing, Badhte Kadam runs community awareness.

TRAP Quoting drug doses for autism. Medication does not alter the core deficits and serves only as an adjunct for severe comorbid symptoms: atypical antipsychotics for irritability, aggression and self-injury; stimulants or atomoxetine for attention symptoms; serotonin reuptake inhibitors for anxiety, depressive or obsessive symptoms; anticonvulsants by seizure semiology. No milligram dose, and no weekly hour count for applied behaviour analysis, could be traced here.

HOLD THIS

40 items, 6 domains, raw total 40 to 200, cut-off 70, certifying 40 per cent disability. It diagnoses and certifies; it does not stage severity, at Kappa 0.14 against the Childhood Autism Rating Scale.

WHAT EARNS THE MARKS

- **Cut-off separated from percentage**: the diagnostic score and the certified disability are different numbers.
- **Psychometrics stated honestly**: strong classification, weak agreement on severity.
- **The counting rule**: all three social communication areas, two of four repetitive areas.
- **The welfare route named**: the statutory schemes, not the clinical diagnosis alone.

SEE ALSO 04 Catatonia: the signs, Bush Francis, the lorazepam challenge
16 Adult ADHD: assessment, ASRS, treatment in India 18 Tics and Tourette syndrome

18 · Tics and Tourette syndrome

NEURODEVELOPMENTAL · TOPIC 18

This boy has sudden repetitive movements. Define a tic and separate it from its near neighbours.

SHORT CASE, CHILD MOVEMENT STATION

THE SHAPE OF THE ANSWER

1 Define a tic	Premonitory urge, suppressibility, waxing and waning course.
2 The three disorders	Motor plus vocal, one kind only, or a short course.
3 Comorbidity	Most have another disorder; treat the disabling one.
4 Behavioural therapy	Awareness training, competing response, functional interventions.
5 The drug ladder	Alpha-2 agonists, then D2 agents, then refractory options.

Three features define a tic: a premonitory sensory urge, suppressibility at the cost of mounting tension, and a waxing and waning course.

- **Concept.** That triad does the differential work, because nothing else on the list has an urge the movement relieves.
- **Key number.** Severity classically peaks between ages 8 to 12 years, then declines or resolves in many during adolescence, with a subset carrying impairing tics into adult life.

1. Define a tic and separate it from its near neighbours.
2. Give the three tic disorders.
3. Which comorbidities, and which do you treat first?
4. When do you treat, and with what behavioural therapy?
5. Give the pharmacological ladder and the monitoring.
6. Does methylphenidate worsen tics?

MOVEMENT	SPEED AND RHYTHM	SUPPRESSIBLE	PREMONITORY URGE	DISTINGUISHING FEATURE
Tic	Rapid, non-rhythmic, paroxysmal	Yes, for variable intervals	**Present in most individuals**	Stereotyped, waxes and wanes, relieved by execution

MOVEMENT	SPEED AND RHYTHM	SUPPRESSIBLE	PREMONITORY URGE	DISTINGUISHING FEATURE
Chorea	Rapid, jerky, non-rhythmic, continuous	No	Absent	Dance-like, flows randomly between body regions
Myoclonus	Shock-like, brief	No	Absent	Cannot be voluntarily inhibited
Dystonia	Sustained or intermittent twisting	No	Absent	Abnormal posturing, action-induced twisting
Stereotypy	Rhythmic, repetitive, prolonged	Interrupted by call or touch	Absent	**Common in autism, soothing or neutral, no urge**
Compulsion	Purposeful ritual	Resisted with rising anxiety	**Preceded by an obsession, doubt or distress**	Performed to rigid rules or to avert harm

Table: the urge column separates the tic from everything else, and the compulsion is driven by cognitive anxiety rather than somatic tension.

The three disorders. Tourette syndrome requires multiple motor tics and one or more vocal tics at some time during the illness, though not concurrently. Persistent tic disorder has motor or vocal tics but not both.

Provisional tic disorder is a shorter course where the criteria for the other two have not been met. Each excludes a substance or other medical condition as cause.

- **Key number.** More than three-quarters of children with a tic disorder have another disorder, and up to 85 per cent of the Tourette syndrome population has a co-occurring neuropsychiatric condition.
- **Concept.** ADHD is commonest and appears earlier than the tics; tic-related obsessive-compulsive disorder emphasises symmetry, order, repeating and just-right perceptions.
- **Concept.** Direct management at the domain causing the greatest functional impairment, which is usually not the tics.

BEHAVIOURAL THERAPY FIRST

Education, family reassurance and watchful waiting are appropriate where tics are mild and non-impairing. Treat when tics cause functional or academic impairment, physical pain or musculoskeletal injury, or social stigmatisation and severe distress. Comprehensive Behavioral Intervention for Tics and habit reversal training are first-line, built on awareness training to detect the premonitory urge, competing response training with an incompatible voluntary contraction held until the urge dissipates, and functional interventions modifying the antecedents that exacerbate tics. In one randomised controlled trial habit reversal therapy produced a 97 per cent reduction in tic symptoms at 18-month follow-up, with 80 per cent of participants tic-free, and therapist-supported remote delivery matches in-person sessions.

LINE	AGENTS	MECHANISM	MONITORING
First	Guanfacine, clonidine	Presynaptic alpha-2 stimulation dampening noradrenergic release	**Baseline and periodic blood pressure and heart rate; taper slowly to avoid rebound hypertension; extended-release guanfacine covers 24 hours, extended-release clonidine 12 hours**
Second	Aripiprazole, risperidone	D2 partial agonism or antagonism	**Weight, body mass index, blood pressure, fasting glucose and lipids before, soon after starting, and at least once a year**
Third	Haloperidol, pimozide	Potent D2 antagonism	**High risk of dystonia, parkinsonism, akathisia and tardive dyskinesia; pimozide prolongs QTc, so baseline and annual electrocardiogram**
Refractory	Tetrabenazine, deutetrabenazine, valbenazine, ecopipam; botulinum toxin for focal tics	VMAT2 inhibition depleting presynaptic dopamine; ecopipam is a D1 antagonist studied in phase 3 paediatric trials	Specialist oversight for mood and parkinsonian effects; tetrabenazine risks depression, fatigue, parkinsonism

RULE **Methylphenidate does not systematically worsen tics and remains first-line for impairing ADHD.** The old avoidance rested on studies using excessive amphetamine doses. Alpha-2 agonists benefit both tics and ADHD; if a stimulant clearly exacerbates tics, lower the dose and combine with guanfacine or clonidine. For comorbid obsessive-compulsive disorder, exposure and response prevention with a serotonin reuptake inhibitor at higher doses is first-line, and augmenting with a second-generation antipsychotic helps approximately one-third of patients.

TRAP Quoting what this material cannot support: the duration and age-of-onset thresholds for the tic disorders, the structure and score ranges of the Yale Global Tic Severity Scale, the proportion with coprolalia, the milligram doses in the ladder above, or the hold time for a competing response. Coprolalia is neither required nor sufficient, because the criteria ask for vocal tics of any kind. Whether Tourette syndrome is a notified benchmark disability, and any certification percentage, is likewise untraced. Admission of a minor under the Act is governed by Section 87; Section 89 covers supported admission of adults with high support needs and is not the minors provision. Deep brain stimulation is reserved for intractable adult cases, targeting the centromedian nucleus, substantia periventricularis and nucleus ventro-oralis internus crosspoint, or the globus pallidus internus.

HOLD THIS

Urge, suppressibility, waxing and waning. Treat the domain that disables, usually the ADHD or the obsessive-compulsive disorder, and alpha-2 agonists cover both.

WHAT EARNS THE MARKS

- **The urge named first**: it separates a tic from chorea, myoclonus, dystonia and stereotypy.
- **The disabling domain treated**: usually the ADHD or the obsessive-compulsive symptoms, not the tics.
- **Behavioural therapy offered first**: awareness training and a competing response, ahead of any drug.
- **Methylphenidate defended**: it does not systematically worsen tics and stays first-line for impairing ADHD.

SEE ALSO 10 Functional neurological disorder: positive signs and the explanation

11 Two iatrogenic cases: tardive syndromes and lithium toxicity 16 Adult ADHD: assessment, ASRS, treatment in India

19 · Eating disorders and the SCOFF

NEURODEVELOPMENTAL · TOPIC 19

This underweight young woman complains of constipation and low mood. How do you screen her for an eating disorder?

SHORT CASE, GENERAL AND LIAISON STATION

THE SHAPE OF THE ANSWER

1 Ask the screen	Five questions, one point each, and the cut-off.
2 Physical compromise	Vital signs, skin, parotids, wasting, amenorrhoea.
3 Laboratory and cardiac	The acid-base picture and the electrocardiogram changes.
4 Purging or laxatives	Alkalosis against a normal anion-gap acidosis.
5 Refusal of nourishment	Capacity, supported admission, then emergency treatment.

Nobody arrives saying they have an eating disorder, so the screen has to be asked.

- **Concept.** Most present as normal-weight or underweight women with indirect complaints: constipation, bloating or fluid retention.
- **Concept.** Others come with unexplained weight loss despite a normal baseline weight, or with mood lability, anxiety, sleep disruption or impaired concentration.
- **Trap.** Reading obesity as a presenting sign. Most people who are overweight do not have a formal eating disorder.

1. What does SCOFF stand for, and how is it scored?
2. Which physical signs indicate compromise?
3. Which laboratory and cardiac changes matter?
4. Purging against laxative abuse.
5. What points to inpatient medical stabilisation?
6. Which statutory provisions apply to a patient refusing nourishment?

The screen. The questionnaire was developed by Morgan, Reid and Lacey in 1999 and published in the British Medical Journal as a brief 5-question screen for an eating disorder of any type,

analogous to the CAGE screen for alcohol.

- **Concept.** Sick asks about self-induced vomiting to relieve fullness, where the British original uses "sick" to mean vomiting. Control asks about lost control over how much is eaten.
- **Key number.** One asks about losing more than one stone, that is 14 pounds or 6.35 kg, in a 3-month period.
- **Concept.** Fat asks about believing oneself fat when others say one is too thin. Food asks whether food dominates life.

Each affirmative answer scores 1 point and each negative 0, for a total of 0 to 5, and a score of 2 or higher is the validated cut-off, which in the original validation gave a sensitivity of 100 per cent and a specificity of 87.5 per cent.

TRAP The stone-to-pounds error. One stone is a fixed unit, 14 pounds or 6.35 kg, and one widely circulated version of the item prints 15 pounds, which is an arithmetic error in that source. Quote 14 pounds and 6.35 kg and name the discrepancy if pressed. The second error is treating a positive screen as a diagnosis: it establishes suspicion and calls for a structured interview and an assessment of acute medical risk.

SYSTEM	EXAMINATION FINDING	MECHANISM
Vital signs	**Hypothermia with impaired temperature regulation, resting sinus bradycardia, arterial hypotension**	Downregulated metabolic rate and blunted sympathetic tone
Skin	Acrocyanosis, xerosis, diffuse alopecia, soft downy lanugo over trunk and limbs	Peripheral vasoconstriction, protein depletion, starvation hormonal change
Head and neck	**Bilateral painless parotid hypertrophy, that is sialadenosis**	Autonomic hyperstimulation from purging
Gastrointestinal	Delayed gastric emptying, postprandial fullness, stubborn constipation	Gastric hypomotility, smooth muscle atrophy, enteric neuropathy
Endocrine	Secondary amenorrhoea or oligomenorrhoea	Hypothalamic-pituitary-gonadal suppression from hypoleptinaemia and starvation
Musculoskeletal	Wasting of temporalis, deltoids and interossei; premature osteopenia and osteoporosis	Skeletal muscle proteolysis and uncoupled bone remodelling

Laboratory. Recurrent self-induced vomiting produces a hypokalaemic, hypochloraemic metabolic alkalosis: loss of gastric hydrochloric acid depletes hydrogen and chloride, driving renal bicarbonate retention and secondary aldosterone-mediated potassium excretion.

Serum amylase is raised, reflecting parotid stimulation and repetitive emesis, and mild to moderate normocytic normochromic anaemia can develop.

- **Concept.** The resting electrocardiogram shows sinus bradycardia, low QRS voltage, diffuse T-wave inversion and prolongation of the corrected QT interval.

Concept. Bone densitometry can show severe reduction in mineral density at an early age.

RULE **Vomiting alkalinises, laxatives acidify.** Self-induced vomiting gives hypochloraemic, hypokalaemic metabolic alkalosis; surreptitious laxative abuse loses bicarbonate in diarrhoea and gives a hyperchloraemic, normal anion-gap metabolic acidosis. The companion point is that bulimia nervosa commonly occurs at normal or fluctuating weight, presenting only with lethargy, irregular menses or parotid enlargement, so do not equate eating disorders with emaciation.

TRAP Reciting numeric admission cut-offs. The abnormalities that matter are secure: bradycardia, hypotension, low QRS voltage, T-wave inversion, prolonged corrected QT interval and hypokalaemic hypochloraemic alkalosis. But every numeric threshold usually quoted, namely heart rate, systolic pressure, postural drop, core temperature, body mass index, corrected QT interval, potassium, sodium and phosphate values, and the initial caloric rate for refeeding, could not be traced here, and neither could the name or tier structure of the risk-stratification framework examiners expect. Verify all of it against the primary guideline and quote no figure from memory. Refeeding syndrome itself is real, characterised by potentially fatal shifts in phosphate, potassium and magnesium driven by insulin surges on carbohydrate re-exposure, so reintroduce nutrition cautiously with thiamine cover.

INDIAN PRACTICE NOTE

Where starvation is life-threatening and the patient refuses nourishment the sequence is: assess capacity, because body image distortion and cognitive compromise from starvation often remove it for decisions about nutrition; if capacity is absent, supported admission on the nominated representative's application with medical certification, at least one certifier being a psychiatrist; then the emergency treatment provision to authorise immediate life-saving measures such as nasogastric feeding or intravenous electrolyte repletion. The capacity test is Section 4(1), supported admission is Section 89 for 30 days, and extended supported admission is Section 90. The emergency treatment provision and its maximum duration could not be traced here. Severe weight loss with secondary amenorrhoea in a young Indian woman should also be separated from abdominal tuberculosis, hyperthyroidism, coeliac disease, Addison disease and Sheehan syndrome.

HOLD THIS

Two affirmative answers out of 5 is a positive screen, not a diagnosis, and one stone is 14 pounds or 6.35 kg in 3 months. Vomiting gives alkalosis, laxatives give acidosis.

WHAT EARNS THE MARKS

- **The screen quoted correctly**: one stone as a fixed unit, and a positive result treated as suspicion.
- **The acid-base split**: vomiting gives alkalosis, laxative misuse gives a normal anion-gap acidosis.
- **Normal weight kept in view**: bulimia nervosa presents without emaciation.
- **The statutory sequence**: capacity, then supported admission, then emergency treatment.

SEE ALSO 06 Capacity and the supported-admission ladder

14 After deliberate self-harm: lethality, intent, safety planning 17 Autism: DSM-5, the ISAA, and disability certification

Quick review

The discriminators, in one table	
SHORT CASE SCHEMA	Syndrome, risk, organicity, disposition. Seven core mental functions; thought across stream, form and content. Prune out loud. One alteration of consciousness makes it a medical case.
CATATONIA	At least 3 of 12 features. Two signs on the 14-item screen or the 23-item scale. Volitional signs in items 15 to 23. Lorazepam 1 to 2 mg, reassess at 5, 15 or 30 minutes by route, positive at 50 per cent reduction, titrate to at least 16 mg per 24 hours.
THE RIGID SYNDROMES	Hypokinetic with hyporeflexia is NMS; clonus, mydriasis and loud bowels is serotonin syndrome; neuroleptic-naive after excited catatonia is malignant catatonia. Creatine kinase above 1000 U/L, but raised in 70 per cent of febrile patients on antipsychotics without it.
CAPACITY	Understand, appreciate, communicate, Section 4(1), presumed. Recently equals Section 89, 30 days, one psychiatrist plus one professional. Consistently over time equals Section 90, 2 psychiatrists. Review weekly then fortnightly.
EPILEPSY PSYCHOSIS	Lucid interval 8 to 72 hours defines postictal psychosis. Interictal after 10 to 15 years, warm affect, no negative deficit. Forced normalisation needs over 50 per cent spike reduction; treat by reducing the antiepileptic. Avoid clozapine.
MEDICAL MIMICS	Steroid psychiatry within 3 to 5 days of a dose change while lupus settles; lupus cerebritis with the flare, improving on more steroid. Risk above 40 mg prednisone daily. Porphyria: haloperidol and chlorpromazine safe, quetiapine and clonazepam unsafe.
HIV	Brain injury, not disorder. Static decline on suppression is legacy; new decline means cerebrospinal fluid escape, CD8 encephalitis or immune reconstitution. Valproate first-line, carbamazepine avoided, ritonavir raises risperidone and lowers olanzapine.
FUNCTIONAL DISORDER	Rule in on Hoover and entrainment, not on normal scans. Show the sign to the patient. Hardware intact, software failing. No anti-seizure drugs for functional seizures, no opioids.
TARDIVE AND LITHIUM	Do not raise the dose to mask movements; switch to clozapine and add a VMAT2 inhibitor. Anticholinergics worsen classic dyskinesia and help dystonia. Lithium trough at 12 hours; chronicity, not the number, grades severity.
ALCOHOL	Seizures 12 to 48 hours, delirium tremens around 96 hours. CIWA-Ar of 8 starts treatment, 15 triggers rescue. Glucuronidated benzodiazepine in liver failure. No phenytoin. Parenteral thiamine, and correct the magnesium.

The discriminators, in one table	
SELF-HARM	Planning predicts damage, lethal intent predicts hopelessness, item 8 loads on neither. No scale clears a patient. Safety plan with means restriction, not a no-suicide contract.
CANNABIS PSYCHOSIS	Visual hallucinations, preserved insight, few negative symptoms, heavy use. Conversion 40 per cent to 45 per cent, highest of all substances, in the genetically vulnerable. Urine proves exposure, not timing. Substance unknown means no benzodiazepine.
NEURODEVELOPMENTAL	ADHD: 5 symptoms, before age 12, 2 settings, 6 months; duplicate Schedule X, no teleconsultation. Autism: 40 items, cut-off 70, certifying 40 per cent. Tics: urge, suppressibility, fluctuation; treat the disabling comorbidity. SCOFF: 2 of 5, one stone is 14 pounds.

HOLD THIS

Every short case asks four questions in order: what is the syndrome, who is at risk, what is the body doing, and what happens in the next hour. Answer them in that order and the case answers itself.

PART IV

IV

The Neurology Short Case

The neurological examination in the order it is watched, the localisation ladder, and the cases a psychiatry examiner gives.

01 Examination in exam order

02 Localisation

03 The cases

04 Neurology viva questions

05 Quick review

The Neurology Short Case

Four sections · 24 topics · the practical neurology set

The neurology short case is scored on sequence. **Say where the lesion is before you say what it is.** Each topic runs in the order the hands move, then the ladder that turns findings into a level, then the cases a psychiatry examiner keeps on the ward.

Every topic is built the same way: the examiner's question, the shape of the answer, the body in short typed blocks, what earns the marks, then where to read next.

Grids carry the discriminator shaded, a Rule or Trap band holds the sentence that earns or loses the mark, and the hold box is the one line to take into the room. Where the library behind this note does not establish a figure, the trap says so and prints no number.

Examination in exam order

1 · Where before what: the localisation ladder

EXAMINATION · TOPIC 01

This patient has weakness of one arm and leg. Where is the lesion, and what makes you say so?

NEUROLOGY SHORT CASE, OPENING ANSWER

THE SHAPE OF THE ANSWER

1 What	Distribution of weakness, motor type, sensory involvement
2 Where	One rung from cortex to muscle, named aloud
3 Discriminator	The single sign that fixes that rung
4 Why	Vascular, inflammatory, neoplastic, infectious or degenerative

Three questions, in this order. Answering the third one first loses the case, because **localisation is what makes investigation targeted.**

- **Concept.** What: the distribution of weakness, the motor type, and whether sensation is involved.
- **Concept.** Where: one rung from cortex to muscle, given as a level rather than a guess.
- **Concept.** Why: vascular, inflammatory, neoplastic, infectious or degenerative, offered after the level.

RUNG	THE WEAKNESS	DISCRIMINATOR
Cortex	Disproportionate: face and arm, or leg alone	Higher cortical signs accompany it
Internal capsule	Dense, proportionate across face, arm and leg	Equal weakness with no cortical sign
Brainstem	Contralateral hemiparesis	Crossed signs: cranial one side, limbs the other
Spinal cord	Para or quadriparesis, face spared	A sensory level, and a normal jaw jerk

RUNG	THE WEAKNESS	DISCRIMINATOR
Root	Myotomal, with radicular pain	**One root feeds muscles in several named nerves**
Plexus	Multiple roots and nerves in one limb	**Too wide for a nerve, confined to one limb**
Nerve	Named territory, or glove and stocking	**Sensory boundary follows the nerve**
Junction	Fatigable, ocular and bulbar predilection	**Worsens on repetition, recovers on rest**
Muscle	Symmetrical, proximal more than distal	**Reflexes preserved out of proportion to weakness**

Two facts make a cord level defensible at the bedside.

- **Key number.** The cord is described as 8 cervical, 12 thoracic, 5 lumbar, 5 sacral and 2 coccygeal segments.
- **Concept.** It ends at the conus around the L1 or L2 vertebral level, and compression below that gives purely lower motor neuron deficits.
- **Key number.** Segmental reflexes pin the level: biceps C5 and C6, triceps predominantly C7, patellar L2 to L4, ankle S1.

TRAP Calling an acutely flaccid areflexic patient a lower motor neuron case. Acute cord transection and sudden cerebral insults begin flaccid, and spasticity appears over days to weeks. Look for an extensor plantar, which can appear early and settles it.

HOLD THIS

Weakness without sensory loss localises to the motor unit: anterior horn cell, junction or muscle. Sensory involvement lifts you higher, crossed signs mean brainstem, a trunk level means cord.

WHAT EARNS THE MARKS

- **Where before what**: the sequence is scored, not the length of the differential
- **A named rung**: cortex, capsule, brainstem, cord, root, plexus, nerve, junction or muscle
- **The discriminator said aloud**: crossed signs, a sensory level, fatigability, preserved reflexes
- **Aetiology last**: it shows the localisation drove the investigation you ask for

SEE ALSO 09 Upper versus lower motor neuron, and the mixed picture 10 Spinal cord syndromes 11 Brainstem syndromes and crossed signs

2 · Higher mental function and lobe testing

EXAMINATION · TOPIC 02

Test this patient's higher mental functions and tell me which lobe is affected.

SHORT CASE; OSCE COGNITIVE STATION

THE SHAPE OF THE ANSWER

1 Arousal and attention	Awake, alert, not delirious, before any focal claim
2 Frontal	Fluency, Luria sequence, go and no-go, proverbs
3 Temporal	Delayed recall, category fluency, autobiographical milestones
4 Parietal, occipital	Gerstmann items, neglect tests, agnosia with intact acuity
5 Name the lobe	One localisation, with the test that showed it

Attention before anything else. Cognitive testing rests on an awake alert state mediated by an intact ascending reticular activating system.

So separate delirium, with acute onset and fluctuating inattention, from dementia **before attributing any deficit to focal cortical disease**.

- **Concept.** Screen attention with letter cancellation, then digit span forward at one digit per second.
- **Key number.** A normal forward span is 5 to 7 digits, and Trail Making A connects numbers from 1 to 25.

Frontal battery. Trail Making B; the Luria fist-edge-palm series, three with you and three alone, normal at 6 or higher; go and no-go then conflicting instructions; proverbs and similarities; phonemic fluency on F, A and S for one minute each; then grasp and utilisation behaviour.

Temporal battery. Digit span backward, three to five words recalled after three to five minutes, autobiographical milestones, and category fluency, where below 13 items in a minute indicates temporal or semantic impairment.

LOBE	BEDSIDE TEST	DISCRIMINATOR
Frontal	Fluency, Luria sequence, go and no-go, proverbs	**Perseveration and environmental dependency**
Temporal	Word recall at three to five minutes, category fluency	**Recall fails while registration is intact**
Dominant parietal	Calculation, writing, finger naming, right-left commands	**Gerstmann tetrad**
Non-dominant parietal	Extinction, line bisection, cancellation, cube copy	**Neglect with anosognosia**
Occipital	Confrontation fields, object and face naming	**Agnosia with intact acuity**

The Frontal Assessment Battery is the composite to name: 6 subtests each scored 0 to 3 for a maximum of 18, with a cut-off of 12 carrying a reported sensitivity of 77% and specificity of 87% against Alzheimer type dementia.

TRAP Finger agnosia against finger anomia. In anomia the patient cannot name the finger you touch but points correctly to the one you name; in agnosia both directions fail. Test both or the sign is not established.

HOLD THIS

Test attention first and say so aloud. Dominant hemisphere equals language, calculation and praxis; non-dominant equals space, attention and body schema.

Change the cue. Observe the action.

Three ways to examine a learned gesture; no proprietary test form.

1 REQUEST

Pantomime using a comb

2 IMITATE

Copy the demonstrated action

3 OBJECT

Use the actual comb

Check elemental function before interpreting an action error.

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Examine a learned action on verbal request, by imitation and with an actual familiar object. The comb diagrams represent a novel demonstration of task context, not a validated test item. Before attributing errors to apraxia, assess comprehension, power, sensation and coordination. Improvement with a different cue is informative but not obligatory.

Record the type of error rather than assuming non-cooperation.

Schematic prepared by Luna for Weave Viva, based on P3-33. Original vector diagram.

WHAT EARNS THE MARKS

- **Attention tested and declared first**: a focal claim on an inattentive patient is unscorable
- **One named test per lobe**: the examiner marks the test, not the adjective
- **Both directions of a sign**: finger agnosia needs naming and pointing tested
- **The composite quoted properly**: subtest count, maximum score, cut-off, then its accuracy

SEE ALSO 03 Speech and language 12 Hemispheric syndromes 20 Dementia with focal signs, frontal syndrome

3 · Speech and language

EXAMINATION · TOPIC 03

This patient's speech is abnormal. Is this dysarthria or dysphasia, and where is the lesion?

SHORT CASE; VIVA TABLE ON APHASIA

THE SHAPE OF THE ANSWER

1 Handedness	Written down first; it sets the dominant hemisphere
2 Separate the three	Dysarthria, dysphasia, apraxia of speech
3 Fluency, comprehension	Places the lesion front or back
4 Repetition	Separates perisylvian from transcortical syndromes
5 Name it	Syndrome plus lesion site, in one sentence

Three words, three lesions. Say which of the three you are describing before you localise it.

- **Concept.** Dysarthria is a motor disorder of speech with semantics and syntax preserved.
- **Concept.** Dysphasia is impaired language processing from dominant hemisphere damage.
- **Concept.** Apraxia of speech is impaired planning of volitional oral motor programs, with no weakness and no linguistic breakdown.
- **Concept.** Pure dysarthria is confirmed when comprehension, reading, grammatical writing and gesture are preserved.

Record handedness first. Language sits in the left hemisphere in about 99% of right-handers, and the two source figures for left-handers are quoted as separate pairs rather than blended.

Those pairs are left dominance in about 66.7%, with the right carrying much of language in the other 33.3%; or right dominance in only 30%. The examination has six components and takes fewer than 10 minutes, against about 30 minutes for a full battery.

SYNDROME	FLUENCY	COMPREHENSION	REPETITION	LESION
Broca	Non-fluent	Good	**Impaired**	Left frontal or frontoparietal, including Broca area
Transcortical motor	Non-fluent	Good	**Normal**	Left frontal, excluding Broca and Wernicke areas
Global	Non-fluent	Poor	**Impaired**	Large left frontotemporoparietal

SYNDROME	FLUENCY	COMPREHENSION	REPETITION	LESION
Mixed transcortical	Non-fluent	Poor	**Normal**	Watershed lesions isolating perisylvian cortex
Wernicke	Fluent	Poor	**Impaired**	Left temporoparietal, including Wernicke area
Transcortical sensory	Fluent	Poor	**Normal**	Left temporoparietal, excluding Wernicke area
Conduction	Fluent	Good	**Disproportionately impaired**	Subcortical, under superior temporal or inferior parietal
Anomic	Fluent	Good	**Normal**	Small lesion anywhere in the left language network

RULE **Repetition separates perisylvian from transcortical.** Fluency and comprehension place the lesion front or back; repetition says whether the core arc is in it, and that one test turns four syndromes into eight.

DYSARTHRIA	SOUND	LEVEL
Spastic	Harsh, strained-strangled, slow	**Bilateral upper motor neuron, pseudobulbar**
Flaccid	Hypernasal, breathy, nasal escape	**Lower motor neuron, brainstem nuclei or nerves**
Ataxic	Scanning, staccato, fluctuating loudness	**Cerebellar**
Hypokinetic	Monotone, quiet, rushed, palilalia	**Basal ganglia, nigrostriatal**
Hyperkinetic	Unpredictable arrests, voice tremor	**Basal ganglia, caudate and putamen**

HOLD THIS

Fluency, comprehension, repetition, with handedness written down first. Hand a mute patient a pen: grammatical writing makes it dysarthria, not aphasia.

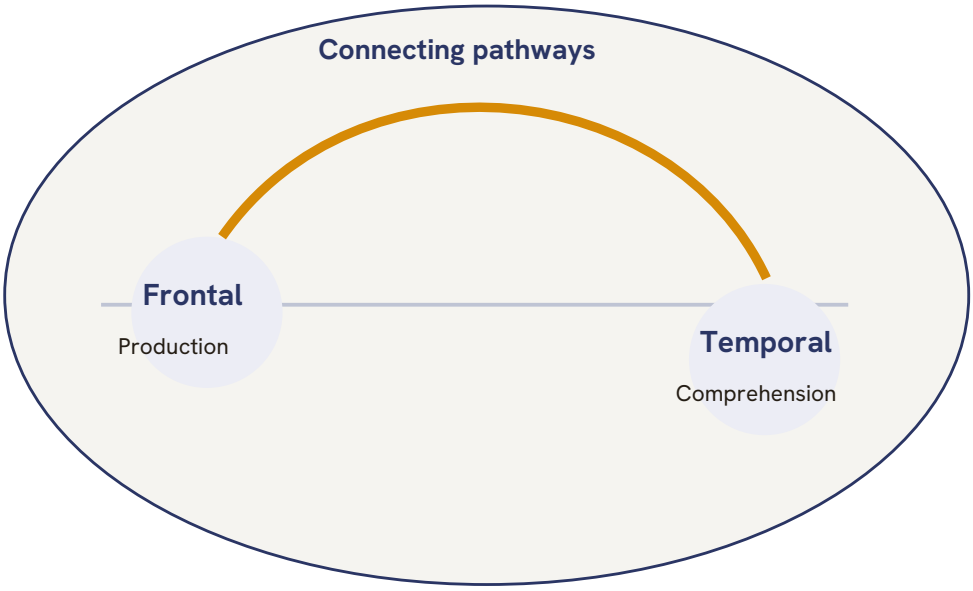
WHAT EARNS THE MARKS

- **Handedness recorded and justified**: it decides which syndrome is even available
- **The triad in order**: fluency, comprehension, repetition, each demonstrated
- **Repetition used as the hinge**: it turns four syndromes into eight
- **The pen test**: grammatical writing in a mute patient rules out aphasia

SEE ALSO 02 Higher mental function and lobe testing 04 Cranial nerves in exam order 12 Hemispheric syndromes

Test the network, not one label.

A classical left-hemisphere teaching model, deliberately simplified.



FLUENCY **COMPREHENSION** **REPETITION**

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. The classical language model links posterior temporal processing, frontal speech production and connecting pathways. Bedside assessment separates fluency, comprehension and repetition; preserved repetition helps distinguish transcortical patterns. Modern language function is distributed, so this schematic is not a literal one-area/one-function lesion map.

Describe fluency, comprehension and repetition before classifying aphasia.

Schematic prepared by Luna for Weave Viva, based on P3-32. Original vector diagram.

4 · Cranial nerves in exam order

EXAMINATION · TOPIC 04

Examine this patient's cranial nerves and localise the deficit you find.

SHORT CASE; OSCE CRANIAL NERVE STATION

THE SHAPE OF THE ANSWER

1 **Set out and inspect**	Tray ready, then speech, face, lids, pupils, alignment
2 **Run the order**	Olfactory through twelfth, without doubling back
3 **Isolate the palsy**	Primary position, then the discriminating manoeuvre
4 **Localise**	Nucleus, nerve or junction, and say which one
5 **Two decisive tests**	Forehead for the face, consensual response for the pupil

Set the tray out first. Pen torch, Snellen chart, Ishihara plates, ophthalmoscope, cotton wool, neuro-tip, a 512 Hz tuning fork and a glass of water.

Inspect before you test. Speech quality for the ninth and tenth nerves, facial asymmetry at rest for the seventh, ptosis for the third nerve or a sympathetic lesion or the junction, then pupil size, ocular alignment, and the aids by the bed.

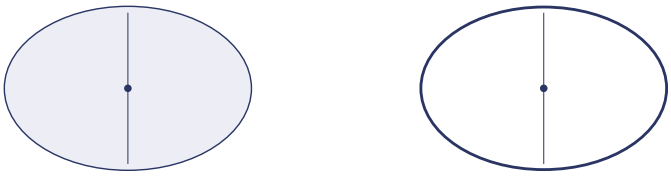
1. Olfactory: ask about change in smell, then separate scents to each nostril.
2. Optic: pupils; acuity at 6 metres with glasses worn, reducing distance then counting fingers, hand movements and light perception; direct, consensual, swinging light and accommodation; colour and inattention; fields and blind spot; fundoscopy.
3. Third, fourth and sixth: lids and primary position, an H pattern with a target at 30 cm, then Hirschberg and cover-uncover.
4. Trigeminal: sensation over three divisions after a sternal baseline, masseter bulk and clench, jaw opening against resistance, corneal reflex and jaw jerk.
5. Facial: taste and hyperacusis, the face at rest, then eyebrows, forced eye closure, cheek puffing, teeth and whistling.
6. Vestibulocochlear: whisper test, Rinne and Weber with the 512 Hz fork, Unterberger and head thrust.
7. Ninth and tenth: palate at rest and on phonation, cough quality, a swallow of 3 teaspoons of water.
8. Eleventh and twelfth: trapezius shrug and sternocleidomastoid against resistance; tongue at rest for wasting and fasciculation, then protrusion and power against each cheek.

In a relative afferent defect both pupils dilate relatively as the swinging light reaches the affected eye, and the separation from an efferent third nerve palsy is the consensual response, because in an afferent defect the affected pupil still constricts to light in the healthy eye.

Name the missing visual space.

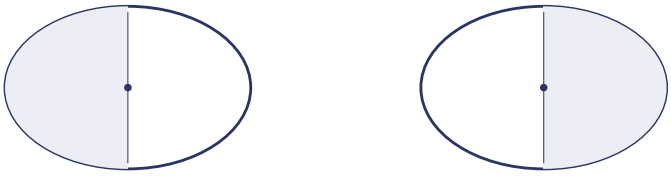
Patient's left and right visual fields; shading means loss.

Left monocular loss



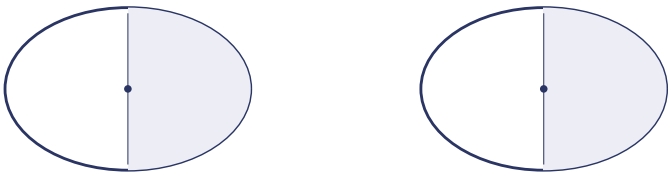
L eye R eye

Bitemporal loss



L eye R eye

Right homonymous loss



L eye R eye

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Idealised field patterns: monocular loss, bitemporal loss and homonymous loss. Each pair represents the left and right eye, not brain hemispheres. Monocular loss suggests a prechiasmal problem; bitemporal loss points to the chiasm; homonymous loss points behind the chiasm on the opposite side. Real defects need formal examination and may be incomplete.

Identify whether the pattern respects the vertical meridian.

Schematic prepared by Luna for Weave Viva, based on P3-03. Original vector diagram.

PALSY	PRIMARY POSITION	DISCRIMINATOR
Third	Down and out, marked ptosis, mydriasis	**Ptosis plus dilated pupil plus down and out**
Fourth	Extorsion and slight elevation	**Head tilt away from the lesion with chin tuck**
Sixth	Convergent squint	**Isolated failure of abduction**

HEARING	RINNE, AFFECTED EAR	WEBER
Normal	Air greater than bone bilaterally	**Midline**
Conductive loss	Bone greater than air, Rinne negative	**Lateralises to the affected ear**
Sensorineural loss	Air greater than bone, both reduced	**Lateralises to the normal ear**

TRAP Testing facial sensation at the angle of the mandible, which is supplied by the greater auricular nerve from C2 and C3 and not the mandibular division. And the right sternocleidomastoid turns the head to the left, so resist at the left jaw when testing it.

HOLD THIS

A brisk jaw jerk means a bilateral upper motor neuron lesion above the mid-pons, and both limbs of that reflex run in V3. Forehead movement decides the facial palsy; the consensual response decides the pupil.

WHAT EARNS THE MARKS

- **Inspection before instruments**: speech, lids, pupils and alignment are free findings
- **The order kept**: doubling back reads as an unrehearsed examination
- **Forehead movement**: it separates upper from lower motor neuron facial palsy
- **The consensual response**: it separates an afferent defect from a third nerve palsy

SEE ALSO 03 Speech and language 08 Cerebellum, gait and the fundus 21 Spot signs: face, pupils, praxis, skin

5 · Motor system: bulk, tone, power, coordination

EXAMINATION · TOPIC 05

Examine the motor system in this patient's limbs and tell me what you find.

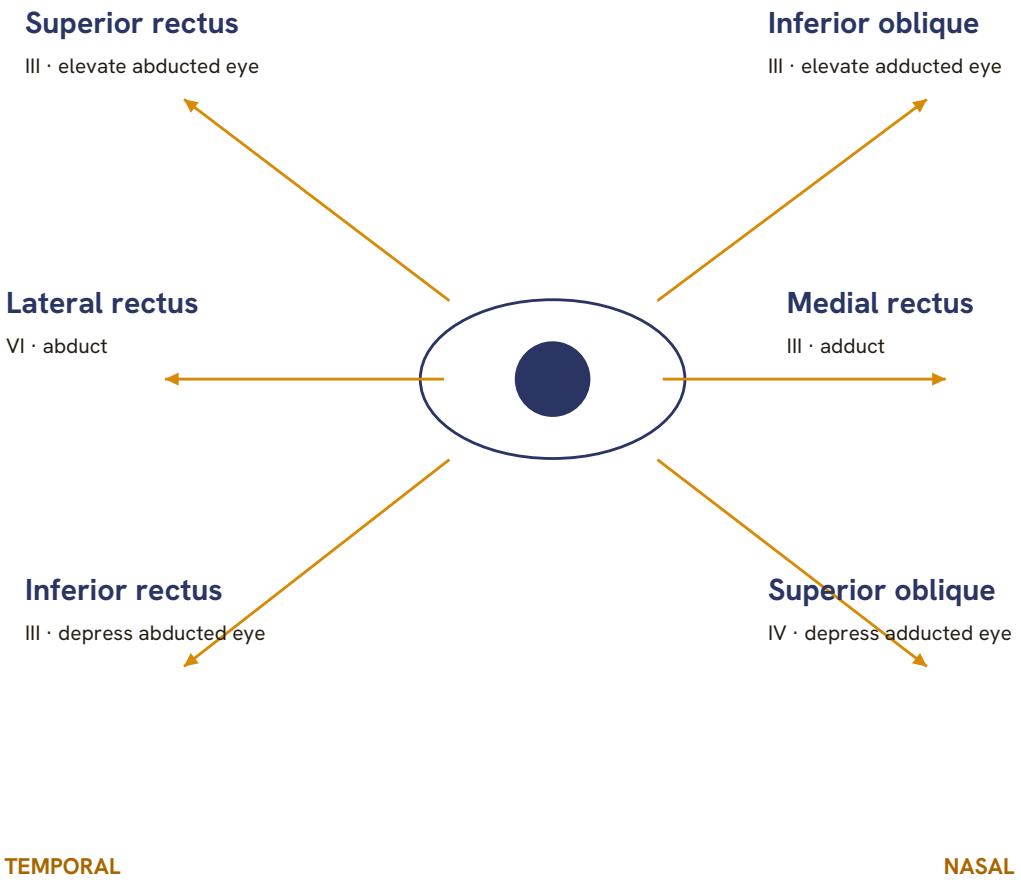
SHORT CASE; OSCE MOTOR STATION

THE SHAPE OF THE ANSWER

1 **Inspect**	Bulk, fasciculation, posture, abnormal movements
2 **Tone**	Velocity-dependent or not, and in which muscle groups
3 **Power**	Graded against the scale you name, group by group
4 **Coordination**	Tested only once power is grade 4 or 5
5 **Synthesise**	Upper or lower motor neuron, then the level

Follow the eye through six positions.

One RIGHT eye, viewed face-on: temporal is left on this page.



Schematic ▪ Original teaching diagram ▪ September 2026

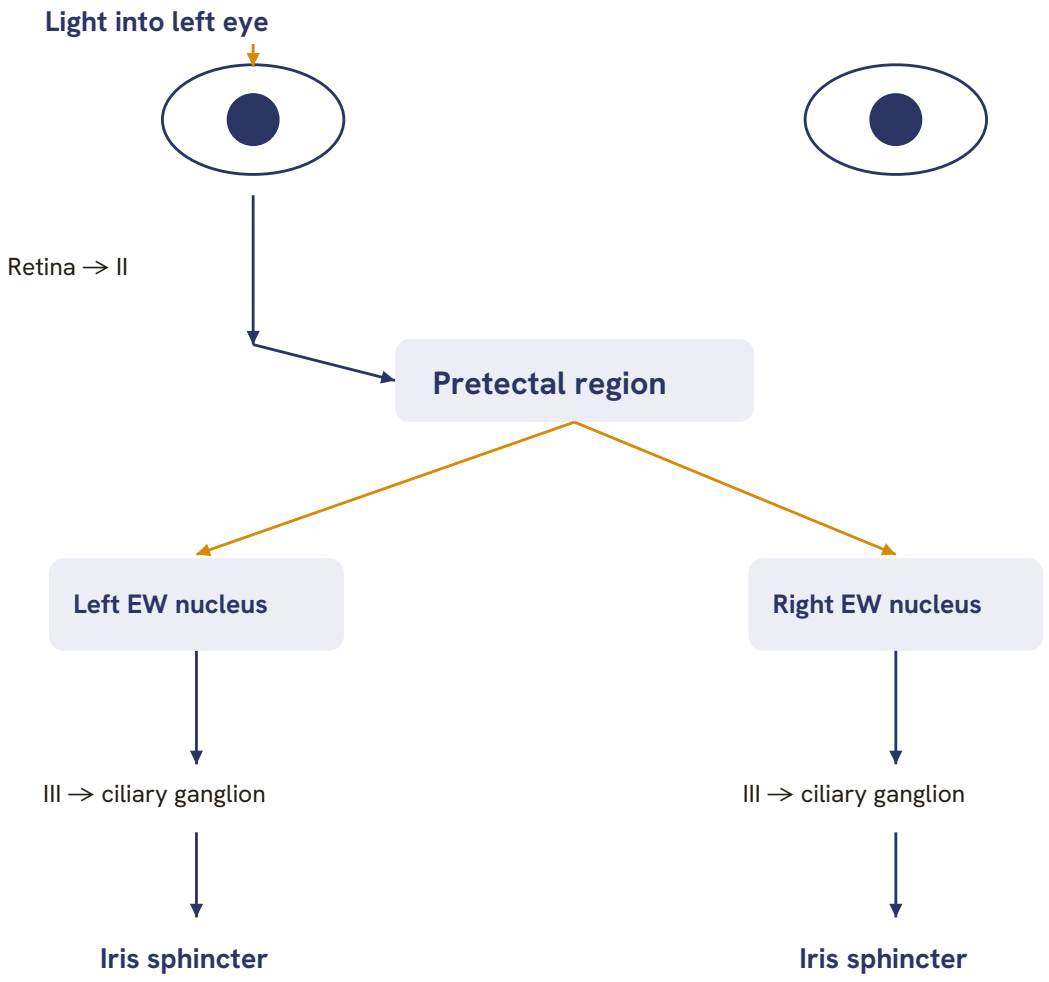
Schematic. Six cardinal gaze positions for the right eye. In abduction, elevation and depression emphasise superior and inferior rectus; in adduction they emphasise inferior and superior oblique. Lateral rectus is supplied by VI, superior oblique by IV and the others by III. The diagram is a muscle-action guide, not an eye photograph or an isolated-muscle test.

State the gaze position before identifying the weak action.

Schematic prepared by Luna for Weave Viva, based on P3-03. Original vector diagram.

One light. Two pupils.

Trace afferent input, then bilateral parasympathetic output.



Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Light input travels from the retina through the optic nerve to the pretectal region, which projects bilaterally to Edinger–Westphal nuclei. Each efferent limb travels through III and the ciliary ganglion to its iris sphincter. The bilateral connection explains direct and consensual constriction. This simplified pathway omits intermediate anatomy and is not a lesion-confirmation test.

Separate an afferent defect from an efferent defect.

Schematic prepared by Luna for Weave Viva, based on P3-31. Original vector diagram.

Inspection earns marks before your hands move. Inspect bulk at the shoulder girdle, thenar and hypothenar eminences, first dorsal interossei and quadriceps. Wasting means a lower motor neuron lesion or disuse, and fasciculations mean lower motor neuron pathology.

In a psychiatric case the same inspection is a **movement-disorder screen**, so name what you are looking at.

- **Concept.** Tardive dyskinesia: stereotyped tongue protrusion and lip smacking after neuroleptic exposure.
- **Concept.** Chorea: irregular movement flowing between muscle groups.
- **Concept.** Myoclonus: shock-like twitches. Dystonia: sustained patterned posturing.
- **Concept.** Pseudoathetosis: writhing fingers from proprioceptive deafferentation.

Pronator drift. Arms out with palms up for 20 to 30 seconds, then repeated with eyes closed. Pronation with downward drift means a contralateral corticospinal lesion, because pyramidal paresis weakens the supinators disproportionately.

Tone. With the limb supported, circumduct shoulder and wrist, flex the elbow, roll each leg and lift the knee briskly, where the heel leaves the bed in hypertonia. Then ankle clonus: dorsiflex and evert briskly and hold, where more than 5 beats is pathological.

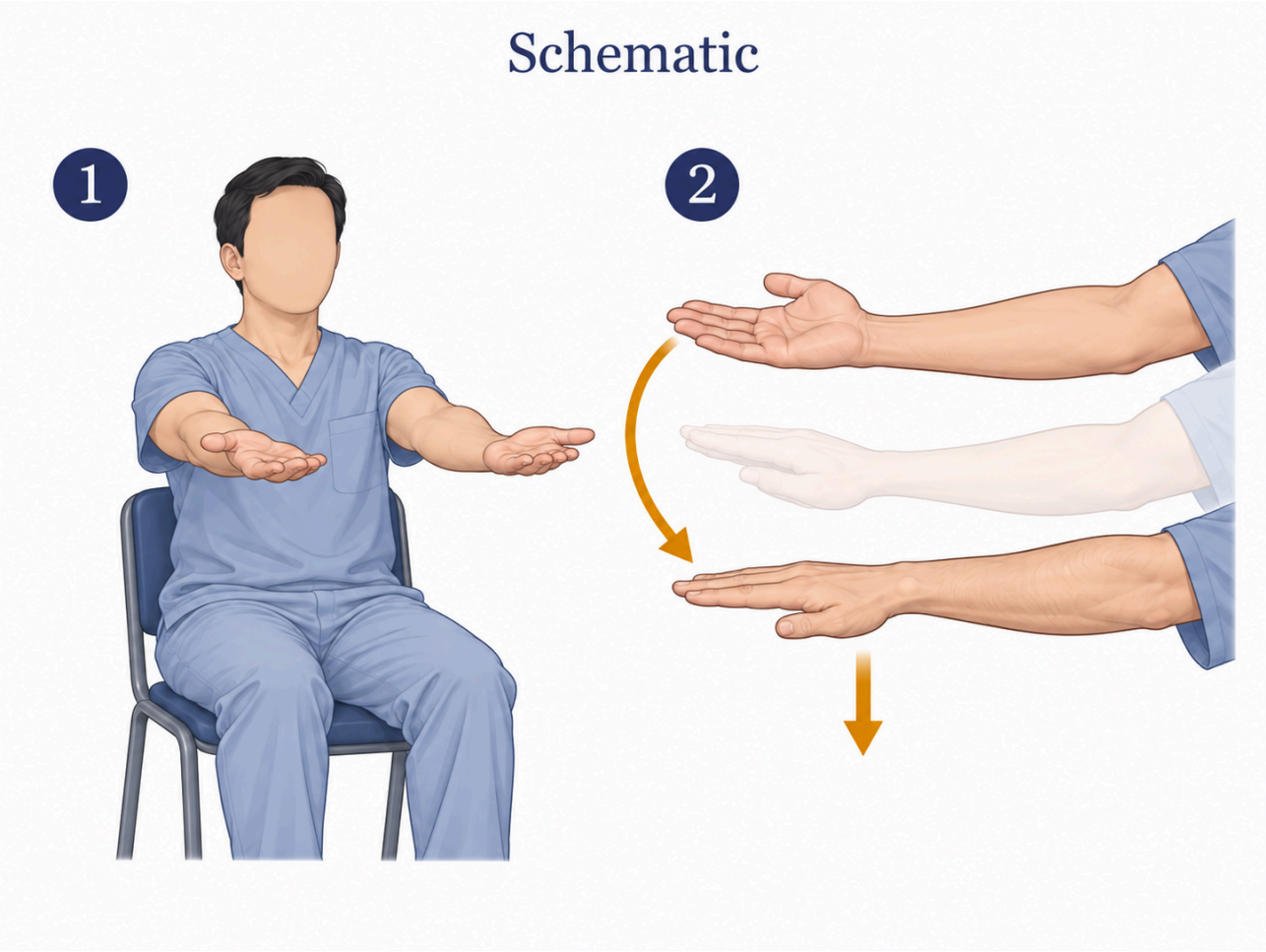
FEATURE	SPASTICITY	RIGIDITY	PARATONIA
Substrate	Pyramidal	Basal ganglia	Frontal, diffuse cortical
Velocity	**Dependent: rises with faster stretch**	**Independent: constant at any speed**	**Inconsistent: varies with force applied**
Direction	Arm flexors, leg extensors	Equal across flexors and extensors	Variable in all directions
Pattern	Clasp-knife	Lead-pipe or cogwheel	Eases on distraction
Company	Hyperreflexia, clonus, extensor plantars	Rest tremor, bradykinesia, hypomimia	Cognitive impairment, release signs

GRADE	MEDICAL RESEARCH COUNCIL DEFINITION
0 to 2	No contraction; flicker; active movement with gravity eliminated
3	**Against gravity, collapsing against added resistance**
4	**Against gravity and resistance, subdivided mild, moderate or severe**
5	Normal power

- RULE **Grade 4 is the wide grade and examiners ask how you subdivide it.** The expansion to give is mild, moderate or severe. Confirm power is grade 4 or 5 before calling limb clumsiness cerebellar, because upper motor neuron weakness mimics dysmetria.

Pronator drift: position and direction

Companion to original page 223



Schematic. 1: seated starting position with arms extended and palms up. 2: inset illustrating pronation and downward drift. Compare both arms during the manoeuvre; the right-hand inset is a movement illustration, not another patient. The still cannot demonstrate the observation period or establish a lesion.

Explain the manoeuvre and observe both arms.

Schematic prepared by Luna for Weave Viva, based on P3-04. AI-generated educational illustration.

HOLD THIS

Spasticity is velocity-dependent and clasp-knife; rigidity is velocity-independent and lead-pipe or cogwheel; paratonia rises with your own force. Clonus beyond 5 beats and an extensor plantar make the upper motor neuron case.

WHAT EARNS THE MARKS

- **Inspection reported first**: wasting and fasciculation change the answer before any manoeuvre
- **Tone tested at two speeds**: it is the only way to separate spasticity from rigidity
- **Grade 4 subdivided**: mild, moderate or severe, offered without being asked
- **Power before coordination**: pyramidal weakness mimics dysmetria

SEE ALSO 07 Reflexes, clonus, plantars and the release signs

09 Upper versus lower motor neuron, and the mixed picture 14 Parkinson's disease, and the parkinsonism it is not

6 · Sensory system: modalities, dermatomes, patterns

EXAMINATION · TOPIC 06

Examine the sensory system and give me the pattern of loss.

SHORT CASE; VIVA TABLE ON SENSORY PATTERNS

THE SHAPE OF THE ANSWER

1 Hypothesis first	Test to confirm a pattern, rather than to survey
2 Two systems	Spinothalamic against dorsal column, tested separately
3 Map the boundary	Dermatome, named nerve, trunk level or stocking
4 Dissociation	The finding that localises rather than describes
5 State the level	Highest dermatome involved, with its segmental reflex

Sensory testing is a hypothesis test, not a survey. Two systems, two routes, and the route is what localises.

- **Concept.** Pinprick and temperature run in the spinothalamic tract, whose fibres cross the anterior white commissure within a few segments.
- **Concept.** The lowest sacral fibres are laminated most superficially, so extrinsic compression hits sacral segments early and central lesions spare them.
- **Concept.** Vibration, joint position and discriminative touch run uncrossed in the dorsal columns.

LEVEL	KEY SENSORY POINT	MOTOR AND REFLEX CORRELATE
C6	Dorsal thumb	**Wrist extensors; brachioradialis reflex**
C7	Dorsal middle finger	**Elbow extensors; triceps reflex**
L3	Medial knee	**Knee extensors; patellar reflex**
L4	Medial malleolus	Ankle dorsiflexors
L5	Dorsum of the foot, first webspace	**Great toe extensors; no easily elicited reflex**
S1	Lateral calcaneus, the lateral heel	**Plantar flexors; Achilles reflex**
S4 to S5	Perianal	Voluntary anal sphincter; anal reflex

TRAP The lateral epicondyle for C5 and the medial epicondyle for T1 appear in abbreviated lists but are not the points on the worksheet most Indian examiners mark against, and the correct wording for those two levels is not established in the library behind this note. For S1 the discrepancy is resolved: the point is the lateral heel, not the lateral malleolus.

Dissociated loss is the pattern that wins short cases, so look for it deliberately.

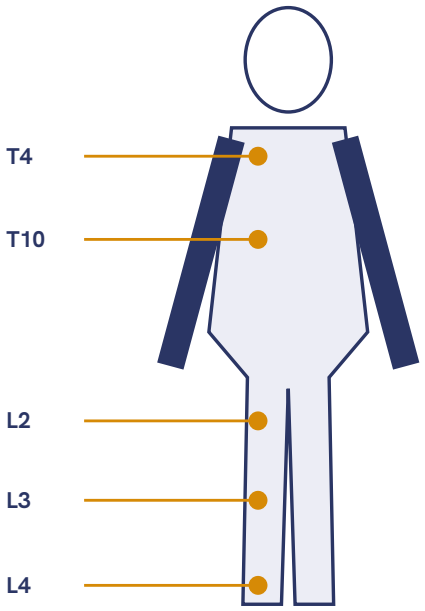
The syringomyelic form destroys the decussating spinothalamic fibres, abolishing pain and temperature bilaterally in a suspended cape across shoulders, arms and upper trunk, while the dorsal columns keep vibration and position. That explains painless burns, wasted small hand muscles and Charcot joints.

- **Concept.** Anterior spinal artery ischaemia spares the posterior columns.
- **Concept.** Lateral medullary infarction gives ipsilateral facial with contralateral body thermoalgesic loss.
- **Trap.** A craniocervical lesion compressing the lemniscal decussation reverses the picture, and lost vibration over the clavicles is the giveaway.

FEATURE	LENGTH-DEPENDENT POLYNEUROPATHY	SENSORY GANGLIOPATHY	FUNCTIONAL LOSS
Distribution	Symmetric, legs before arms	**Asymmetric, patchy, not length-dependent**	**Abrupt cuff at wrist or ankle crease**
Transition	**Broad gradient to normal**	Patchy borders	Abrupt, along an inked line
Modalities	All impaired distally	Position and vibration severe, pain spared	Discrepant: analgesia with intact touch
Reflexes	Ankle jerks lost early	Widespread areflexia	Normal or brisk, symmetric
Causes	Diabetes, alcohol, toxins, uraemia	Sjogren, paraneoplastic, pyridoxine excess	Functional neurological disorder

Sample roots at recognisable landmarks.

Selected sensory points, not a complete dermatome atlas.



C5	Lateral elbow
C6	Thumb
C7	Middle finger
C8	Little finger
T1	Medial elbow
T4	Nipple level
T10	Umbilicus
L2	Anterior thigh
L3	Medial knee
L4	Medial ankle
L5	Dorsum of foot
S1	Lateral foot

Detailed hand landmarks are listed at right.

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Selected dermatome landmarks organise a bedside screen: C5 lateral elbow, C6 thumb, C7 middle finger, C8 little finger, T1 medial elbow, T4 nipple level, T10 umbilicus, L2 anterior thigh, L3 medial knee, L4 medial ankle, L5 dorsum of foot and S1 lateral foot. Dermatomes overlap and vary; test and map the actual sensory border rather than treating these points as exact boundaries.

Relate a sensory point to a root, then compare it with a peripheral nerve territory.

Schematic prepared by Luna for Weave Viva, based on P3-05. Original vector diagram.

HOLD THIS

Dissociated loss is the sentence that localises. Suspended cape sparing the legs is syringomyelia; bilateral pain and temperature loss with intact vibration is anterior cord; bilateral vibration and position loss with intact pain is posterior column.

WHAT EARNS THE MARKS

- **A pattern, not a map**: name the pattern you were testing for and why
- **The level with its reflex**: a sensory level is only as good as its segmental check
- **Sacral sparing read correctly**: it separates central from extrinsic compression
- **Functional loss discriminated**: an abrupt crease-line boundary with intact touch

SEE ALSO 01 Where before what: the localisation ladder 10 Spinal cord syndromes 11 Brainstem syndromes and crossed signs

7 · Reflexes, clonus, plantars and the release signs

EXAMINATION · TOPIC 07

Elicit the reflexes and tell me what they add to your localisation.

SHORT CASE; OSCE REFLEX STATION

THE SHAPE OF THE ANSWER

1 Technique	Position, relaxation, reinforcement, before calling one absent
2 Grade	Name the scale, then grade each reflex on it
3 Plantar	Lateral sole only, then report it in accepted words
4 Superficial reflexes	The ones that add a segmental level
5 Read together	Reflexes with the plantar decide the motor neuron

A reflex is not absent until you have reinforced it. The arc is monosynaptic, with large myelinated group Ia afferents synapsing directly on alpha motor neurons, which is why reflexes are preserved in small-fibre neuropathy.

Grading has no single accepted system, so **state which scale you are using** before you quote a number.

- Key number. The five-point scale: 0 absent despite reinforcement, 1 elicited only with reinforcement, 2 normal, 3 brisk, 4 with clonus.
- Key number. The six-point bedside scale grades non-sustained clonus 4+ and sustained clonus 5+.

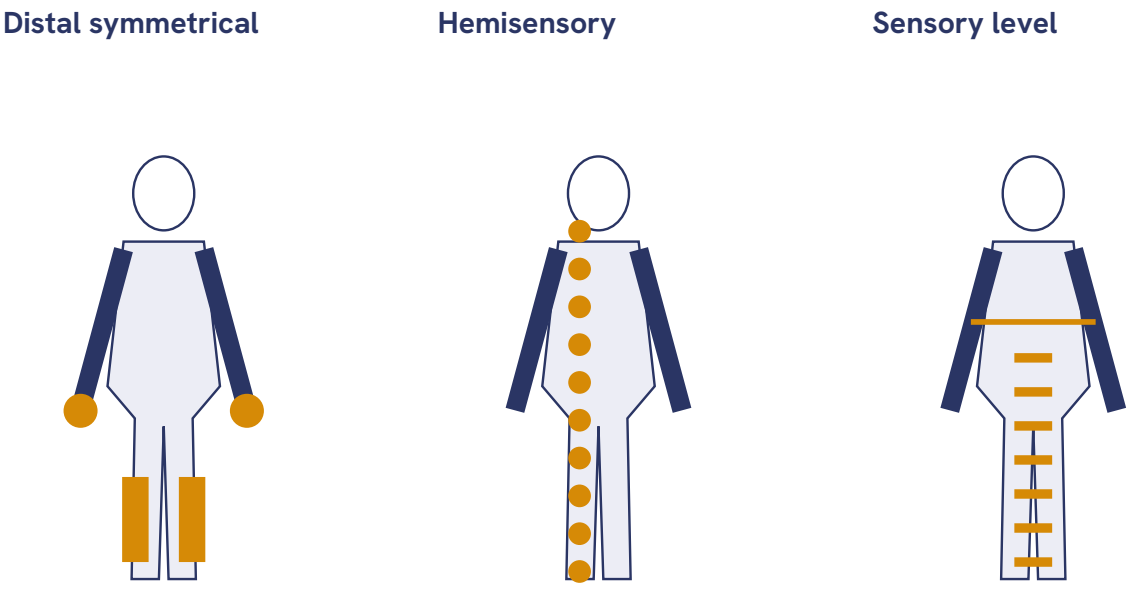
Reinforcement. Jendrassik described in 1885 hooking the flexed fingers together and pulling them apart while the tendon is struck, and facilitation lasts as long as the traction continues, up to 10 seconds.

In normal elderly patients an apparently absent ankle jerk is elicited in 70% of instances. For the arms, ask the patient to clench the jaw and look away.

TRAP Documenting a "negative Babinski". The sign is abnormal: it is present or absent. Acceptable wordings are "plantar reflex is flexor" or "toes down-going", "absent", or "Babinski sign present".

Map the shape before naming the cause.

Idealised distributions, deliberately separated from diagnostic labels.



1 / Compare distal and proximal regions.

2 / Test the same modality on both sides.

3 / Delineate a reproducible border.

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Conceptual sensory patterns: distal symmetrical stocking–glove loss, a hemisensory distribution and a sensory level across the trunk. Shading marks regions to map, not a validated patient finding. These shapes guide localisation; actual borders, modalities, onset and associated signs must be examined.

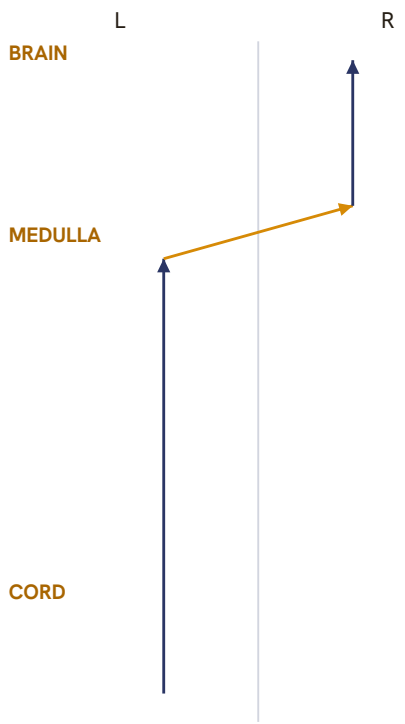
Describe the distribution and modality before proposing the lesion.

Schematic prepared by Luna for Weave Viva, based on P3-05. Original vector diagram.

Ask where the pathway crosses.

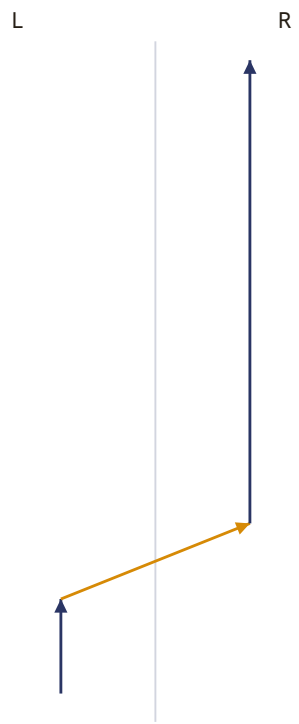
Body pathways only: face pathways are not shown.

Position / vibration



Crosses in medulla

Pain / temperature



Crosses near cord entry

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Simplified ascending body sensory pathways. Dorsal-column fibres ascend ipsilaterally and cross in the medulla before continuing in the medial lemniscus. Pain and temperature fibres entering the cord cross near their entry level and ascend contralaterally in the anterolateral system. Segmental spread is omitted. This crossing difference helps explain dissociated sensory loss.

Use the crossing level to predict the side of a sensory deficit.

Schematic prepared by Luna for Weave Viva, based on P3-05. Original vector diagram.

REFLEX	PATHWAY	WHY IT MATTERS
Corneal	**Afferent V1, efferent VII**	Depressed in cerebellopontine angle lesions and coma
Abdominal	Lower thoracic segments	**Asymmetry marks an upper motor neuron lesion on the absent side**
Cremasteric	**Genitofemoral and ilioinguinal, L1 and L2**	Absent in torsion, L1 or L2 injury, ilioinguinal surgery
Anal wink	**Afferent pudendal, efferent S2, S3, S4**	Conus and lower sacral integrity in trauma or cauda equina
Bulbocavernosus	**S2, S3, S4**	Monitors resolution of spinal shock

HOLD THIS

Grade 0 means absent after reinforcement, and the reinforcement works in the cord, not in the spindle. Stroke the lateral sole only, and report the plantar as flexor, absent, or Babinski present.

WHAT EARNS THE MARKS

- **Reinforcement demonstrated**: it is what makes an absent reflex a finding
- **The scale declared**: a grade without its scale is not a grade
- **The plantar worded correctly**: flexor, absent, or Babinski present
- **Superficial reflexes used**: abdominal asymmetry and the sacral reflexes add a level

SEE ALSO 05 Motor system: bulk, tone, power, coordination 09 Upper versus lower motor neuron, and the mixed picture 16 The wasted limb: motor neuron disease and neuropathy

8 · Cerebellum, gait and the fundus

EXAMINATION · TOPIC 08

Examine the cerebellar system, then walk this patient, then look at the fundus.

SHORT CASE; OSCE GAIT AND FUNDUS STATION

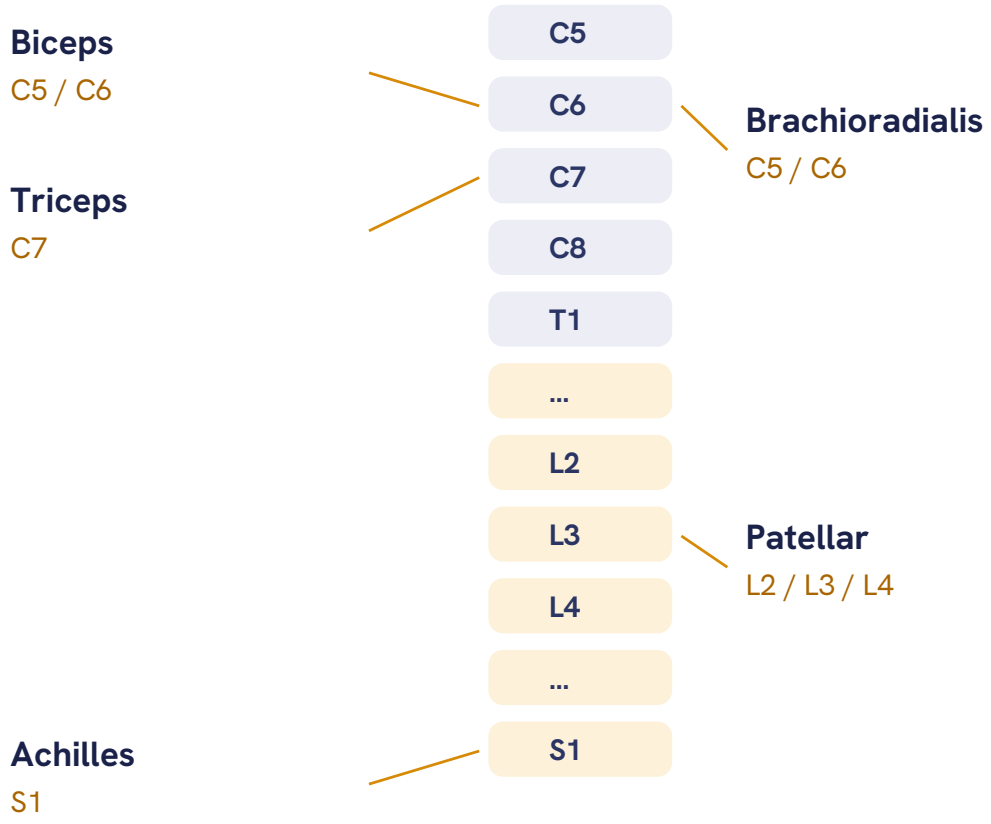
THE SHAPE OF THE ANSWER

1	**Head to foot**	Titubation, eyes, speech, then the limb tests
2	**Trunk and stance**	Sit unsupported, then stand, then Romberg
3	**Gait**	Five metres, three views, then the named pattern
4	**Fundus**	The disc, then the discriminator against optic neuritis
5	**Localise**	Midline against hemispheric, and which side

Head to foot, then stand the patient up. Inspect for titubation; eyes for broken pursuit and gaze-evoked nystagmus beating towards the lesion; speech for scanning dysarthria.

Reflexes have levels.

Segmental associations for the motor examination; not hammer-placement guidance.



COMPARE SIDES, THEN INTERPRET THE PATTERN

Root contributions overlap. Combine reflexes with power and sensation.

These labels are chapter-level associations, not proof of a single-root lesion.

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Biceps and brachioradialis C5–C6; triceps C7; patellar L2–L4; Achilles S1, following the chapter. The connecting lines organise segmental associations; they do not show tendon anatomy or hammer position. Root contributions overlap, so combine reflexes with strength and sensation.

Compare sides and combine the pattern with power and sensation.

Schematic prepared by Luna for Weave Viva, based on P3-06. Original vector diagram.

Then the limb tests. The Stewart-Holmes rebound, pressing the outstretched forearms down by roughly 10 cm and releasing so the affected arm flies past baseline; finger-to-nose with your finger about 50 cm away; rapid alternating hand turning; heel-to-shin.

Then reflexes and posture. Reflexes with the legs hanging free, looking for a pendular knee jerk swinging more than 4 times, where 4 or fewer is normal; then the trunk sitting unsupported; then gait.

FEATURE	MIDLINE, VERMAL	HEMISPHERIC, LATERAL
Dominant deficit	Axial and locomotor equilibrium	Appendicular limb incoordination
Trunk	**Cannot sit unsupported; falls backwards**	**Truncal stability relatively preserved**
Limb tests in bed	**May be entirely normal**	Dysmetria, past-pointing, intention tremor
Tone and reflexes	Limb tone often normal; titubation	Ipsilateral hypotonia, rebound, pendular reflexes
Causes	Chronic alcohol and malnutrition, medulloblastoma	Hemisphere infarct, astrocytoma, metastasis, plaque

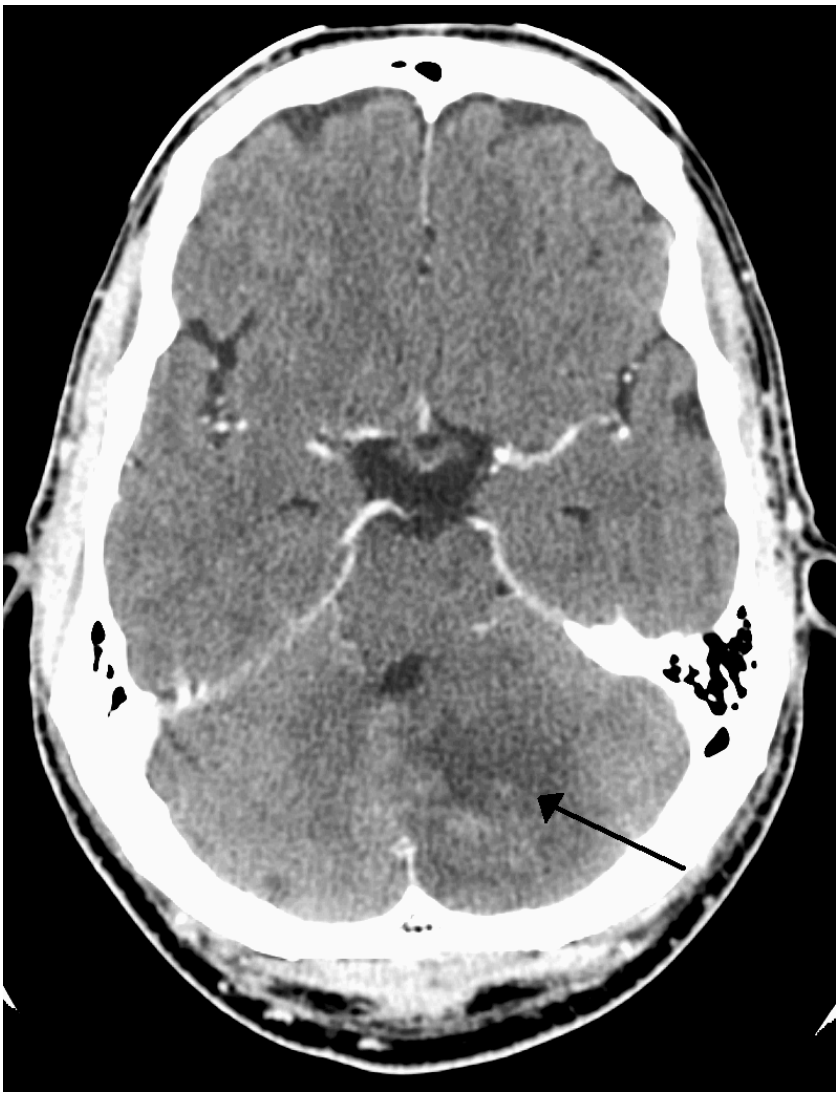
TRAP Examining a bed-bound patient and concluding the cerebellum is normal. Vermian lesions spare appendicular coordination, so finger-nose and heel-shin in bed can be normal, and truncal ataxia is missed unless you sit the patient unsupported, stand them and walk them.

Walk the patient about 5 metres barefoot, observing from front, behind and side, and read posture, base width, stride length, cadence, symmetry, arm swing, clearance and turning. Gait disturbance affects around 15% of individuals by age 60 and more than 80% of those over age 85.

GAIT	MECHANISM	DISCRIMINATOR
Hemiplegic	Unilateral upper motor neuron lesion; leg functionally too long	**Circumduction in a lateral arc with the knee held extended**
Spastic diplegic	Bilateral periventricular white matter injury	**Thighs cross the midline: scissoring**
Cerebellar ataxic	Vermis or hemisphere	**Unsteady with eyes open and closed alike**
Sensory ataxic	Dorsal columns or large sensory fibres	**Stamping, much worse in the dark; Romberg positive**
Parkinsonian	Basal ganglia, dopamine depletion	**Narrow base with festination and reduced arm swing**
High-stepping	Dorsiflexor weakness, foot drop	**Vertical lift of the foot, not a lateral arc**

Left cerebellar infarct on CT

Companion to original page 228



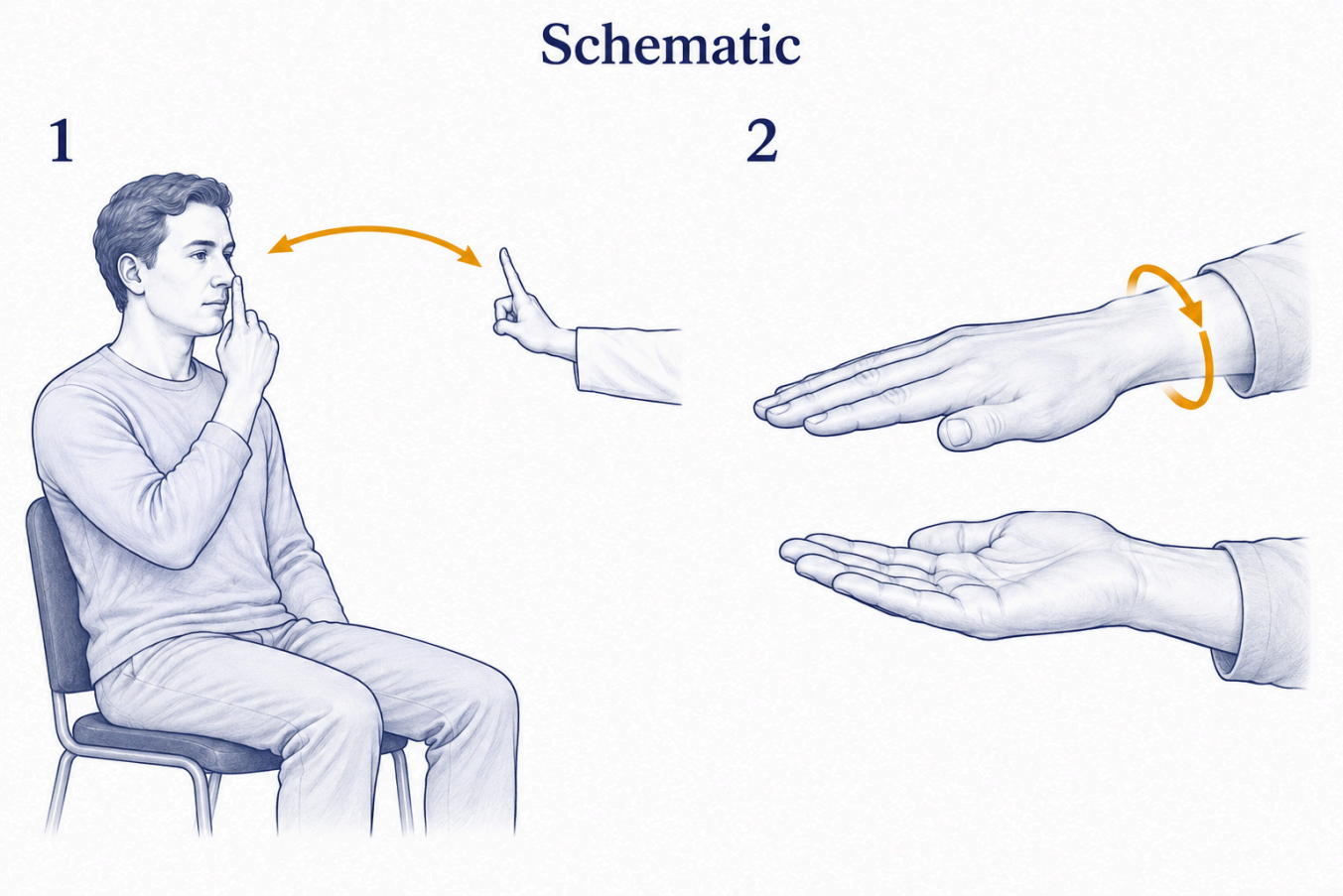
CT, axial plane. The source arrow identifies left cerebellar hypoattenuation in a stroke attributed to vertebral artery occlusion. The image is CT, not MRI. Vertebral artery occlusion is the source-case explanation and is not directly demonstrated by this slice.

Connect a posterior-fossa lesion to the cerebellar examination and verify image-side orientation.

James Heilman, MD. Leftsidedcerebellarstroke.PNG. CC BY-SA 3.0. https://commons.wikimedia.org/wiki/File:Leftsidedcerebellarstroke.PNG Licence: https://creativecommons.org/licenses/by-sa/3.0 Unmodified.

Finger–nose and rapid alternating movements

Companion to original page 228



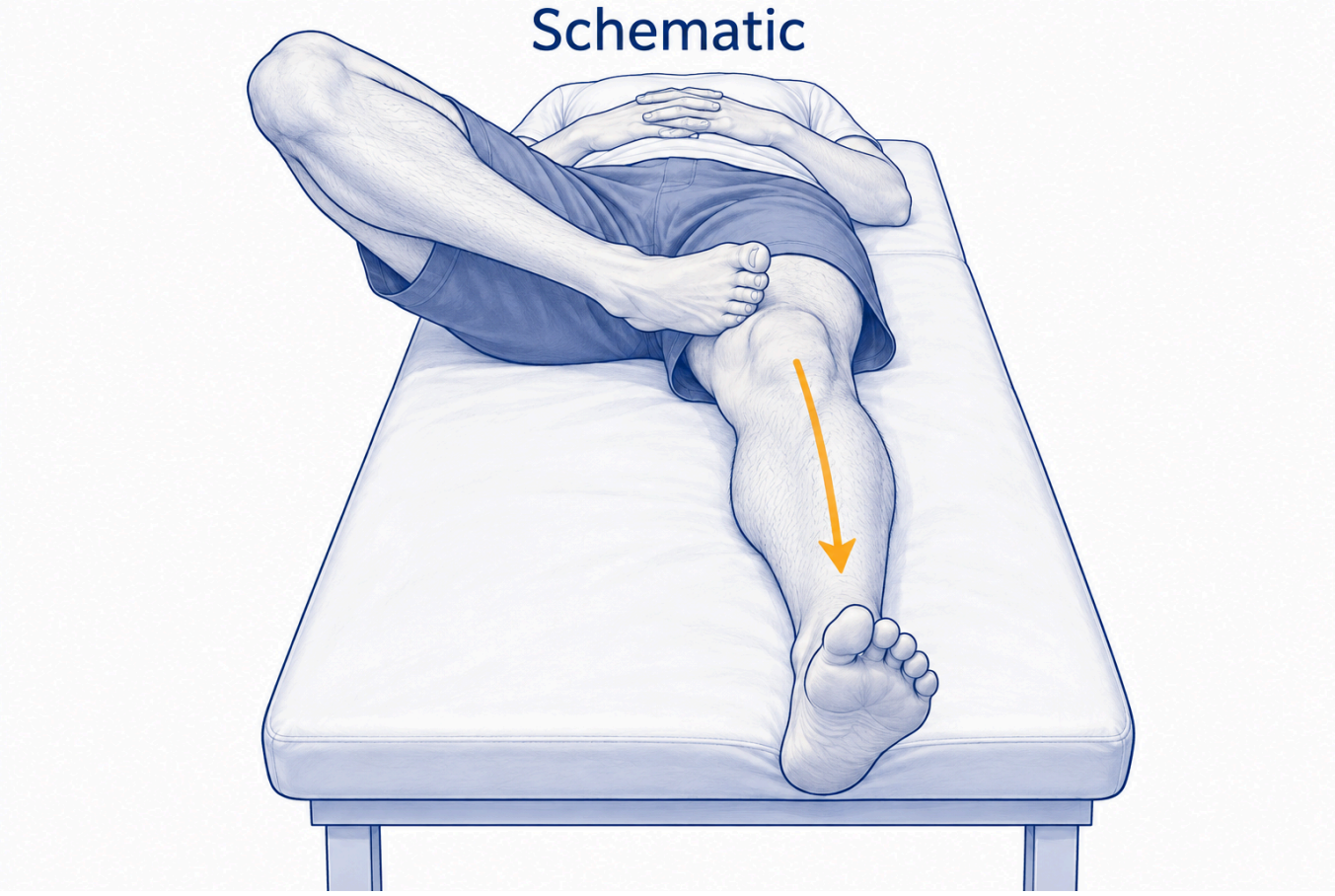
Schematic. Upper panel: the patient alternates their index finger between their nose and the examiner's target. Lower panel: repeated pronation and supination against the opposite palm. Observe accuracy, rhythm and smoothness on each side. Static positions illustrate the manoeuvres; they cannot show dysmetria, intention tremor or dysdiadochokinesia.

Compare sides while accounting for weakness, pain and comprehension.

Schematic prepared by Luna for Weave Viva, based on P3-07. AI-generated educational illustration.

Heel–shin: follow the opposite tibia

Companion to original page 228



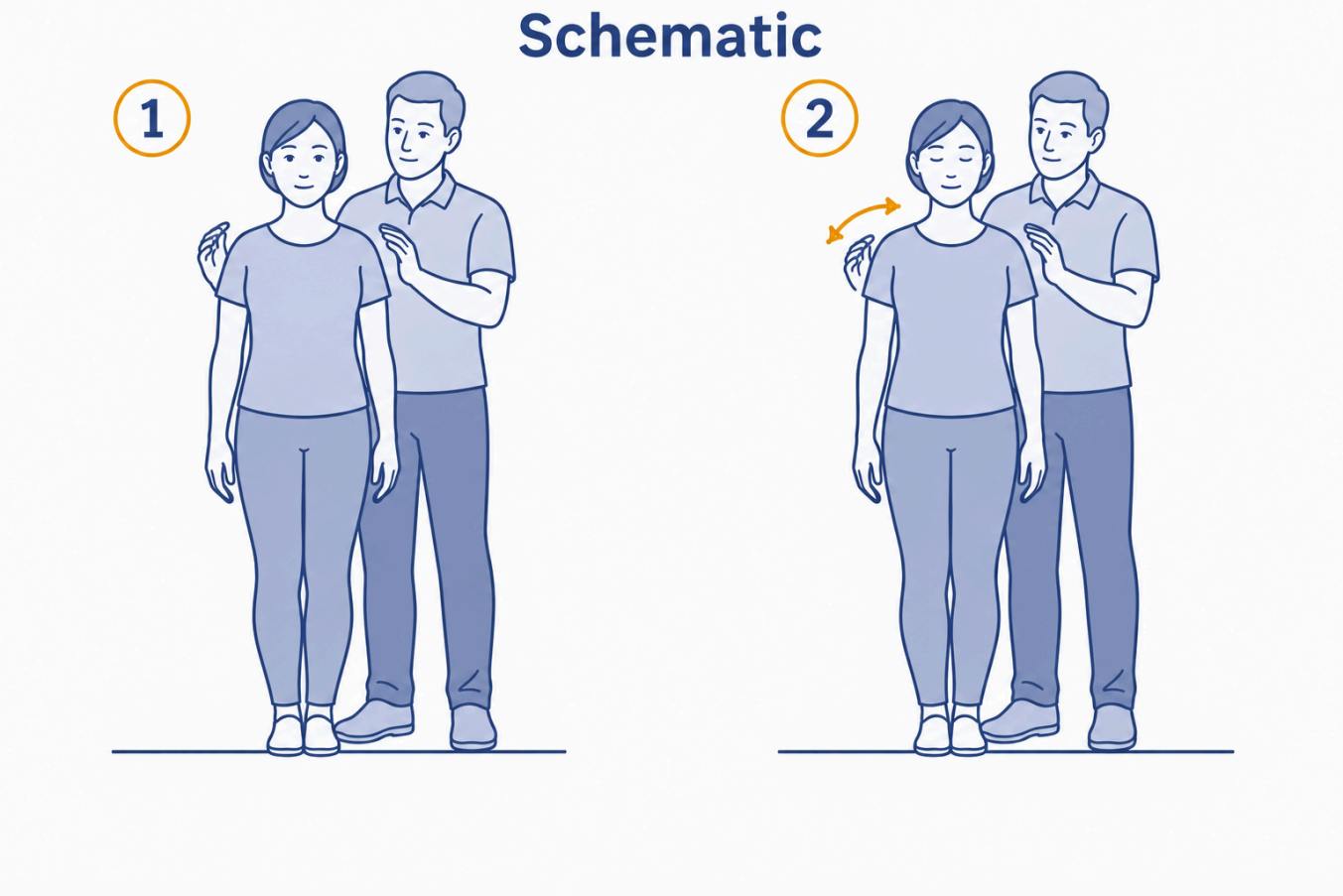
Schematic. Supine starting position: place one heel on the opposite knee, then slide it down the shin towards the ankle. The arrow shows the intended path, not a measured performance. Observe deviation, irregularity and repeated loss of contact; interpret these alongside power and position sense.

Demonstrate the task before assessing the movement.

Schematic prepared by Luna for Weave Viva, based on P3-07. AI-generated educational illustration.

Romberg: compare vision available and removed

Companion to original page 228



Schematic. 1: stand with feet together and eyes open. 2: close the eyes only when safe, with the examiner close enough to guard against a fall without steadying the patient. Increased instability on eye closure can support sensory or vestibular dependence. Instability already present with eyes open needs broader interpretation. This illustration cannot establish a positive sign.

Guard the patient and stop before a fall; do not test unsupported when unsafe.

Schematic prepared by Luna for Weave Viva, based on P3-07. AI-generated educational illustration.

GAIT	MECHANISM	DISCRIMINATOR
Waddling	Bilateral hip abductor weakness	**Alternating lateral trunk sway with lumbar hyperlordosis**
Apraxic, frontal	Frontal lobe, higher-level planning	**Supine cycling and stepping movements preserved**

The fundus. A psychiatrist carries an ophthalmoscope for a short list of reasons, and each one is examinable.

- **Concept.** A frontal tumour or chronic subdural presenting as personality change.
- **Concept.** Raised pressure before electroconvulsive therapy.
- **Concept.** Idiopathic intracranial hypertension, associated with lithium among other agents.
- **Concept.** Malignant hypertension presenting as delirium, and nutritional optic neuropathy in alcohol dependence.

Approach 10 to 15 degrees temporal to fixation and trace a vessel centrally, because branches point like arrows to the disc.

FEATURE	PAPILLOEDEMA, EARLY	OPTIC NEURITIS	ISCHAEMIC OPTIC NEUROPATHY
Acuity	**Preserved early**	**Rapidly and markedly reduced**	**Sudden profound loss**
Pupil	No relative afferent defect	Marked relative afferent defect	Marked relative afferent defect
Pain	Headache worse lying flat	Pain on eye movement	Absent unless giant cell arteritis
Fields	Enlarged blind spot	Central or centrocaecal scotoma	Altitudinal defect

TRAP Which antipsychotics injure the retina or anterior segment, at what dose threshold and with what monitoring interval, is not established in the library behind this note. Examiners ask it: name the question and print no figure. And with papilloedema present, imaging precedes lumbar puncture.

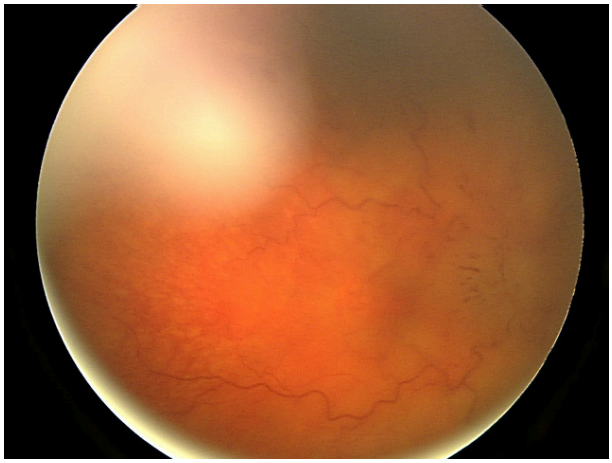
Fig 1.1 The examination as one descending column: attention, speech, cranial nerves, motor, sensory, reflexes, coordination, gait, fundus. Each station carries the one sign that changes the localisation, and the column is walked in the same order every time.

HOLD THIS

Romberg is not a cerebellar test, and a pendular knee jerk beyond 4 swings is. Papilloedema means raised pressure specifically, acuity is preserved early, and visible venous pulsations reassure while their absence does not.

Severe papilloedema: real fundus photograph

Companion to original page 229



Real fundus photograph of the right eye, described by the source as severe papilloedema. Inspect the elevated, poorly defined optic-disc region in this source example. A single fundus photograph does not measure intracranial pressure or determine its cause. Display width is limited to preserve the available source detail.

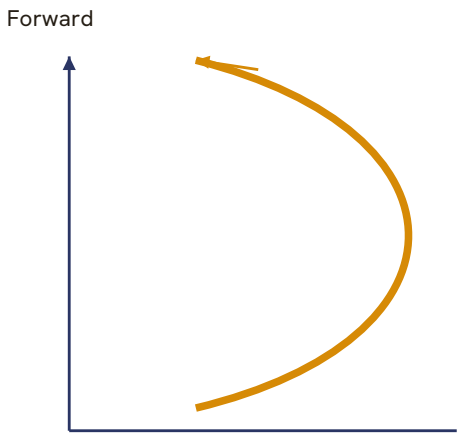
Inspect the swollen disc and obscured margins. Fundus companion to the cited cerebellum/gait/fundus chapter.

Shveta Bansal , Timothy Dabbs and Vernon Long. Fundal photograph showing severe papilloedema in the right eye. Journal of Medical Case Reports 2008;2:86. CC BY 2.0. https://commons.wikimedia.org/wiki/File:Fundal_photograph_showing_severe_papilloedema_in_the_right_eye.jpg Licence: https://creativecommons.org/licenses/by/2.0. Unmodified.

Watch the path of the foot.

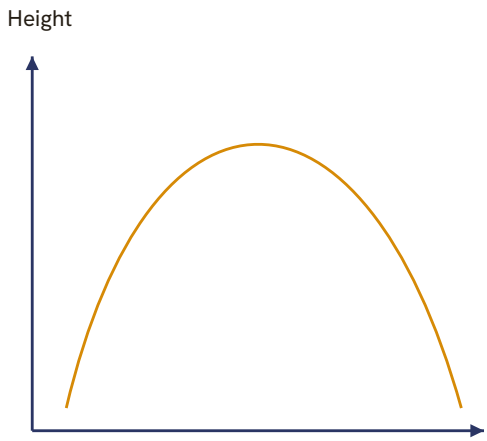
Motion diagrams distinguish trajectories; not motion-capture recordings.

CIRCUMDUCTION · TOP VIEW



Outward arc

HIGH-STEPPING · SIDE VIEW



Greater clearance

Observe joint movement, foot clearance and symmetry.

A drawing cannot show timing, speed or balance.

Schematic ▪ Original teaching diagram ▪ September 2026

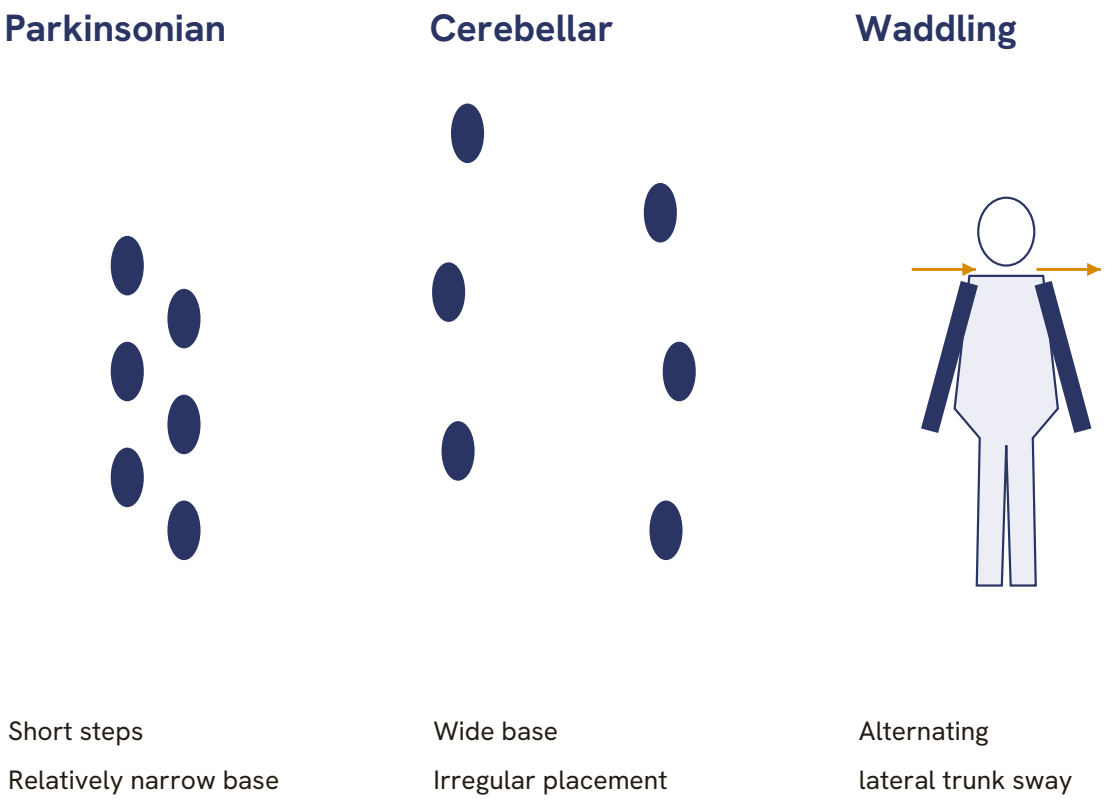
Schematic. Circumduction moves the swinging leg outward in an arc; high-stepping increases hip and knee flexion to clear the foot. The first diagram is a top-view foot path; the second is a side-view clearance path. Axes differ deliberately and are labelled. These schematic trajectories are not measured data or diagnostic by themselves.

Separate lateral swing from increased vertical clearance.

Schematic prepared by Luna for Weave Viva, based on P3-08. Original vector diagram.

Compare base, step length and trunk.

Top-view footprint patterns and a frontal trunk-sway cue.



Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Idealised contrasts: short steps with a relatively narrow base in a parkinsonian pattern; a wide, irregular base in a cerebellar pattern; alternating lateral trunk movement in a waddling pattern. Real gait findings vary and may overlap. These footprints and arrows are conceptual, not a recording, and do not establish an aetiology.

Describe base, stride, regularity, arm swing and trunk movement.

Schematic prepared by Luna for Weave Viva, based on P3-08. Original vector diagram.

WHAT EARNS THE MARKS

- **The trunk tested**: a vermal lesion leaves the limb tests normal in bed
- **Romberg placed correctly**: it is a proprioceptive test, and saying so earns the mark
- **The gait named with its mechanism**: circumduction, scissoring, stamping, festination
- **The fundus discriminated**: acuity and the afferent defect separate papilloedema from neuritis

SEE ALSO 05 Motor system: bulk, tone, power, coordination 14 Parkinson's disease, and the parkinsonism it is not 20 Dementia with focal signs, frontal syndrome

Localisation

9 · Upper versus lower motor neuron, and the mixed picture

LOCALISATION · TOPIC 09

Is this weakness upper or lower motor neuron, and what would a mixed picture mean?

SHORT CASE; VIVA TABLE ON MOTOR NEURON SIGNS

THE SHAPE OF THE ANSWER

1 Six features	Bulk, fasciculation, tone, weakness pattern, reflexes, plantar
2 Call it	Which motor neuron, and on which side
3 Look for mixing	Wasting with brisk reflexes changes the diagnosis
4 The acute trap	Acute lesions begin flaccid and areflexic
5 Name the level	The rung of the ladder the pattern puts you on

One dichotomy carries most of the marks. Upper motor neurons sit in the motor cortex and brainstem nuclei and descend in the corticospinal and corticobulbar tracts; lower motor neurons arise in the anterior horns and cranial motor nuclei and run in ventral roots, plexuses and nerves.

FEATURE	UPPER MOTOR NEURON	LOWER MOTOR NEURON
Bulk	Disuse atrophy only	**Marked, early atrophy**
Fasciculations	**Absent**	**Present and characteristic**
Tone	Spastic, clasp-knife; reduced acutely	Hypotonic or flaccid
Weakness	Pyramidal: arm extensors, leg flexors weaker	Segmental, root or named-nerve
Reflexes	Hyperreflexia; reduced acutely	Hyporeflexia or areflexia
Plantar	**Extensor: Babinski present**	**Flexor or absent**
Hand signs	Hoffmann and pronator drift positive	Absent

FEATURE	MOTOR NEURON DISEASE	CERVICAL SPONDYLOTIC MYELOPATHY
Sensation	**Preserved**	**Early proprioceptive loss, trunk level, dorsal column ataxia**
Bulbar	**Tongue wasting and fasciculation, brisk jaw jerk**	**Absent: the process is below the foramen magnum**
Lower motor neuron signs	Widespread across four regions	Confined to the arms at the compressed level
Neck provocation	Absent	**Lhermitte sign on active neck flexion**
Imaging	No structural compression	Narrowed canal, osteophytes, cord signal change

Amyotrophic lateral sclerosis is commonest, with onset in the sixth and seventh decade, about 5% familial, lower limb onset in 30% and bulbar onset in 20%.

Primary lateral sclerosis is pure upper motor neuron at 2% of cases, and progressive muscular atrophy pure lower motor neuron at 4%. Extraocular movements are spared, and death is from respiratory failure.

RULE **Brisk reflexes in a wasted, fasciculating limb with normal sensation is the sentence that makes motor neuron disease.** Look at the tongue and elicit the jaw jerk before committing, because those two separate it from cervical myelopathy.

INDIAN PRACTICE NOTE

Dextromethorphan with quinidine is the guideline agent for pseudobulbar affect but is not a routinely marketed or stocked Indian formulation, and Indian availability is not established in the library behind this note. Name it as the guideline option, then say Indian practice manages pseudobulbar affect with an off-label antidepressant, and quote no drug or dose for that second limb from this note.

HOLD THIS

An extensor plantar is the sign that survives spinal shock. Wasting with brisk reflexes is mixed disease; wasting with lost reflexes and normal sensation is anterior horn or peripheral; weakness with sensory loss lifts you off the motor unit.

WHAT EARNS THE MARKS

- **All six features offered**: a two-feature answer reads as a half-examined patient
- **The mixed picture named**: wasting with brisk reflexes is a diagnosis, not a contradiction
- **Acute flaccidity explained**: spasticity takes days to weeks to appear
- **Tongue and jaw jerk**: they separate motor neuron disease from cervical myelopathy

SEE ALSO 01 Where before what: the localisation ladder 07 Reflexes, clonus, plantars and the release signs 16 The wasted limb: motor neuron disease and neuropathy

Name the level first.

A schematic localisation ladder from cortex to muscle.

Cortex	Cortical signs with disproportionate weakness
↓	
Internal capsule	Dense, proportionate face–arm–leg weakness
↓	
Brainstem	Cranial-nerve and opposite-side limb signs
↓	
Spinal cord	Sensory level; look below the lesion
↓	
Root / plexus / nerve	Myotomal or named-nerve pattern
↓	
Neuromuscular junction	Fatigability; test repetition
↓	
Muscle	Predominantly proximal weakness

CHECK THE EXCEPTION

Acute central lesions can begin with reduced tone and reflexes.
Use the distribution and the rest of the examination together.

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. A sequence of localisation levels from cortex to muscle with the bedside finding that supports each level. Acute central lesions can initially have reduced tone and reflexes. The ladder links distributions to anatomical levels rather than prescribing a diagnosis. Use the accompanying sensory, cranial-nerve and reflex findings to test the proposed level.

Name the anatomical level before proposing the cause.

Schematic prepared by Luna for Weave Viva, based on chapter P3-11. Original edition artwork; publication licence to follow the edition.

10 · Spinal cord syndromes

LOCALISATION · TOPIC 10

This patient has a spastic paraparesis. Give me the level, then the cord syndrome.

SHORT CASE; VIVA TABLE ON CORD SYNDROMES

THE SHAPE OF THE ANSWER

1 **Vertical level**	Limbs involved, highest sensory dermatome, segmental reflexes
2 **Horizontal pattern**	Which tracts are in the lesion, which are spared
3 **Name the syndrome**	One of the recognised cord syndromes, with its spared tract
4 **The paradox**	Absent ankle jerks with bilateral extensor plantars
5 **Investigate**	Imaging, then the deficiency screen, in that order

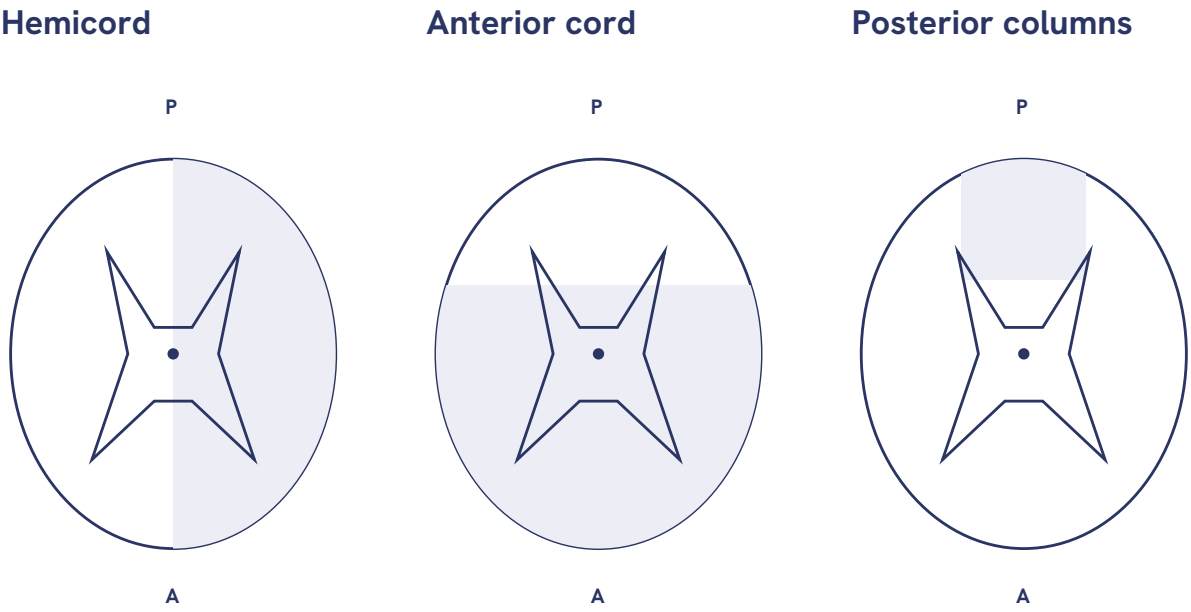
Two axes, then a name. Vertical and horizontal localisation are two separate questions.

- **Concept.** Vertical: the extremities involved, the uppermost dermatome with sensory loss, and the segmental reflexes.
- **Concept.** Horizontal: which tracts sit inside the lesion.
- **Key number.** The cord runs from the medulla to the conus between L1 and L2, with 31 functional segments.
- **Key number.** Spinothalamic fibres cross in the anterior white commissure classically within 1 to 2 segments of entry.

SYNDROME	TRACTS DAMAGED	SPARED	PROFILE
Complete transection	All	**Nothing below the level**	Para or quadriplegia, complete sensory loss, sphincter paralysis
Brown-Sequard	Ipsilateral corticospinal and dorsal columns; crossing spinothalamic	**Touch, by dual pathways**	Ipsilateral motor and proprioceptive loss, contralateral thermoalgesic loss
Anterior cord	Ventral two-thirds	**Dorsal columns**	Bilateral paralysis and thermoalgesic loss, vibration intact
Central cord	Central grey, crossing fibres, medial corticospinal	**Sacral segments**	Arms worse than legs, distal hand clumsiness, burning dysaesthesia
Posterior column	Bilateral dorsal columns	**Corticospinal and spinothalamic**	Sensory ataxia, positive Romberg, power and pain intact
Subacute combined degeneration	Dorsal columns and lateral corticospinal tracts	**Anterior columns**	Vibration and position loss, spastic paraparesis, extensor plantars

Which tract is interrupted?

Schematic cord sections: posterior at top, anterior at bottom.



Posterior columns: position and vibration

Lateral corticospinal tract: descending motor pathway

Anterolateral system: pain and temperature

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Idealised cord lesions: hemicord, anterior cord and posterior columns. Shading indicates lesion extent, not MRI signal. Relate corticospinal, dorsal-column and anterolateral pathways to the shaded region. Real lesions do not always respect these tidy boundaries, and the level and timing influence the examination.

Predict affected and preserved modalities before naming the syndrome.

Schematic prepared by Luna for Weave Viva, based on P3-13. Original vector diagram.

Brown-Sequard gives ipsilateral spastic paresis with ipsilateral vibration and position loss, and contralateral pain and temperature loss beginning 1 to 2 segments below the level, because the fibres cross that far above their entry.

It has the best prognosis of the incomplete syndromes, with a secondary source reporting up to 99% regaining independent ambulation. **Anterior cord syndrome** spares the posterior columns, which are fed by the paired posterior spinal arteries, and carries the poorest prognosis, with only 10% to 20% achieving functional motor recovery.

Subacute combined degeneration is the case a psychiatrist is most likely to be handed, because psychiatric symptoms can be the only manifestation, and cobalamin deficiency is documented in 6% of general hospital psychiatric admissions.

The triad is dorsal column loss with a stepping-on-cotton gait, corticospinal loss with spastic paraparesis and extensor plantars, and a peripheral neuropathy that abolishes the ankle jerks. That gives the examinable paradox of **absent ankle jerks with bilateral extensor plantars**.

TRAP A normal serum vitamin B12 does not exclude the diagnosis: it is normal in up to 30% of patients with genuine tissue deficiency, so order methylmalonic acid and homocysteine. Spinal imaging is also normal in 60% to 85% of cases; when abnormal it shows symmetric dorsal column T2 hyperintensity, the inverted V or rabbit-ears sign. And giving folate before establishing the B12 level corrects the anaemia while the cord disease progresses.

HOLD THIS

The contralateral level in Brown-Sequard sits 1 to 2 segments below the true lesion. Absent ankle jerks with bilateral extensor plantars means a combined myeloneuropathy, and in practice that means B12.

WHAT EARNS THE MARKS

- **Two axes answered separately**: a level without a tract pattern is half an answer
- **The spared tract named**: it is what makes the syndrome the one you called
- **The paradox recognised**: lost ankle jerks with extensor plantars is a combined lesion
- **The deficiency screen offered**: a normal level does not close the question

SEE ALSO 01 Where before what: the localisation ladder 06 Sensory system: modalities, dermatomes, patterns 16 The wasted limb: motor neuron disease and neuropathy

11 · Brainstem syndromes and crossed signs

LOCALISATION · TOPIC 11

What does a crossed deficit tell you, and how do you level a brainstem lesion?

SHORT CASE; VIVA TABLE ON BRAINSTEM SYNDROMES

THE SHAPE OF THE ANSWER

1 **Recognise it**	Ipsilateral cranial palsy with a contralateral long tract deficit
2 **Vertical level**	The cranial nerve involved gives midbrain, pons or medulla

One hemicord. Different sides of loss.

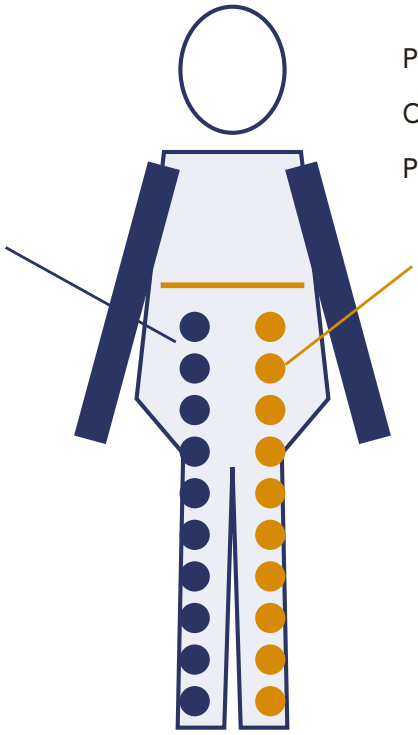
Right hemicord example; the person is shown from the front.

PATIENT RIGHT

Ipsilateral below lesion

Motor

Position / vibration



PATIENT LEFT

Contralateral below lesion

Pain / temperature

Face-on view: patient right is page left.

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Idealised right hemicord syndrome: below the lesion, motor weakness and loss of position/vibration are ipsilateral, while pain/temperature loss is contralateral and often starts below the lesion level. Segmental signs at the lesion are omitted. The coloured body regions are explanatory overlays, not a clinical photograph.

Explain the asymmetry using the different crossing levels.

Schematic prepared by Luna for Weave Viva, based on P3-13. Original vector diagram.

3 Horizontal side	Medial 4M compartment against the lateral compartment
4 Name the syndrome	Wallenberg or Dejerine, with its discriminator
5 Exclude locked-in	Vertical saccades and blinking to command, before writing coma

Crossed signs mean brainstem, and nothing else does. An alternating syndrome is an ipsilateral lower motor neuron cranial nerve palsy with a contralateral long tract deficit.

Cranial motor nuclei exit ipsilaterally without crossing, the fourth nerve excepted because it decussates dorsally in the anterior medullary velum. The corticospinal tract descends uncrossed through crus, basis pontis and pyramid, and undergoes approximately 90% crossing at the caudal medullary decussation.

LEVEL	CRANIAL NERVES	LOCALISING FEATURES
Midbrain	**III and IV**	Ptosis, mydriasis, impaired adduction and vertical movement
Pons	**V, VI, VII, VIII**	Impaired mastication and midface sensation; abduction failure or gaze palsy; lower motor neuron facial weakness; deafness and vertigo
Medulla	**IX, X, XI, XII**	Dysphagia, hoarseness, lost gag; tongue wasting with deviation on protrusion

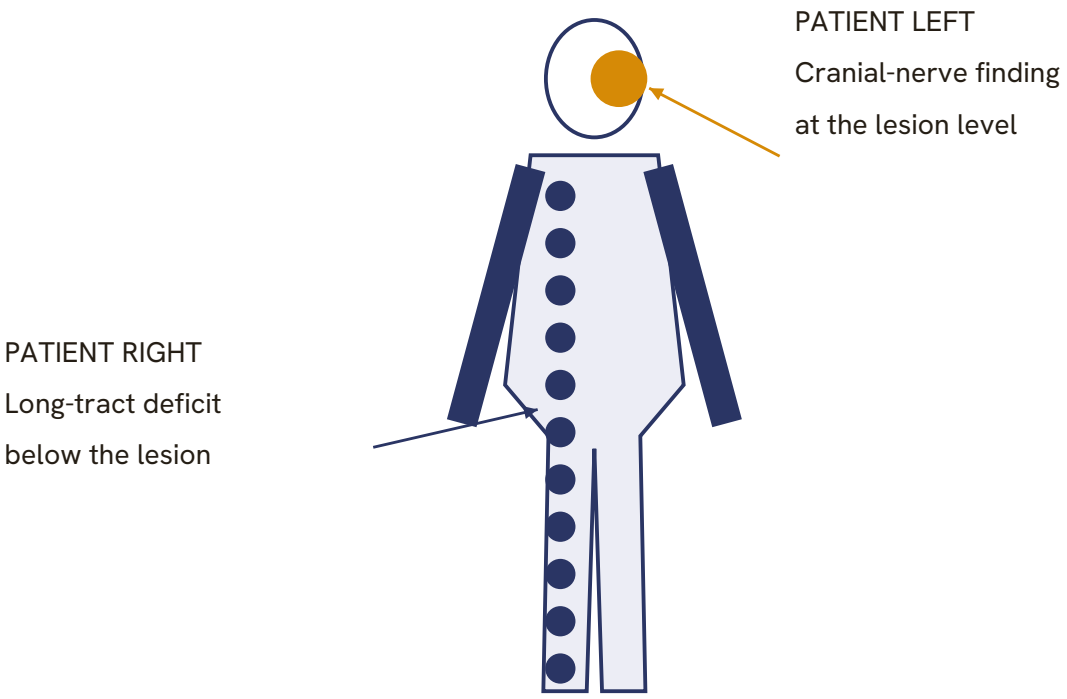
The horizontal axis has a medial and a lateral compartment, and the 4M rule carries the medial side.

- **Concept.** Motor cranial nuclei whose Roman numerals divide into 12, meaning III, IV, VI and XII.
- **Concept.** The medial longitudinal fasciculus, giving internuclear ophthalmoplegia.
- **Concept.** The motor pathway, giving contralateral hemiparesis.
- **Concept.** The medial lemniscus, giving contralateral loss of vibration, position and discriminative touch.

The lateral compartment carries the spinal trigeminal complex for ipsilateral facial pain and temperature, the cerebellar peduncular pathways for ipsilateral ataxia, the spinothalamic tract for contralateral body pain and temperature, and descending sympathetic fibres for ipsilateral Horner syndrome.

The face and body point across.

A left-sided brainstem example, illustrated face-on.



Determine the actual nerve and tract; “crossed” is a localisation clue.

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Crossed signs combine an ipsilateral cranial-nerve finding with a contralateral long-tract deficit. This schematic shows the laterality principle, not one universal syndrome: the affected nerve and tract depend on the brainstem level. Midbrain, pontine and medullary syndromes have different combinations.

Name the cranial nerve, then the opposite-side body deficit.

Schematic prepared by Luna for Weave Viva, based on P3-14. Original vector diagram.

FEATURE	LATERAL MEDULLARY (WALLENBERG)	MEDIAL MEDULLARY (DEJERINE)
Cranial deficit	IX and X with dysphagia; V with facial thermoalgesic loss	**XII with wasted fasciculating tongue**
Tongue	**Midline protrusion**	**Deviates towards the lesion**
Limb power	**Preserved**	**Contralateral spastic hemiparesis**
Sensory	Crossed thermoalgesic: ipsilateral face, contralateral body	Contralateral lemniscal loss
Horner and ataxia	Both ipsilateral and prominent	Both absent

RULE **In any patient with acute anarthria and quadriplegia, test voluntary vertical saccades and blinking to command before writing down coma.** Reading extensor posturing as decerebrate rigidity is the error that misses a conscious patient.

HOLD THIS

Ipsilateral cranial, contralateral limbs, and the cranial nerve gives the level. Medial lesions take the even numbers, the lemniscus and the pyramid; lateral lesions take pain and temperature, ataxia and the sympathetic chain, and spare power.

WHAT EARNS THE MARKS

- **The word crossed used precisely**: cranial on one side, long tract on the other
- **The nerve as the level**: III and IV midbrain, V to VIII pons, IX to XII medulla
- **Medial against lateral**: even numbers and the lemniscus, or pain, ataxia and Horner
- **Locked-in excluded aloud**: it is the error that misses a conscious patient

SEE ALSO 04 Cranial nerves in exam order 06 Sensory system: modalities, dermatomes, patterns 13 Hemiparesis and the stroke short case

12 · Hemispheric syndromes: dominant and non-dominant

LOCALISATION · TOPIC 12

Which hemisphere is affected here, and what did you test to say so?

SHORT CASE; VIVA TABLE ON PARIETAL SIGNS

THE SHAPE OF THE ANSWER

1	**Set the hemisphere**	Handedness first, then dominant or non-dominant
2	**Dominant signs**	Language, calculation, writing, praxis
3	**Non-dominant signs**	Neglect, anosognosia, dressing and constructional apraxia
4	**Test neglect**	Double simultaneous stimulation, line bisection, cancellation
5	**Name it**	Lobe plus hemisphere, with the deficit that showed it

Set the hemisphere, then the lobe. The dominant hemisphere is the language hemisphere, left in over 95% of right-handers and in over 60% to 70% of left-handers, mediating verbal processing, analytical arithmetic and praxis programming, while the non-dominant hemisphere

carries visuospatial processing, directed spatial attention, geographical orientation and emotional prosody.

DOMAIN	DOMINANT LESION	NON-DOMINANT LESION
Language	**Broca, Wernicke, conduction or anomic aphasia**	**Grammar intact; prosody and musical memory impaired**
Praxis	Ideomotor and ideational apraxia, often bilateral	Dressing and constructional apraxia
Parietal syndrome	**Gerstmann tetrad**	**Hemispatial and personal neglect, anosognosia**
Spatial cognition	Orientation preserved; symbolic calculation fails	Gets lost in familiar places

Neglect is failure to report, respond or orient to contralesional stimuli, not attributable to a primary sensory or motor defect.

Classify it into **4 subtypes** and test each one.

- **Concept.** Spatial neglect: the contralesional half of space is not explored.
- **Concept.** Personal neglect: the left cheek is unshaved and the left arm undressed.
- **Concept.** Motor neglect: contralesional limbs are underused despite preserved power.
- **Concept.** Representational neglect: the contralesional side of a recalled scene is omitted.

RULE **Hemianopia is known to the patient, neglect is not.** A patient with an isolated field defect turns head and eyes to the blind side to explore; a patient with neglect denies the deficit, does not initiate saccades into the neglected side, and extinguishes contralesional stimuli on double simultaneous stimulation while detecting single ones.

APRAXIA	CORE DEFICIT	WHAT YOU SEE	LOCALISATION
Limb-kinetic	Loss of fine independent finger movement	**Coarse, clumsy dexterity with small objects**	Corticospinal or premotor
Ideomotor	Breakdown between concept and execution	**Spatial errors on pantomime; body part used as the object**	Dominant inferior parietal lobule, arcuate fasciculus
Ideational	Loss of the plan of a multistep sequence	**Steps out of order: sealing the envelope before the letter goes in**	Dominant parieto-temporal, bilateral networks

INDIAN PRACTICE NOTE

Test a multilingual patient in the language acquired in early childhood as well as any second language, because arithmetic retrieval and overlearned drills are strongest in the first.

Where schooling has been limited, failure at written arithmetic frequently reflects education rather than angular gyrus pathology. Assess calculation with currency handling and market change at the premorbid functional level before calling it acalculia.

HOLD THIS

Dominant parietal gives Gerstmann and limb apraxia; non-dominant parietal gives neglect, anosognosia, dressing and constructional apraxia. Balint has vision but cannot assemble or reach; Anton has no vision and denies it.

WHAT EARNS THE MARKS

- **Handedness before hemisphere**: the syndrome list depends on it
- **Extinction demonstrated**: double simultaneous stimulation is what proves neglect
- **The tetrad named**: agraphia, acalculia, finger agnosia, right-left disorientation
- **Education and language allowed for**: calculation tested at the premorbid level

SEE ALSO 02 Higher mental function and lobe testing 03 Speech and language 13 Hemiparesis and the stroke short case

13 · Hemiparesis and the stroke short case

LOCALISATION · TOPIC 13

This patient has a hemiparesis. Localise it, then take me through the acute management.

LONG CASE AND SHORT CASE; STROKE VIVA TABLE

THE SHAPE OF THE ANSWER

1 Distribution	Face, arm and leg, and which of them is worst
2 Cortical or not	Aphasia, neglect, field cut, gaze deviation, each asked for
3 Territory	The vessel, then what imaging should show
4 Acute pathway	Glucose, imaging, then thrombolysis assessment
5 Prevention	Targets by domain, plus the psychiatric review

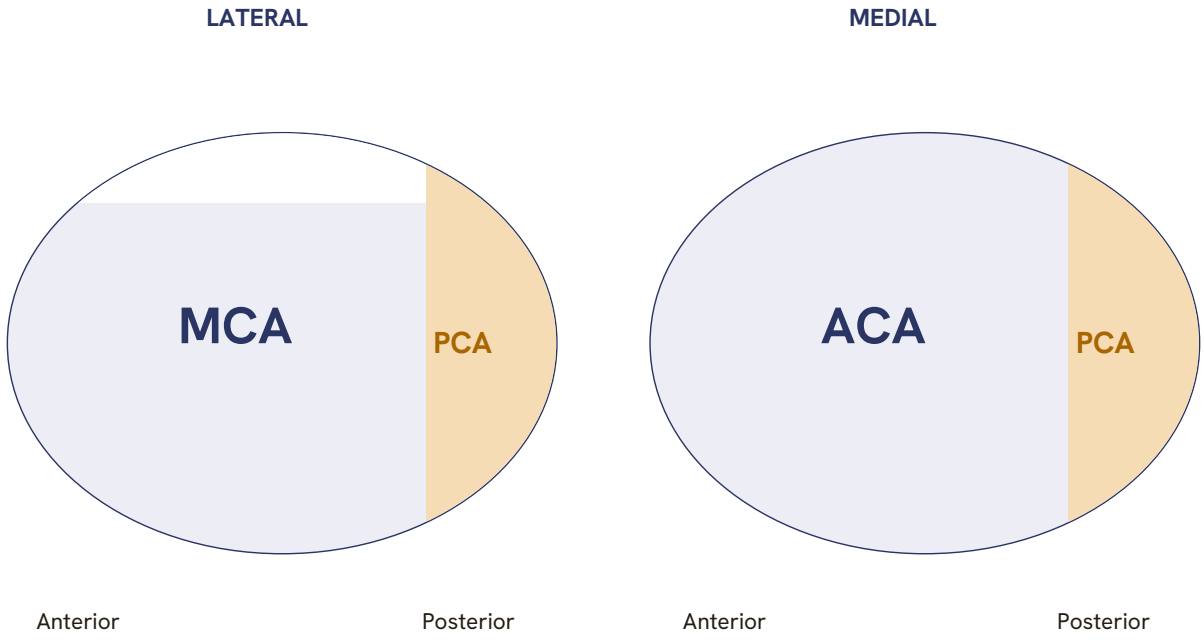
The distribution of the weakness is the localisation. Five patterns, five levels.

- **Concept.** Face and arm worse than leg: the lateral convexity, in the middle cerebral territory.
- **Concept.** Leg worse than face and arm: the parasagittal cortex, in the anterior cerebral territory.
- **Concept.** Equal involvement: the posterior limb of the internal capsule, or the basis pontis.
- **Concept.** Crossed signs: brainstem.
- **Concept.** Face spared with a normal jaw jerk: high cervical cord.

1. Is this an upper or lower motor neuron deficit, and where does the facial weakness sit?
2. What is the distribution across face, arm and leg, and what level does that give?
3. Are there cortical signs, and which ones did you look for?
4. Is this lacunar or cortical, and what makes you say so?
5. Which vascular territory, and what does that predict on imaging?
6. How would you assess severity, and what are the limitations of the scale you quote?
7. Is this patient a thrombolysis candidate, and what would exclude them?
8. What secondary prevention would you start, with targets?

Relate the surface to the artery.

Idealised hemispheric territories; borders vary between people.



Surface territories are a starting point.

Check deep structures, border zones and clinical context.

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Conceptual cortical arterial territories: MCA over much of the lateral convexity, ACA over the medial frontal/parietal surface and PCA over the occipital region. Deep perforator territories and border zones are not mapped. This is a spatial teaching diagram, not a perfusion study or a patient-specific arterial atlas.

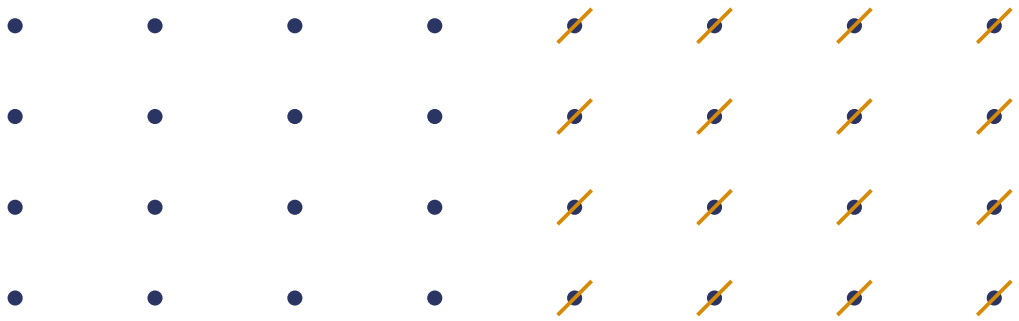
Connect the affected body region and cortical signs to a plausible territory.

Schematic prepared by Luna for Weave Viva, based on P3-16. Original vector diagram.

Attention and vision are different questions.

Original demonstration marks, not a validated cancellation test.

Left-sided omissions



Unmarked left space

Marked right space

Compare with formal visual-field examination

Left field shaded in BOTH eyes: left homonymous loss

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Left-sided omissions on an original cancellation example illustrate inattention to one side of space. A homonymous field defect instead concerns visual-field loss assessed in both eyes. Neglect and field loss may coexist; this diagram does not distinguish them in a real patient without examination. The cancellation array is a teaching example, not a scored instrument.

Check visual fields and attention separately; consider extinction.

Schematic prepared by Luna for Weave Viva, based on P3-15. Original vector diagram.

9. What will you look for psychiatrically, and what would you do about it?

LACUNAR SYNDROME	SITE	FEATURES
Pure motor hemiparesis	Posterior limb or basis pontis	**Equal face, arm and leg weakness with no sensory, visual or cortical deficit; 33% to 50% of lacunes**
Ataxic hemiparesis	Posterior limb, basis pontis or corona radiata	**Homolateral cerebellar ataxia with pyramidal hemiparesis, leg more than arm**
Dysarthria-clumsy hand	Genu of internal capsule or basis pontis	**Severe dysarthria with contralateral hand clumsiness, worst on writing**
Pure sensory stroke	Ventral posterolateral thalamic nucleus	**Contralateral numbness of face, arm and leg**
Mixed sensorimotor	Thalamus with adjacent posterior limb	**Hemiparesis with hemisensory loss**

TRAP The striatocapsular infarct sits between the categories: a comma-shaped infarct of caudate, putamen and anterior limb measuring at least 30 mm long and 10 mm wide, from proximal middle cerebral occlusion, which can give subcortical hemiparesis plus transient cortical deficits despite sparing the cortex. Do not call it a lacune because the weakness is dense.

TRAP The stroke severity scale is quotable by domain but not by number from the library behind this note: the item count, the total range and the severity bands are not established here. The limitation is the examinable part anyway, because the scale is weighted towards anterior circulation and dominant-hemisphere deficits, so posterior circulation strokes with isolated vertigo, ataxia or dysphagia, and non-dominant strokes with anosognosia or subtle neglect, can score low despite significant impairment.

The acute pathway opens with airway, breathing, circulation and an immediate capillary glucose, because hypoglycaemia is a common mimic.

Then emergency non-contrast computed tomography, to separate ischaemic stroke, around 85% of cases, from intracerebral haemorrhage, around 15%.

Then thrombolysis assessment: onset to administration within 4.5 hours, age 18 years or older, and no haemorrhage and no early ischaemic change over more than one third of the middle cerebral territory.

DOMAIN	TARGET OR ACTION
Blood pressure	**Office target below 130/80 mm Hg, one of the most critical interventions**
Diabetes	**Screen after stroke; HbA1c at or below 7%. New type 2 diabetes is found in 11.5% of acute ischaemic strokes and prediabetes in 36.2%**

DOMAIN	TARGET OR ACTION
Lipids	**Atorvastatin 80 mg daily if LDL above 100 mg/dL; atherosclerotic target LDL below 70 mg/dL, adding ezetimibe if needed**
Antiplatelet	**Aspirin 50 to 325 mg daily, or clopidogrel 75 mg daily, or aspirin 25 mg with extended-release dipyridamole 200 mg twice daily**
Atrial fibrillation	**Oral anticoagulation; atrial fibrillation carries a 4 to 5 fold increase in stroke risk**
Lifestyle	At least 10 minutes four times a week of moderate or 20 minutes twice a week of vigorous exercise; smoking doubles stroke risk
Sleep apnoea	Screen and treat with positive airway pressure; present in 38% to 40% of stroke patients

HOLD THIS

Proportionate weakness with no cortical sign is a capsular lacune; disproportionate weakness with any cortical sign is cortical. Ask for aphasia, neglect, a field cut and gaze deviation explicitly, because their absence is what makes the lacunar diagnosis.

WHAT EARNS THE MARKS

- **Distribution before diagnosis**: the pattern is the localisation
- **Cortical signs reported as absent**: their absence is what makes a lacune
- **Glucose before imaging**: the mimic is checked in the first minute
- **Targets with the scale's limits**: quote the domain, then say what the scale misses

SEE ALSO 09 Upper versus lower motor neuron, and the mixed picture 11 Brainstem syndromes and crossed signs 23 Head injury, headache and focal symptoms in the history

The cases

14 · Parkinson's disease, and the parkinsonism it is not

THE CASES · TOPIC 14

Examine this patient and tell me whether this is Parkinson's disease.

SHORT CASE; LONG CASE COMORBIDITY; MOVEMENT DISORDER VIVA

THE SHAPE OF THE ANSWER

1 **Is it parkinsonism**	Bradykinesia with tremor or rigidity, demonstrated aloud
2 **Show the decrement**	Repetition tasks, then the Froment manoeuvre
3 **Asymmetry and tempo**	Onset side, and when postural instability appeared
4 **Criteria**	Supportive features, absolute exclusions, red flags
5 **Treatment**	Motor complications, then psychosis without motor harm

Parkinsonism is a syndrome, Parkinson's disease is a diagnosis. Parkinsonism is bradykinesia with at least 1 of rest tremor or rigidity.

The syndrome has **4 cardinal features**: resting tremor, rigidity, bradykinesia and postural instability.

- **Key number.** Postural instability is the late feature, typically emerging after 8 or more years, so an early appearance points away from the diagnosis.

Bradykinesia is slowness with progressive reduction in speed and amplitude on repetition, and the decrement is what candidates omit.

Ask for movements as fast and as large as possible, for at least 10 repetitions.

- **Concept.** Finger tapping, fist opening and closing, forearm pronation and supination.
- **Concept.** Toe tapping, the most sensitive lower limb test, and heel tapping with the leg lifted at least 3 inches.

1. Is there parkinsonism, and which features did you demonstrate?
2. Show me the decrement, and show me the Froment manoeuvre.
3. Is it symmetric or asymmetric, and when did postural instability appear?
4. Which absolute exclusion criteria and which red flags did you look for?
5. Is this clinically established or clinically probable disease, and stage it.
6. What motor complications does the drug history suggest, and how would you address them?
7. How would you treat psychosis without worsening the motor state?

CATEGORY	CONTENT
Core	**Bradykinesia with at least 1 of rest tremor or rigidity**
Supportive, need 2 for established disease	Clear sustained dopaminergic response, or more than 30% objective improvement, or marked predictable wearing-off; levodopa-induced dyskinesia; documented limb rest tremor; an ancillary test with over 80% specificity, meaning documented olfactory loss or cardiac sympathetic denervation
Absolute exclusions	**Cerebellar signs; downward supranuclear gaze palsy; probable behavioural variant frontotemporal dementia or progressive aphasia within 5 years; features confined to the lower limbs over 3 years; dopamine blocker or depleting agent exposure; no response to levodopa at 600 mg per day or more; cortical sensory loss, limb ideomotor apraxia or progressive aphasia; normal presynaptic dopaminergic imaging; a known alternative cause**

CATEGORY	CONTENT
Red flags	Wheelchair within 5 years; no progression over 5 or more years; severe bulbar dysfunction within 5 years; inspiratory stridor; severe autonomic failure within 5 years, meaning an orthostatic drop of 30 mm Hg systolic or 15 mm Hg diastolic within 3 minutes; recurrent falls more than once a year within 3 years; disproportionate anterocollis or contractures within 10 years; absent nonmotor features despite 5 years; unexplained pyramidal signs; symmetry throughout
Established against probable	**Established needs 2 supportive criteria and zero red flags, specificity target 90% or more; probable allows no more than 2 red flags counterbalanced one for one, targets 80% or more**

FEATURE	PEAK-DOSE DYSKINESIA	DIPHASIC DYSKINESIA
Timing	**1 to 2 hours after the dose, at peak level**	**Twice: as the drug takes effect and as it wears off**
Phenomenology	Fluid choreiform movement of head, trunk and arms	Stereotypic, often painful dystonic movement, mainly legs
Response to dose	**Worsens with increases, improves with modest reduction**	**Associated with subtherapeutic levels, improves with higher doses**

INDIAN PRACTICE NOTE

Pimavanserin is not marketed in India, which is a practice note rather than a sourced figure. Cite it as the approved reference agent, then say the workable options on an Indian ward are low-dose quetiapine or clozapine, with clozapine under baseline and serial absolute neutrophil count monitoring. First-generation antipsychotics and the potent D2-blocking atypicals can precipitate prolonged akinesia and rigidity crises.

RULE **Cognitive impairment within 1 year of motor onset makes it dementia with Lewy bodies, which is an absolute exclusion.** Dementia arising years after established motor disease is Parkinson's disease dementia, and dementia is not itself an exclusion.

HOLD THIS

Bradykinesia plus one of tremor or rigidity is parkinsonism; established disease needs 2 supportive criteria and zero red flags. Early falls, early autonomic failure, downgaze palsy, cerebellar signs and symmetry are the five things that take the diagnosis away.

WHAT EARNS THE MARKS

- **The decrement demonstrated**: slowness alone is not bradykinesia
- **Established against probable**: two supportive criteria and the red flag count
- **The five take-aways listed**: early falls, autonomic failure, downgaze palsy, cerebellar signs, symmetry
- **Psychosis handled safely**: the reference agent named, then the workable ward option

SEE ALSO 05 Motor system: bulk, tone, power, coordination 08 Cerebellum, gait and the fundus 15 Tremor, chorea and Wilson's disease

15 · Tremor, chorea and Wilson's disease

THE CASES · TOPIC 15

Classify this tremor, and tell me what you would exclude before you name it.

SHORT CASE; MOVEMENT DISORDER VIVA TABLE

THE SHAPE OF THE ANSWER

1 **Activation**	Rest, postural, kinetic or task-specific
2 **Frequency, latency**	Re-emergent after seconds, against immediate on posturing
3 **Drug history**	The agent, the tremor pattern, the timing
4 **Exclusions**	Thyroid, calcium, glucose, liver, and copper when young
5 **The young patient**	Copper studies before any lifelong psychiatric label

Classify tremor by what activates it. Rest tremor occurs when the part is relaxed and fully supported, typically at 3 to 6 Hz, attenuating on action and augmenting on mental distraction.

Action tremor occurs during contraction. It subdivides into postural at 4 to 12 Hz, isometric, kinetic including intention tremor which amplifies towards a target, and task-specific.

FEATURE	PARKINSONIAN REST TREMOR	ESSENTIAL TREMOR
Frequency	3 to 6 Hz	4 to 12 Hz
Symmetry at onset	Unilateral, asymmetric	Bilateral, largely symmetric
Latency on posturing	**Re-emergent after 5 to 10 seconds**	**Immediate, within 1 to 2 seconds**
Tone	Cogwheel and lead-pipe rigidity	Normal
Spiral	Small, tightly constricted, quivery	Large, jagged, wide-amplitude
Alcohol	No significant suppression	**Acute suppression with small doses, 30 to 60 g**
Transporter imaging	Abnormal, 78% sensitivity and 97% specificity	Normal

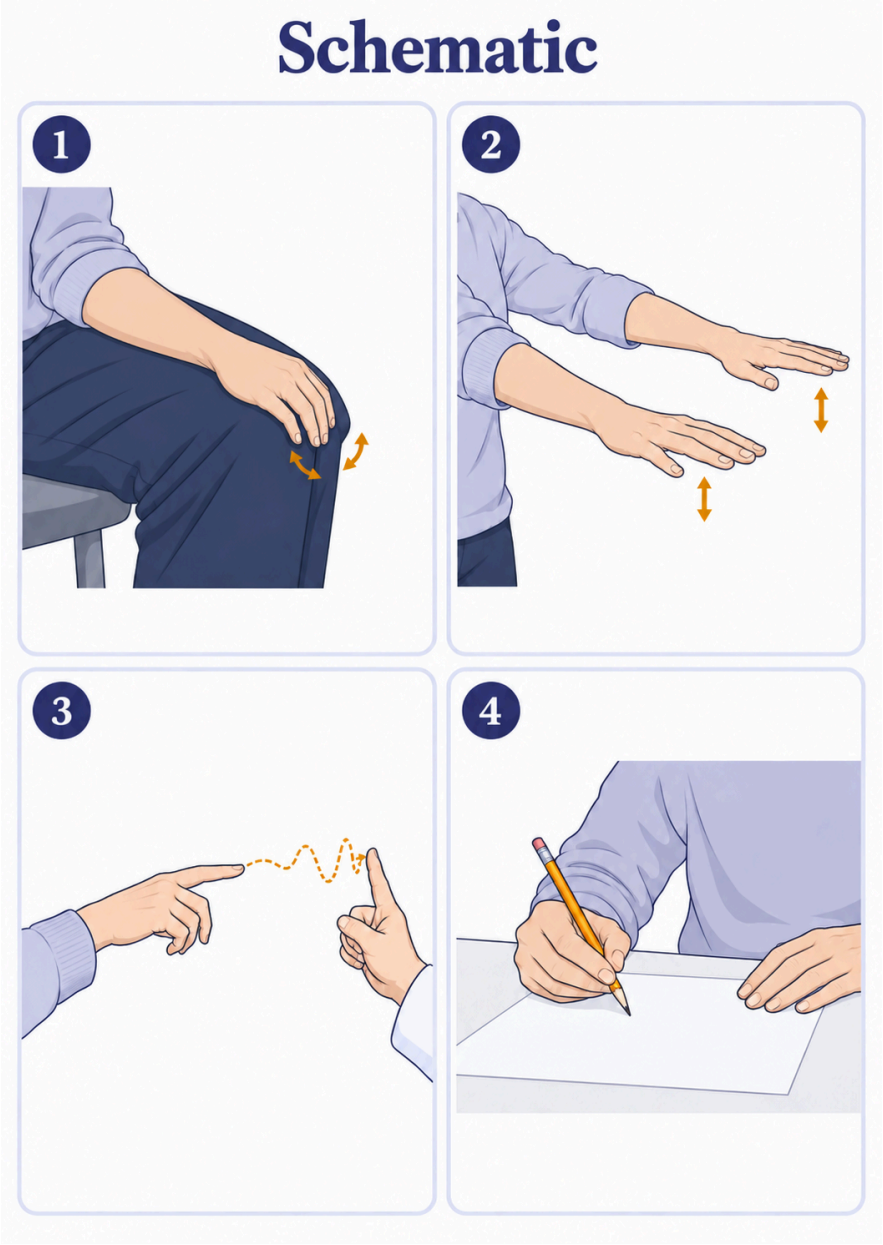
Drug-induced tremor develops within hours to weeks of starting or escalating an agent.

Lithium gives postural, resting and intention tremors; valproate gives postural and resting tremor; serotonin reuptake inhibitors give fine postural tremor; and antipsychotics give a parkinsonian rest tremor at 3 to 6 Hz with cogwheel rigidity, through striatal D2 blockade.

Valproate tremor has its own figures. Postural upper limb tremor in about half of patients, 49% on the right and 48.3% on the left, against 15% and 13.2% on other anticonvulsants.

Tremor: examine the activation condition

Companion to original page 242



Schematic. 1: supported rest. 2: posture against gravity. 3: movement towards a target, illustrating intention as a kinetic subtype. 4: a writing task. The target panel depicts increasing oscillation near a target, while the writing panel depicts a task context. Arrows are conceptual motion cues, not measured waveforms; frequency, latency and diagnostic significance require observation.

Distinguish rest, postural, kinetic and task-specific activation conditions.

Schematic prepared by Luna for Weave Viva, based on P3-23. AI-generated educational illustration.

Specific treatment was required in 23.7% against 5.6%, women scored higher at mean total scores of 14.54 against 9.56 with P = 0.034, and correlation with dose per kilogram or serum concentration was weak or absent.

RULE **Screen out reversible causes before accepting a tremor as essential or drug-induced.** Thyroid function, calcium and parathyroid hormone, glucose and liver function; add ammonia, electrolytes and vitamin B12 where indicated; take a caffeine, alcohol and full medication history in the same sitting; and under 40 years add ceruloplasmin and urinary copper.

DOMAIN	LIFETIME PREVALENCE IN HUNTINGTON DISEASE
Motor features	**Over 99%, with chorea in 96.8%**
Depression	**64.5%, commonest; 58.2% of men against 70.4% of women**
Irritability	Over half, with a male excess and an association with tobacco use
Apathy and cognitive impairment	Each over half; cognition drives functional decline
Psychosis	**Over 11%, least prevalent, negatively associated with years of education**

TRAP Establishing clinical onset of Huntington disease on nonmotor features alone, when depression cannot be separated reliably from primary affective illness or from distress about carrying the mutation, so unequivocal motor signs remain the standard. And starting tetrabenazine without screening for depression, since it depletes presynaptic monoamines and can worsen depression and raise suicide risk. Genetic anticipation and intergenerational repeat instability are reliably examined but are not established in the library behind this note.

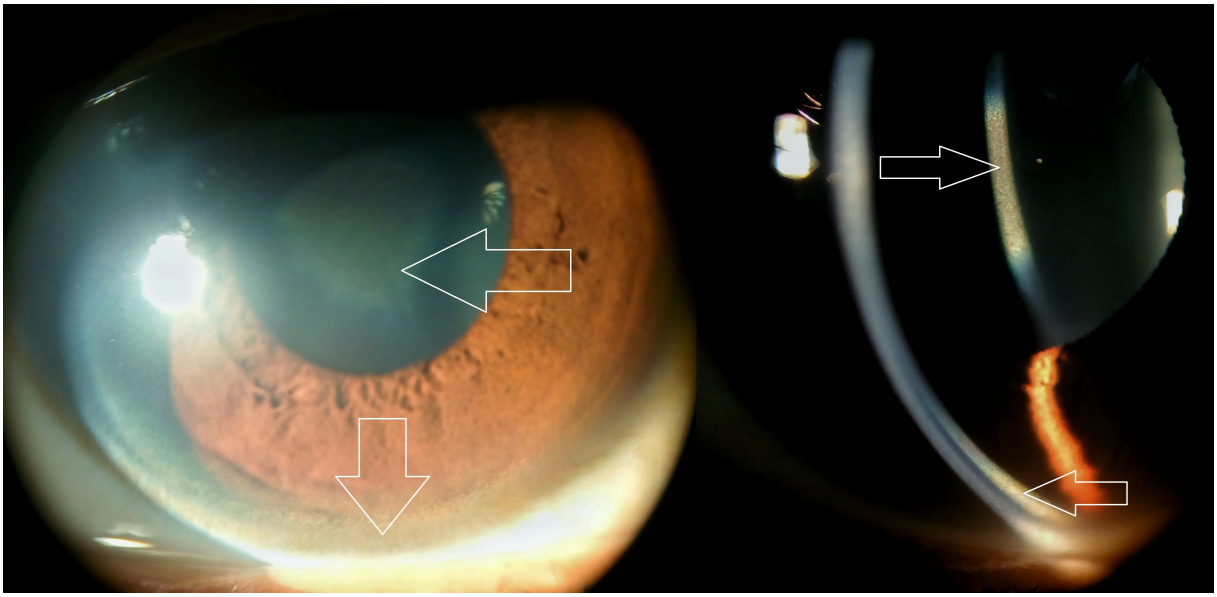
Wilson's disease is autosomal recessive, from variants in ATP7B on chromosome 13q14.3.

Defective biliary excretion causes hepatic copper accumulation, then release of toxic non-ceruloplasmin-bound free copper into brain, corneal Descemet's membrane and kidney. Onset falls between 3 and 55 years in most patients.

The Indian series to quote is 282 patients over three decades, where presentation was neurological in 69.1%, hepatic in 15.9%, purely psychiatric in 2.4% and presymptomatic in 5.3%. In another Indian cohort of 307 patients the average diagnostic delay was 2 years, with prior misdiagnoses of schizophrenia, acute psychosis and rheumatic chorea.

Kayser–Fleischer ring and sunflower cataract

Companion to original page 243



Clinical ocular photographs showing peripheral corneal copper deposition and a sunflower cataract. The source labels the Kayser–Fleischer ring at Descemet membrane and the cataract at the anterior lens capsule. The peripheral corneal ring and the lens opacity are different anatomical findings. The photographs demonstrate ocular signs; they do not establish Wilson disease independently.

Separate the peripheral corneal ring from lens involvement; use the slit illumination in the source.

Imrankabirhossain. KF ring and Sunflower cataract.jpg. CC BY-SA 4.0. https://commons.wikimedia.org/wiki/File:KF_ring_and_Sunflower_cataract.jpg
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TEST	NORMAL	VALUE IN WILSON'S DISEASE
Serum ceruloplasmin	Above 20 mg/dL	**Below 10 mg/dL strongly suggestive; normal in 5% of patients**
24-hour urinary copper	Below 40 mcg per 24 h	**Above 100 mcg per 24 h confirms overload**
Hepatic copper	Below 40 mcg per g dry weight	**Above 250 mcg per g confirms; below 50 mcg per g excludes**
Kayser-Fleischer ring	Absent	**About 95% of neurological presentations, about 50% of hepatic ones**

TRAP Starting D-penicillamine at full dose. Paradoxical neurological worsening occurs in 20% to 40% of patients with pre-existing neurological symptoms in the first months, because rapid mobilisation spikes circulating free copper, and it can reach status dystonicus or mutism. Start low and titrate slowly, or use trientine or zinc as primary therapy, and co-prescribe pyridoxine 25 to 50 mg per day. Siblings carry a 1 in 4 risk and are screened.

HOLD THIS

Under 40 years, psychiatric symptoms plus any movement disorder means slit lamp, ceruloplasmin and 24-hour urinary copper before a lifelong psychiatric label. A normal ceruloplasmin does not exclude it, and an absent ring does not either.

WHAT EARNS THE MARKS

- **Activation class named first**: rest, postural, kinetic or task-specific
- **Re-emergence timed**: it is the bedside test that separates the two common tremors
- **The drug chart read aloud**: agent, pattern and timing together
- **Copper studies in the young**: slit lamp, ceruloplasmin and urinary copper

SEE ALSO 14 Parkinson's disease, and the parkinsonism it is not 05 Motor system: bulk, tone, power, coordination 24 Drugs, substances, pedigree, and the questions they ask

16 · The wasted limb: motor neuron disease and neuropathy

THE CASES · TOPIC 16

This limb is wasted. What are the reflexes doing, and what is the diagnosis?

SHORT CASE; VIVA TABLE ON WASTING AND NEUROPATHY

THE SHAPE OF THE ANSWER

1	**Reflexes first**	Brisk, retained or lost in the wasted muscle
2	**Sensation**	Any loss lifts you off the motor unit
3	**Regions**	Bulbar, cervical, thoracic and lumbosacral involvement
4	**Bulbar or pseudobulbar**	Tongue, gag and jaw jerk decide it
5	**Mimics**	The ones an Indian ward actually shows

A wasted limb asks one question: what are the reflexes doing?

Brisk or retained reflexes in a wasted, weak, fasciculating muscle is the most characteristic sign in motor neuron disease, because a pure lower motor neuron lesion abolishes the reflex while concurrent corticospinal loss removes descending inhibition.

- **Concept.** Lost reflexes with stocking sensory loss: neuropathy.
- **Concept.** Lost reflexes with normal sensation and proximal weakness: myopathy or anterior horn disease.

REGION	UPPER MOTOR NEURON	LOWER MOTOR NEURON
Bulbar	**Brisk jaw jerk, hyperactive gag, spastic dysarthria, pseudobulbar affect**	**Tongue wasting, bilateral tongue fasciculation, palatal paresis**
Cervical	Spastic hypertonia, hyperreflexia, Hoffmann sign	Hand intrinsic and shoulder girdle wasting, fasciculation, cramps
Thoracic	Preserved or hyperactive abdominal reflexes in paretic walls	Paraspinal weakness, paradoxical abdominal motion
Lumbo-sacral	Spastic catch, brisk jerks, clonus, extensor plantar	Foot drop, quadriceps wasting, widespread fasciculation

Bulbar against pseudobulbar palsy is the commonest question here, and it is answered from three findings.

Bulbar palsy is lower motor neuron, from degeneration of the medullary nuclei: a flaccid wasted tongue with undulating fasciculation, palatal paresis with reduced or absent gag, nasal air escape, nasal regurgitation of liquids, and a jaw jerk that is normal or absent.

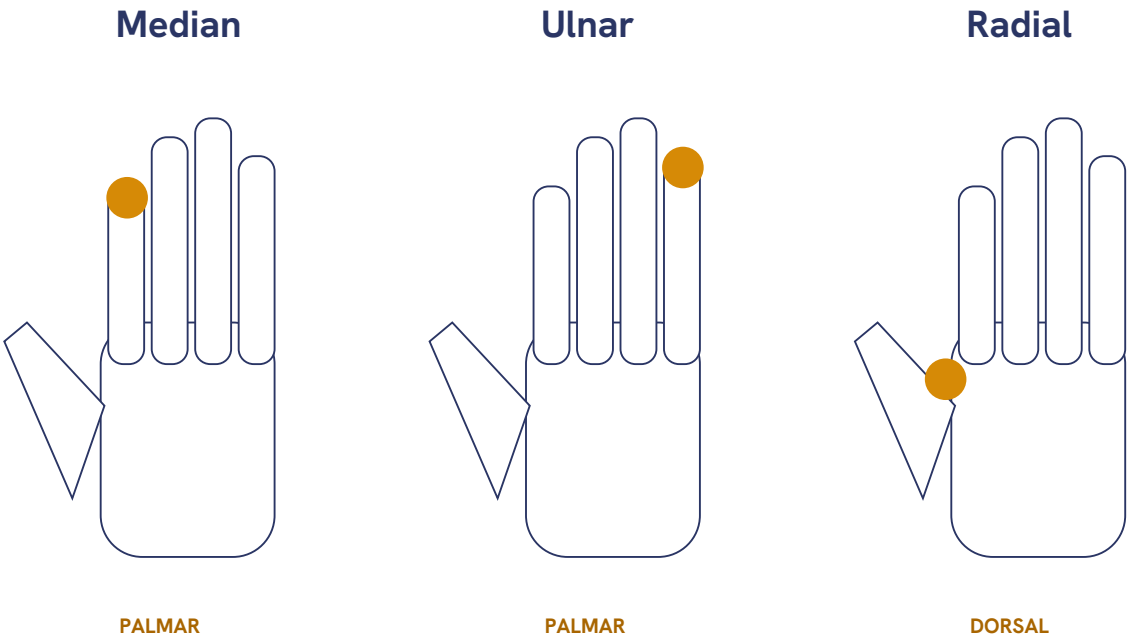
Pseudobulbar palsy is upper motor neuron, from bilateral corticobulbar disruption: a contracted spastic tongue with no atrophy, a brisk jaw jerk, an exaggerated gag, strained slow speech, and pathological laughter and crying.

TRAP In a young Indian man with insidious wasting of one hand and forearm, think Hirayama disease rather than juvenile motor neuron disease, because it stabilises after a few years and flexion imaging shows forward displacement of the posterior dural sac. Other mimics: inclusion body myositis over 50 years with deep finger flexor and quadriceps weakness; post-polio syndrome with stable giant motor unit potentials over 10 mV; Kennedy disease with perioral myokymia; and neurolathyrism from grass pea consumption, endemic in parts of central and eastern India.

PATTERN	CAUSES	BEDSIDE FINDINGS
Mononeuropathy	Entrapment, trauma, ischaemia, leprosy	**Deficit confined to one named nerve**
Mononeuritis multiplex	Vasculitis, diabetes, leprosy, connective tissue disease, HIV	**Asymmetric patchy deficits in multiple nerves**
Distal symmetrical sensorimotor	Diabetes, alcohol, nutritional deficiency, renal disease, drugs	**Glove and stocking loss, distal wasting, early Achilles reflex loss**

Use a small, discriminating sensory zone.

Simplified autonomous test regions; not full nerve territories.



Median: index finger pad

Ulnar: little finger pad

Radial: first dorsal web space

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Hand sensory test regions: palmar index fingertip for median nerve, palmar little fingertip for ulnar nerve and dorsal first web space for superficial radial nerve. These are selected discriminating zones, not drawings of each nerve's whole cutaneous supply. Compare sides and correlate with motor findings.

Do not confuse a nerve territory with a dermatome.

Schematic prepared by Luna for Weave Viva, based on P3-18. Original vector diagram.

PATTERN	CAUSES	BEDSIDE FINDINGS
Symmetrical proximal motor	Guillain-Barre syndrome, chronic inflammatory demyelinating polyradiculoneuropathy, arsenic, porphyria, vincristine	**Girdle weakness with early areflexia**
Autonomic predominant	Amyloidosis, diabetes, paraneoplastic, heavy metals	**Orthostatic hypotension, gastroparesis, anhidrosis**

Diabetic neuropathy affects 25% to 50% of people with diabetes, and remains unidentified on routine assessment in up to 50% of mild presentations.

Large fibres. A 128 Hz fork at the hallux and medial malleolus, and the 10-g monofilament at hallux, metatarsal heads and heel until it buckles for about 1 second. Loss marks ulceration risk, and the combination raises sensitivity and specificity over either test alone.

Small fibres. Pinprick and cold over the dorsum of the foot.

AGENT	PATTERN
Lithium	**Sensorimotor peripheral neuropathy on chronic use**
Amitriptyline	**Peripheral neuropathy even at low doses used for pain**
Disulfiram	**Painful predominantly axonal sensorimotor polyneuropathy**
Phenytoin	**Distal axonal neuropathy with blunted ankle jerks**
Nitrous oxide	**Acute functional B12 deficiency and rapid myeloneuropathy**
Pyridoxine excess	**Chronic intake of 0.2 to 10 g per day gives distal sensory neuropathy over 1 month to 3 years**

TRAP Ordering electrodiagnostic studies reflexively: they are not required for typical chronic symmetrical distal axonal polyneuropathy, having changed management in only 2 of 368 patients in one series. Refer when onset is acute or subacute, the distribution asymmetric or multifocal, weakness predominantly motor, autonomic involvement prominent, or screening unrevealing with progressive disability. Acquired demyelination slows conduction velocity below 75% of the lower limit of normal with conduction block; axonal disease loses amplitude and preserves velocity.

HOLD THIS

Brisk reflexes in a wasted fasciculating limb with normal sensation is motor neuron disease; absent ankle jerks with bilateral extensor plantars is B12. Look at the tongue in the first and check the plantars in the second.

WHAT EARNS THE MARKS

- **The reflexes read before the diagnosis**: they are the discriminator in a wasted limb
- **The tongue examined**: wasting and fasciculation are looked for, not assumed
- **A named neuropathy pattern**: mononeuropathy, multiplex, distal symmetrical, proximal, autonomic
- **Mimics offered unprompted**: Hirayama, inclusion body myositis, post-polio, Kennedy, lathyrism

SEE ALSO 09 Upper versus lower motor neuron, and the mixed picture 10 Spinal cord syndromes 17 Fatigable and flaccid weakness

17 · Fatigable and flaccid weakness: myasthenia, GBS, CIDP

THE CASES · TOPIC 17

How do you demonstrate fatigability, and how do you separate the junction from the nerve?

SHORT CASE; VIVA TABLE ON NEUROMUSCULAR DISEASE

THE SHAPE OF THE ANSWER

1	Elicit fatigue	Four sites, each one timed rather than estimated
2	Confirm	Bedside tests quoted with their accuracy
3	Crisis	Myasthenic against cholinergic, on pupils and secretions
4	Drug list	The psychotropics that worsen transmission
5	Nerve and root	The acute and chronic inflammatory pair, separated on tempo

Fatigability is elicited, not accepted. The junction has a safety margin, since transmission can decline by up to 50% without clinical weakness, and antibody-mediated receptor loss erodes it.

Test in four places. Sustained upgaze without blinking for 1 minute, or for 2 to 3 minutes, where a droop of 1.5 mm or more is specific for ocular myasthenia; the Cogan lid twitch after downgaze held for 10 to 15 seconds; bulbar fatigability on counting aloud; and limb fatigability on sustained abduction or repeated squats.

PROTOCOL	SENSITIVITY	SPECIFICITY	IN MILD PTOSIS
Ice applied at rest	43.3%	No false positives among controls	27.8%
Ice after 2 minutes of sustained upgaze	73.3%	96.7%	72.2%

FEATURE	MYASTHENIC CRISIS	CHOLINERGIC CRISIS
Pupils	Normal or mydriatic	**Constricted**
Secretions	Normal to reduced	**Copious watery bronchorrhoea and salivation**
Autonomic	Tachycardia, diaphoresis, pallor	**Bradycardia, cramps, diarrhoea**
Edrophonium	**Transient improvement**	**Exacerbation**
Action	Ventilate, immunoglobulin or plasma exchange, find the trigger	Stop the anticholinesterase, atropine

CLASS	AGENTS	GUIDANCE
Antipsychotics	Haloperidol, chlorpromazine, clozapine, olanzapine, quetiapine	**Postsynaptic blockade or anticholinergic effect; ptosis, dysarthria and dysphagia overlap with drug side effects, so diagnosis is delayed in schizophrenia**
Mood stabilisers	Lithium	**Impaired presynaptic release and altered ion permeability; avoid rapid escalation**
Anticonvulsants	Carbamazepine, phenytoin, phenobarbital, gabapentin	**Blockade of presynaptic calcium influx or channel modulation**
Sedative-hypnotics	Benzodiazepines, opioids	**Central respiratory depression compounding diaphragmatic weakness**

TRAP The severity classification of myasthenia gravis, and the older staging it replaced, are not established in the library behind this note, so take the class definitions from a standard text. Thymectomy beyond thymoma is in the same position: what is carried is that most patients between adolescence and 60 years are treated surgically whether or not a thymoma is present. Lambert-Eaton syndrome is the mirror image, with hyporeflexia, dry mouth and post-exercise facilitation after 10 to 15 seconds of maximal contraction.

Guillain-Barre syndrome requires 2 cardinal features: progressive motor weakness in more than 1 limb, and generalised areflexia, or distal areflexia with marked proximal hyporeflexia.

Supportive features are progression over days up to 4 weeks, relative symmetry, mild sensory symptoms, bilateral facial weakness, autonomic dysfunction, demyelination in at least 2 nerves, raised cerebrospinal fluid protein with 10 or fewer mononuclear cells per cubic millimetre, and recovery starting within 2 to 4 weeks.

TRAP Two early-normal traps. Cerebrospinal fluid protein is frequently normal in the first 1 to 2 weeks and normal overall in 10% of patients, so quote the threshold as protein above 0.55 g/L with 10 or fewer mononuclear cells and repeat the puncture rather than excluding the diagnosis. Nerve conduction studies can be entirely normal early: in a cohort of 71 patients, 37% studied within 4 days had normal studies, because the inflammation is proximal at the roots.

PARAMETER	GUILLAIN-BARRE SYNDROME	CHRONIC INFLAMMATORY DEMYELINATING POLYRADICULONEUROPATHY
Progression	**Nadir within 4 weeks**	**Progresses or relapses beyond 8 weeks**
Course	Monophasic, relapse in 2% to 3%	Relapsing-remitting or slowly progressive
Autonomic dysfunction	Frequent and severe, approximately 70%	Rare and mild
Corticosteroids	**Ineffective and not indicated**	**First-line maintenance therapy**

INDIAN PRACTICE NOTE

Acute flaccid paralysis in any child under 15 years of age must be notified immediately to the local surveillance officer under the national polio surveillance programme, whatever the presumed diagnosis, with two adequate stool specimens collected and shipped under reverse cold chain.

Candidates who discuss immunoglobulin and omit the notification lose the public-health mark. The exact reporting interval and specimen timing are not established in the library behind this note, so state the duty rather than a deadline.

HOLD THIS

Fatigability with normal sensation and preserved reflexes is the junction; symmetrical ascending areflexic weakness is the root and nerve. Steroids help in the chronic form and not the acute one, and that line separates the two on treatment.

WHAT EARNS THE MARKS

- **Fatigue produced, not asserted**: four sites, timed, with the droop measured
- **The two crises separated**: pupils, secretions and heart rate do it at the bedside
- **The psychotropic list offered**: it is the psychiatry mark in this case
- **Steroids placed correctly**: maintenance in the chronic form, not the acute one

SEE ALSO 16 The wasted limb: motor neuron disease and neuropathy 05 Motor system: bulk, tone, power, coordination 24 Drugs, substances, pedigree, and the questions they ask

18 · Epilepsy short case

THE CASES · TOPIC 18

Classify this patient's seizures, then manage the status epilepticus in front of you.

LONG CASE; SHORT CASE; EPILEPSY VIVA TABLE

THE SHAPE OF THE ANSWER

1 **Three levels**	Seizure type, epilepsy type, then syndrome
2 **Aetiology**	Structural, genetic, infectious, metabolic, immune or unknown
3 **Drug choice**	By seizure type, and by childbearing potential
4 **Status ladder**	Time-banded doses, read off the clock
5 **Counselling**	Driving, machinery, heights, flames, and the record

Classify at three levels and say which level you are at. Seizure type, then epilepsy type meaning focal or generalised or combined or unknown, then epilepsy syndrome, with aetiology evaluated at every stage as structural, genetic, infectious, metabolic, immune or unknown.

The 2017 operational classification sorts seizures by onset into 3 groups, focal, generalised and unknown, with the diagnostic confidence threshold for distinguishing focal from generalised set at 80%.

SEIZURE TYPE OR SUBGROUP	FIRST-LINE MONOTHERAPY	CAUTIONS
Focal onset	**Lamotrigine or levetiracetam**	Levetiracetam with caution in active depression or anxiety
Generalised onset, men and postmenopausal women	**Valproic acid, lamotrigine or levetiracetam**	Carbamazepine and phenytoin worsen myoclonus
Generalised onset, women of childbearing potential	**Lamotrigine or levetiracetam**	Valproic acid contraindicated for teratogenicity and neurodevel-opmental risk
Absence	**Ethosuximide or valproic acid**	Carbamazepine, phenytoin and gabapentin can aggravate absence
Myoclonic	**Clonazepam, valproic acid or levetiracetam**	Phenytoin and carbamazepine exacerbate myoclonic jerks

Status epilepticus is 5 or more minutes of continuous seizure activity, or 2 or more discrete seizures without complete recovery of consciousness between them.

The rationale is physiological. Most convulsive seizures cease within 3 minutes, and by 5 minutes compensatory inhibition is failing through internalisation of synaptic GABA-A receptors. Average mortality is 20%, reaching 57% in adult refractory populations.

1. From 0 to 5 minutes: protect without forceful restraint, position semi-prone head down, high-flow oxygen, wide-bore access, capillary glucose. If hypoglycaemic, 150 to 200 mL of 10%

glucose or 75 mL of 20% glucose. In suspected alcohol misuse or malnutrition, intravenous thiamine 500 mg after the glucose.

2. From 5 to 10 minutes: lorazepam 4 mg intravenously over 2 minutes, repeated once at 4 mg if seizures continue after 5 minutes; without access, buccal midazolam 10 mg repeated once, or intramuscular midazolam 10 mg where trismus prevents buccal delivery. Up to a third of cases are benzodiazepine-resistant.
3. From 15 to 30 minutes: one of three equally effective agents. Levetiracetam 60 mg/kg to a maximum of 4,500 mg over 10 minutes, preferred in polypharmacy. Sodium valproate 40 mg/kg to a maximum of 3,000 mg over 5 minutes, contraindicated in women of childbearing potential and in acute liver disease. Phenytoin 20 mg/kg to a maximum of 2,000 mg at 50 mg per minute, reduced to 25 mg per minute in the elderly or in cardiac disease, under cardiac monitoring.
4. Beyond 30 minutes: intubate and titrate a continuous anaesthetic to burst suppression for a minimum of 24 hours, using propofol 1 to 2 mg/kg then up to 4 mg/kg per hour, midazolam 0.2 mg/kg then 0.05 to 0.5 mg/kg per hour, thiopental 3 to 5 mg/kg then 3 to 5 mg/kg per hour, or ketamine 3 mg/kg then 1 to 10 mg/kg per hour.
5. Super-refractory status continues or recurs 24 hours or more after starting general anaesthesia, and management adds dual maintenance with differing mechanisms, immunomodulation with methylprednisolone 1 g daily for 3 to 5 days or immunoglobulin 0.4 g/kg daily for 5 days or plasma exchange, then magnesium, ketogenic diet, hypothermia, electroconvulsive therapy or emergency resective surgery.

FEATURE	GENERALISED TONIC-CLONIC SEIZURE	CONVULSIVE SYNCOPE	FUNCTIONAL SEIZURE
Prodrome	Focal aura or none	**Lightheadedness, tunnel vision, nausea, pallor**	Often follows emotional distress
Eyes	**Open, often deviated upward**	Closed or drifted open	**Forcefully closed with active resistance**
Motor pattern	**Symmetric tonic extension then synchronous clonus**	Flaccid collapse with brief arrhythmic myoclonus	**Asynchronous flailing, pelvic thrusting, head shaking**
Bite	**Lateral border of the tongue**	Rare	**Tip of tongue or lip**
Recovery	Sustained post-ictal confusion	Clear consciousness within seconds	Prompt recovery or interaction during the event

INDIAN PRACTICE NOTE

Under motor vehicle law an applicant for a licence completes a medical declaration disclosing epilepsy or unheralded attacks of loss of consciousness, and the licensing authority may refuse or revoke a licence where an applicant is medically unfit in a way likely to endanger public safety.

Indian consensus advises a seizure-free interval before private driving, and generally disqualifies commercial and passenger vehicle driving. The statutory form, the section number and the seizure-free duration are not established in the library behind this note, so name the duties and omit the figures.

Criminal law imposes no mandatory duty to report a patient with epilepsy to transport authorities, which makes the record the medico-legal protection: document advice about driving, hazardous machinery, heights and cooking near open flames. Epilepsy has been deleted from the matrimonial statutes that once listed it as a ground for nullity or divorce.

TRAP Calling a focal to bilateral tonic-clonic seizure a secondarily generalised seizure, or a focal impaired awareness seizure a complex partial seizure. The terminology is part of the answer and the examiner is listening for the current words.

HOLD THIS

Five minutes is status, three minutes is what most seizures do on their own, and 4 mg of lorazepam is the dose that prevents refractoriness. Awareness impaired at any point makes a focal seizure focal impaired awareness.

WHAT EARNS THE MARKS

- **The level declared**: say whether you are naming a seizure, a type or a syndrome
- **Current terminology**: focal impaired awareness, and focal to bilateral tonic-clonic
- **The clock quoted**: the ladder is scored on time bands and doses together
- **Childbearing potential addressed**: it changes the first-line choice

SEE ALSO 22 The history frame, and episodic loss of consciousness 19 Headache short case 24 Drugs, substances, pedigree, and the questions they ask

19 · Headache short case

THE CASES · TOPIC 19

This patient has chronic headache. Exclude the secondary causes, then classify it.

LONG CASE; SHORT CASE; HEADACHE VIVA TABLE

THE SHAPE OF THE ANSWER

1 Red flags	Systemic, neurological, onset, age, pattern, position
2 Classify	The primary disorder, by its own criteria
3 Count and time	Attack number, duration, and frequency thresholds
4 Overuse	Two thresholds, and dual coding when both apply
5 Behaviour	The discriminators that need no investigation

Exclude the secondary headache before classifying the primary one. Primary disorders develop without an underlying structural, systemic or exogenous cause, and account for approximately 80% to 90% of outpatient presentations.

The classification has 4 parts: primary headaches in chapters 1 to 4, secondary headaches in chapters 5 to 12, painful cranial neuropathies and facial pain in chapters 13 and 14, and an appendix.

DOMAIN	RED FLAG	POINTS TO
Systemic	Fever, weight loss, night sweats, immunosuppression, pregnancy	**Meningitis, encephalitis, abscess, metastases, vasculitis**
Neurological	Altered sensorium, focal deficit, cranial nerve palsy, pulsatile tinnitus	**Stroke, neoplasm, carcinomatous meningitis**
Onset sudden	Thunderclap onset reaching peak intensity rapidly	**Subarachnoid haemorrhage, reversible cerebral vasoconstriction, dissection**
Older age	New headache or a de novo pattern over the age of 50 years	**Giant cell arteritis, angle-closure glaucoma, intracranial mass**
Progressive pattern	Change in frequency, severity or character over days to weeks	**Expanding mass, venous thrombosis, chronic subdural collection**
Positional and provoked	Worse standing or lying; triggered by Valsalva; papilloedema; onset in pregnancy	**Low cerebrospinal fluid volume, raised pressure, idiopathic intracranial hypertension**

Migraine without aura requires at least 5 attacks lasting 4 to 72 hours untreated, and then two counts have to be satisfied.

At least 2 of 4 pain characteristics: unilateral location; pulsating quality; moderate or severe intensity; aggravation by or avoidance of routine activity such as walking or climbing stairs.

Plus at least 1 of 2 associated features: nausea or vomiting; or photophobia and phonophobia.

RULE **When episodic migraine becomes chronic alongside acute medication overuse, dual coding is mandatory: both chronic migraine and medication-overuse headache are diagnosed.** Examiners penalise either alone. Similarly, when an existing primary headache is made substantially worse, meaning a twofold or greater increase in frequency or severity, by a new causative condition, both diagnoses are assigned.

FEATURE	INFREQUENT EPISODIC TENSION-TYPE	FREQUENT EPISODIC	CHRONIC
Frequency	**Under 1 day per month, fewer than 12 days per year**	**1 to 14 days per month for over 3 months**	**15 or more days per month for over 3 months**
Duration	30 minutes to 7 days	30 minutes to 7 days	Hours to days, or unremitting
Pain, at least 2 of 4	Bilateral; pressing and non-pulsating; mild or moderate; not aggravated by routine activity	The same 4 characteristics	The same 4 characteristics
Associated symptoms	No nausea or vomiting; no more than 1 of photophobia or phonophobia	The same restriction	Mild nausea permitted; no moderate or severe nausea and no vomiting

Cluster headache requires at least 5 attacks of severe strictly unilateral orbital, supraorbital or temporal pain, lasting 15 to 180 minutes untreated.

It also requires at least 1 ipsilateral cranial autonomic feature, among conjunctival injection or lacrimation, nasal congestion or rhinorrhoea, eyelid oedema, forehead sweating, and miosis or ptosis; or else a distinct restlessness during the attack. Frequency runs between 1 every other day and 8 per day.

DRUG CLASS	MONTHLY THRESHOLD	DURATION
Triptans, ergotamine, opioids, combination analgesics	**10 or more days per month**	More than 3 months
Paracetamol, and non-steroidal anti-inflammatory drugs including aspirin	**15 or more days per month**	More than 3 months
Multiple classes, none individually overused	**10 or more days per month in total**	More than 3 months

TRAP Stating that all analgesic overuse requires 15 days per month. The threshold is 10 or more days for triptans, ergotamines, opioids and combination analgesics, and 15 or more only for simple non-opioid analgesics. Comorbidity prevalence figures and prophylaxis dosing are not established in the library behind this note; what is carried is that chronic tension-type headache can be associated with depression although cause and effect are unclear, and that the agents used for migraine prophylaxis include anticonvulsants such as topiramate, valproate and lamotrigine, and tricyclic antidepressants.

COMMON TRAP

Two behavioural discriminators are free marks. During a severe migraine the patient lies still in a quiet dark room because activity aggravates the pain, while during a cluster attack the patient is restless and pacing. And nausea and vomiting are common in migraine, whereas vomiting and moderate or severe nausea are exclusion criteria for tension-type headache.

HOLD THIS

Movement aggravates migraine and does not aggravate tension-type headache; the cluster patient paces while the migraine patient lies still. Chronic daily headache in a patient on acute analgesia is two diagnoses, not one.

WHAT EARNS THE MARKS

- **Red flags by domain**: systemic, neurological, onset, age, pattern, position
- **Criteria counted, not paraphrased**: attacks, duration, and the two of four
- **Dual coding stated**: chronic migraine and medication-overuse headache together
- **The two behavioural signs**: the pacing cluster patient and the still migraine patient

SEE ALSO 18 Epilepsy short case 20 Dementia with focal signs, frontal syndrome 23 Head injury, headache and focal symptoms in the history

20 · Dementia with focal signs, frontal syndrome, multiple sclerosis

THE CASES · TOPIC 20

This patient is demented and has focal signs. What changes in your diagnosis?

LONG CASE; SHORT CASE; DEMENTIA VIVA TABLE

THE SHAPE OF THE ANSWER

1 The focal sign	Which sign it is, and what it argues against
2 The alternatives	Vascular, hydrocephalus, frontotemporal, prion, atypical parkinsonism
3 Imaging markers	Thresholds quoted with their caveats
4 The tap test	What it proves, and what it cannot exclude
5 Frontal syndrome	Tested explicitly, then separated from psychiatric illness

Focal signs in a demented patient change the diagnosis. Alzheimer type dementia presents with insidious cortical amnesia and no early motor deficit, so a focal sign moves you elsewhere.

- **Concept.** Vascular dementia: stepwise deterioration with pyramidal signs.
- **Concept.** Normal pressure hydrocephalus: a higher-level gait disorder, subcortical cognitive impairment, incontinence.
- **Concept.** The frontotemporal spectrum: release reflexes and motor neuron signs.
- **Concept.** Prion disease: myoclonus, ataxia and akinetic mutism.
- **Concept.** An atypical parkinsonian syndrome: early falls or vertical gaze palsy.

MARKER	THRESHOLD	CAVEAT
Evans index	**Above 0.3 defines ventriculomegaly**	20% of cognitively intact adults over 70 years exceed it
Callosal angle	**Acute, below 90 degrees**	Ex-vacuo dilatation gives an obtuse angle
DESH pattern	**Enlarged Sylvian fissures with tight compressed high-convexity sulci**	Atrophy widens rather than compresses those sulci
Radscale	**Total above 4 discriminates shunt-responsive disease**	Composite of ventricular size, angle, periventricular change and DESH

The tap test removes 40 mL of fluid, with standardised gait and cognitive assessment before and at a minimum of 4 hours afterwards, using a timed 10 metre walk in triplicate, where improvement above 10% identifies likely responders.

It is highly specific, with a positive predictive value of 94% in one cohort, but its sensitivity is only 26%.

TRAP Do not say a negative tap test rules out the diagnosis: the 42% figure often quoted beside the sensitivity is not a sensitivity, it is the proportion of test-positive improvers who had had a positive tap test. Do not judge shunt responsiveness on a post-tap cognitive score, because cognition takes months while gait improves within hours. And progressive supranuclear palsy mimics the imaging, so test downgaze saccades, because a vertical supranuclear gaze palsy rules out uncomplicated disease.

REGION	SYNDROME	FEATURES
Dorsolateral prefrontal	**Pseudodepressive**	Apathy, psychomotor slowing, concrete thinking, impaired set-shifting, perseveration
Orbitofrontal	**Pseudopsychopathic, disinhibited**	Disinhibition, tactlessness, punning and jocosity, hyperorality
Medial prefrontal and anterior cingulate	**Apathetic, akinetic**	Abulia, akinetic mutism, emotional indifference, sparse speech

Frontal lobe epilepsy accounts for 20% to 40% of focal epilepsies, and is misdiagnosed as functional seizures, catatonia, panic disorder or behavioural outburst.

The discriminators are countable. Duration is typically not more than 60 seconds and often under 10 seconds, with nocturnal clustering in non-rapid eye movement sleep, and more than 50% of patients experiencing status at some point.

Hypermotor semiology in 11% to 38%, with bicycling, thrashing, pelvic thrusting and screaming; tonic posturing in 32% to 48% and head version in 21% to 60%; rapid recovery without prolonged confusion; and postictal headache in 41% to 53%.

PSYCHIATRIC DIAGNOSIS	WHAT SEPARATES IT
Major depressive disorder	**Pervasive low mood, diurnal variation, worthlessness and guilt, suicidal ideation, executive switching preserved early; the dorsolateral syndrome gives apathetic inertia without depressive thought content**
Bipolar mania	**Sustained elevated mood, grandiosity, flight of ideas, reduced sleep need; orbitofrontal disinhibition gives shallow facetious euphoria and punning without pressured flight of ideas**
Schizophrenia negative symptoms	**Positive features and earlier post-pubertal onset; a frontal lesion presents later with primitive reflexes and no first-rank symptoms**

CRITERION	2017 FRAMEWORK	2024 REVISION
Dissemination in space	**4 sites: periventricular, cortical or juxtacortical, infratentorial, spinal cord**	**5 sites: the optic nerve is added**
Dissemination in time	**Mandatory for single-attack presentations**	**Not mandatory if lesions involve 4 or more of the 5 sites**
Cerebrospinal fluid	Restricted oligoclonal bands only	Bands or a kappa free light chain index above 6.1, treated as equivalent
Advanced imaging	Not incorporated	Central vein sign and paramagnetic rim lesions included
Asymptomatic presentation	Cannot be diagnosed without a clinical event	A radiologically isolated syndrome can be diagnosed with space plus time, fluid positivity, or central vein sign

DIMENSION	PSEUDOBULBAR AFFECT	MAJOR DEPRESSIVE DISORDER
Duration	**Seconds to minutes, paroxysmal**	**Persistent over weeks**
Mood congruence	**Often incongruent, or grossly exaggerated relative to mood**	**Expression matches subjective sadness or guilt**
Vegetative symptoms and suicidality	Absent, though outbursts cause acute distress	Prominent; suicidality common
Substrate	Cortico-ponto-cerebellar disruption and brainstem gating	Fronto-limbic and monoaminergic dysregulation

TRAP Prescribing a full antidepressant regimen for pseudobulbar affect on the assumption that weeping means refractory depression, when it responds to lower doses of serotonergic agents or sigma-1 targeted therapy. The reverse error is dismissing a persistent inter-ictal mood disorder as part of the neurological disease. Which instrument grades pseudobulbar affect, and how interferon-related mood change is separated from disease-driven symptoms, are not established in the library behind this note.

HOLD THIS

Gait, then cognition, then continence, with an acute callosal angle and the DESH pattern. A frontal syndrome survives a normal bedside cognitive screen, so test fluency, sequencing and inhibition explicitly.

WHAT EARNS THE MARKS

- **The focal sign used as evidence**: it is what moves the diagnosis off Alzheimer type
- **Thresholds with caveats**: the Evans index and the callosal angle both need one
- **The tap test read correctly**: high specificity, low sensitivity, gait before cognition
- **Frontal tests named**: fluency, sequencing and inhibition, done out loud

SEE ALSO 02 Higher mental function and lobe testing 08 Cerebellum, gait and the fundus 24 Drugs, substances, pedigree, and the questions they ask

21 · Spot signs: face, pupils, praxis, skin

THE CASES · TOPIC 21

Look at this patient's face and eyes. What is the sign, and what does it mean?

SHORT CASE; OSCE SPOT DIAGNOSIS STATION

THE SHAPE OF THE ANSWER

1 Name the sign	One sentence, in the accepted wording
2 Localise	Nucleus, nerve, junction or pupillary pathway
3 Grade it	The scale or test that changes management
4 Look for company	The findings that shift the diagnosis off the obvious
5 Act	The step that protects this patient today

Some cases are given as a sign, not a story. The forehead decides upper against lower motor neuron facial palsy, because the subnucleus for frontalis and upper orbicularis oculi has bilateral corticobulbar supply.

But a caudal pontine infarct damaging the facial nucleus or its fascicle gives a peripheral pattern, so **look for the company**.

- **Concept.** Contralateral hemiparesis, and an ipsilateral abducens palsy from the proximity of the abducens nucleus and the facial colliculus.
- **Concept.** Trigeminal sensory loss or hearing loss, and cerebellar signs.

GRADE	TERM	FOREHEAD	EYE CLOSURE
I to II	Normal to mild dysfunction	Normal to moderate or good	Complete, with minimal effort at worst
III	Moderate dysfunction	Slight to moderate movement	**Complete with effort**
IV	Moderately severe dysfunction	No movement	**Incomplete**
V to VI	Severe dysfunction to total paralysis	No movement	Incomplete, then absent, with asymmetry at rest

The decisive boundary is between grades III and IV, and it is eyelid closure, because incomplete closure leaves the cornea exposed and mandates lubrication, nocturnal taping and ophthalmological review.

Corticosteroids are the foundation within 72 hours: prednisolone 50 mg daily for 10 days, or 25 mg twice daily for 10 days, or 60 mg daily for 5 days then reducing by 10 mg each day for a further 5 days.

TRAP Three omissions lose this case: not checking the forehead, and so missing a stroke; not performing otoscopy, and so missing concealed vesicles and delaying antiviral therapy; and not palpating the parotid, where a malignancy infiltrating the extratemporal trunk masquerades as a progressive palsy. Steroids started after 72 hours offer minimal proven benefit.

FEATURE	ARGYLL ROBERTSON PUPIL	HOLMES-ADIE TONIC PUPIL
Size and laterality	**Small, irregular, bilateral**	**Initially dilated, unilateral in 80%**
Near response	Preserved, with prompt redilation	**Tonic constriction with prolonged delayed redilation**
Pharmacology	Dilates with atropine	**Constricts to dilute pilocarpine 0.125% within 30 to 60 minutes**
Association	Neurosyphilis and tabes dorsalis	Mostly idiopathic; hyporeflexia in Holmes-Adie syndrome, anhidrosis in Ross syndrome

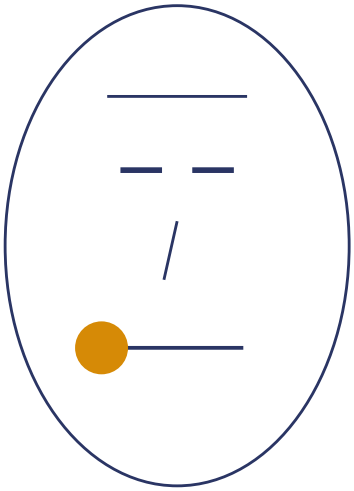
The tonic pupil has a prevalence of 2 per 1000, a female predilection with a ratio of 2.6 to 1, an average onset age of 32 years, and fellow-eye involvement at 4% per year.

The mechanism is aberrant reinnervation after injury to the ciliary ganglion. The ciliary body normally receives 30 times more parasympathetic axons than the iris sphincter, so surviving fibres misroute into the sphincter, and receptor upregulation creates the denervation supersensitivity that the dilute pilocarpine test exploits.

Compare forehead, eye and mouth.

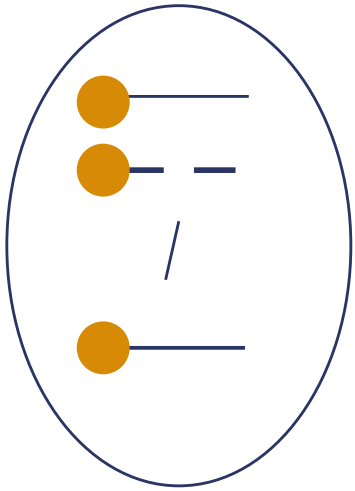
Illustrated weakness is on the patient's RIGHT (page left).

Supranuclear pattern



Forehead relatively spared
Lower face weak

Peripheral VII pattern



Forehead and eye closure weak
Lower face also weak

Amber marks the region to examine; it is not a measured severity score.

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Conceptual facial weakness patterns. A supranuclear lesion commonly causes contralateral lower facial weakness with relative forehead sparing. A peripheral VII lesion affects the ipsilateral upper and lower face. Amber marks weak regions rather than a photograph of asymmetry. Forehead sparing is a localisation clue, not an absolute rule.

Test brow elevation, eye closure and lower facial movement separately.

Schematic prepared by Luna for Weave Viva, based on P3-30. Original vector diagram.

TRAP Mild adduction slowing is missed by 71% of physician observers on smooth pursuit, so test rapid saccades or use an optokinetic drum. Myasthenia gravis simulates the picture as a pseudo-internuclear ophthalmoplegia, discriminated by absent vertical gaze nystagmus on upgaze, fatigable ptosis and a Cogan lid twitch, and no structural lesion. Light-near dissociation is also not pathognomonic of the Argyll Robertson pupil, occurring in tonic pupils, bilateral optic neuropathies, diabetes and dorsal midbrain lesions.

CLINICAL ANCHOR

These signs arrive attached to exposures. Toxic levels of lithium, tricyclics and phenothiazines, and propranolol used for akathisia, are documented causes of drug-induced internuclear ophthalmoplegia and gaze-evoked nystagmus.

Anticholinergic ingestion including Datura gives fixed dilated pupils with tachycardia, delirium and dry skin, and the pilocarpine test is negative in pharmacological mydriasis. Thiamine deficiency gives nystagmus, gaze palsies and internuclear ophthalmoplegia.

Tertiary neurosyphilis presenting as mania or psychosis brings the Argyll Robertson or tonic pupil with it. Central nervous system tuberculoma, common in India, can present with psychiatric symptoms, raised pressure and an internuclear ophthalmoplegia.

TEST	APPERCEPTIVE VISUAL AGNOSIA	ASSOCIATIVE VISUAL AGNOSIA
Matching identical shapes	**Fails**	**Preserved**
Copying line drawings	**Severely impaired**	**Copies intricate drawings accurately**
Naming by sight	Impaired	Impaired
Identification by touch or sound	Promptly recognised	Promptly recognised
Poppelreuter overlapping figures	**Cannot isolate or trace the figures**	**Traces each figure but cannot identify them**

CONDITION	GENETICS AND FREQUENCY	SPOT SIGNS AND CRITERIA	NEUROPSYCHIATRY
Neurofibromatosis type 1	NF1 at 17q11.2 encoding neurofibromin; prevalence 1 in 3,000; about half de novo	**2 or more of 7 criteria: 6 or more cafe-au-lait macules over 5 mm prepubertally or over 15 mm after puberty; axillary or inguinal freckling; 2 or more neurofibromas or 1 plexiform; optic pathway glioma; 2 or more Lisch nodules; a distinctive osseous lesion; an affected first-degree relative**	Learning disorder, visuospatial deficit and attention deficit in at least half

CONDITION	GENETICS AND FREQUENCY	SPOT SIGNS AND CRITERIA	NEUROPSYCHIATRY
Neurofibromatosis type 2	NF2 at 22q1.11 encoding merlin; incidence 1 in 25,000	**Bilateral vestibular schwannomas; juvenile posterior subcapsular cataract; no freckling, cutaneous neurofibromas or Lisch nodules**	Late adolescent onset with tinnitus, hearing loss and disequilibrium
Tuberous sclerosis complex	TSC1 at 9q34 or TSC2 at 16p13.3; 1 in 6,000 to 1 in 9,000; at least half de novo	**2 major criteria, or 1 major with 2 or more minor: 3 or more hypopigmented macules of at least 5 mm, facial angiofibromas, ungual fibromas, shagreen patch, retinal hamartomas, cortical dysplasias, subependymal nodules, giant cell astrocytoma, cardiac rhabdomyoma, renal angiomyolipomas**	Autism spectrum disorder in more than 25%, attention disorder in about half, severe distress in approximately 45% of adults
Fragile X syndrome	CGG expansion in FMR1, expanding across maternal transmissions	**Normal about 5 to 40 repeats, premutation 55 to 200, full mutation over 200; prominent ears, long narrow face, high-arched palate, joint hypermobility, post-pubertal macroorchidism**	Intellectual disability, autism, sensory hypersensitivity, social anxiety; girls milder through X-inactivation
Fetal alcohol spectrum	Prenatal alcohol exposure; an umbrella term rather than one diagnosis	**Cardinal triad of smooth philtrum, thin vermilion border and short palpebral fissures; the neurobehavioural disorder requires more than 13 drinks per month of pregnancy or more than 2 in a single sitting**	Deficits across neurocognition, behavioural regulation and adaptive functioning

TRAP Ordering a chromosomal microarray or routine karyotype for suspected fragile X syndrome, neither of which detects a triplet repeat expansion; a targeted FMR1 repeat analysis is required. Note also that a solitary cafe-au-lait macule is common in healthy people; that an ash-leaf macule is hypopigmented and best seen under an ultraviolet lamp while vitiligo is completely depigmented and acquired; and that angiofibromas appear prepubertally, spare the forehead and lack comedones, unlike acne.

INDIAN PRACTICE NOTE

Intellectual disability, autism spectrum disorder and specific learning disabilities are recognised specified disabilities under disability rights legislation, and a permanent impairment meeting the benchmark threshold entitles the patient to a disability certificate. The benchmark percentage itself is not established in the library behind this note.

Certification is by an authorised board comprising a psychiatrist, a paediatrician or physician and a clinical psychologist, using the Binet-Kamat Test or Malin's Intelligence Scale for Indian Children, the Vineland Social Maturity Scale, and the Indian Scale for Assessment of Autism.

HOLD THIS

Eyelid closure separates facial grade III from IV and decides whether the cornea is at risk. A unilateral dilated pupil with tonic near constriction is Adie; small irregular bilateral pupils with brisk redilation are Argyll Robertson; and recognition that resolves through another sense is agnosia, not anomia.

WHAT EARNS THE MARKS

- **The forehead checked**: it is the sign that keeps a stroke from being missed
- **Otoscopy and the parotid**: concealed vesicles and an infiltrating tumour both hide there
- **The grade boundary used**: eyelid closure is what puts the cornea at risk
- **Pharmacological tests in order**: dilute pilocarpine before any label is attached

SEE ALSO 04 Cranial nerves in exam order 08 Cerebellum, gait and the fundus 12 Hemispheric syndromes

Neurology viva questions

22 · The history frame, and episodic loss of consciousness

VIVA · TOPIC 22

Take a neurological history from this patient who has blackouts.

LONG CASE HISTORY; VIVA TABLE ON BLACKOUTS

THE SHAPE OF THE ANSWER

1 **Three openers**	Age, occupation and handedness, recorded before anything else
2 **The event**	Before, during and after, taken from a witness
3 **Discriminate**	Seizure, syncope, or dissociative event
4 **Tempo**	What the time course says about the pathology
5 **Distribution**	What the symptoms say about the site

In neurology the history outscores the examination. A detailed history is the foundation of neurological diagnosis, and frequently provides more diagnostic value than the physical examination, for both localisation and pathology.

Record three things before any symptom analysis: **age, occupation and handedness**.

Handedness identifies the dominant hemisphere, contralateral to the preferred hand in most individuals. It is what lets you say whether a speech disturbance is a dominant-hemisphere dysphasia, or part of a non-dominant spatial and neglect syndrome.

Episodic loss of consciousness needs a collateral history, because the patient is amnesic for the event.

- **Concept.** Before: precipitants including sleep deprivation, prolonged standing, fasting and alcohol.
- **Concept.** The prodrome: palpitations, diaphoresis and lightheadedness point to cardiovascular or vasovagal syncope.
- **Concept.** A focal aura, such as an epigastric rising sensation or an olfactory hallucination of burning rubber, denotes a localised cortical onset.

FEATURE	GENERALISED TONIC-CLONIC SEIZURE	VASOVAGAL SYNCOPE	DISSOCIATIVE SEIZURE
Onset	Sudden, with or without aura	**Gradual prodrome with nausea, pallor and sweating**	Variable, often after emotional distress
Motor activity	**Synchronous rhythmic clonic jerking**	Flaccid collapse, brief myoclonic jerks possible	**Asynchronous flailing, pelvic thrusting, head thrashing**
Eyes	Open, upward deviation	Closed or drifted open	**Forceful active closure with resistance to opening**
Bite and injury	**Lateral border of the tongue**	Rare bite; trauma from the fall	**Tip of tongue or lip**
Recovery	Prolonged confusion, stertorous breathing, sleep	Rapid reorientation with transient fatigue	Variable, clearing without confusion

- **RULE** **Record handedness in the first line of the history and say why.** Without it you cannot interpret a language disturbance, an apraxia or a neglect syndrome, and the examiner will ask for the reason rather than the fact.

HOLD THIS

Tempo gives the pathology, distribution gives the site, and the witness gives the event. Seconds to minutes is vascular or epileptic; days to weeks is inflammatory or infectious; months to years is degenerative or neoplastic.

WHAT EARNS THE MARKS

- **Handedness with its reason**: the examiner asks for the reason, not the fact
- **A witness account sought**: the patient cannot report the event you are classifying
- **Eyes, motor pattern and bite**: the three findings that separate the three events
- **Tempo mapped to pathology**: seconds, days or years each name a different mechanism

SEE ALSO 03 Speech and language 18 Epilepsy short case
23 Head injury, headache and focal symptoms in the history

23 · Head injury, headache and focal symptoms in the history

VIVA · TOPIC 23

This patient's psychiatric presentation followed a head injury. What do you ask, and why?

LONG CASE HISTORY; VIVA TABLE ON TRAUMA AND HEADACHE

THE SHAPE OF THE ANSWER

1	**Mechanism**	What struck what, and with how much force
2	**Severity indices**	Duration of unconsciousness, then post-traumatic amnesia
3	**Secondary events**	Early seizures, and a lucid interval before deterioration
4	**Headache frame**	SOCRATES, then the primary pattern it fits
5	**Focal symptoms**	Each one localised before any investigation is asked for

Head trauma explains psychiatric presentations that otherwise look primary. Take the mechanism, then the duration of unconsciousness, then post-traumatic amnesia.

Post-traumatic amnesia means both the retrograde loss for events immediately preceding the insult, and the anterograde interval to the return of continuous memory, and it is what indexes severity.

Then the secondary complications: early post-traumatic seizures, and a lucid interval followed by deterioration, which suggests an extradural or acute subdural haematoma.

TRAP Examiners ask for the Glasgow Coma Scale, meaning its three components and the mild, moderate and severe bands, alongside the loss-of-consciousness classification of severity. Neither the component scoring nor the severity cut-offs are established in the library behind this note, and nor are the loss-of-consciousness duration bands, so no figures are printed here. Revise both from a standard reference, and say which scale you are using before you score it.

Headache in the history runs on one framework, remembered as SOCRATES: site, onset, character, radiation, associated symptoms, time course, exacerbating and relieving factors, and severity graded from 0 to 10.

Migraine is recurrent, moderate to severe and throbbing, frequently unilateral, aggravated by routine activity, with nausea and photophobia and phonophobia, returning to baseline within 24 hours.

Tension-type headache is bilateral, band-like and pressing, without systemic features.

Medication-overuse headache is chronic daily headache from regular overuse of acute medication, at 10 or more days per month for triptans, ergotamines, opioids and combination analgesics, against 15 or more days per month for the simple non-opioid analgesics.

INDIAN PRACTICE NOTE

A widely quoted quality standard directs that children under 12 years presenting with headache and red flag symptoms be referred immediately for neurological assessment, issued in January 2021.

It is a United Kingdom standard written for that health service, and no equivalent codified Indian national standard is established in the library behind this note. Cite it as external guidance and say so explicitly, rather than presenting it as Indian policy.

PRESENTATION	SITE	ASSOCIATED FEATURES TO ASK FOR
Monoparesis of arm or face	**Contralateral cerebral cortex**	Field deficit, cortical sensory loss, dysphasia if dominant
Monoparesis of the leg alone	**Contralateral parasagittal cortex, or ipsilateral cord**	Cortical: incontinence and abulia. Spinal: ipsilateral proprioceptive with contralateral thermoalgesic loss
Hemiparesis, face arm and leg equal	**Contralateral internal capsule**	Dense proportional weakness with intact cortical function
Crossed hemiparesis	**Brainstem**	Midbrain third nerve; pontine seventh; medullary Horner, ataxia and contralateral sensory loss
Paraparesis with a sensory level	**Spinal cord**	Trunk cutoff, spasticity, bilateral extensor plantars, early retention

HOLD THIS

Apathy, depression or fluctuating confusion in an elderly or alcohol-dependent patient after a forgotten minor head injury is a chronic subdural haematoma until imaging says otherwise. Ask for Uhthoff and Lhermitte by name, because both are history findings that localise.

WHAT EARNS THE MARKS

- **Amnesia used as the index**: retrograde and anterograde intervals, both asked for
- **The scale named before it is scored**: say which one, then score it
- **The framework used out loud**: SOCRATES keeps the headache history complete
- **Subdural suspected early**: a forgotten minor injury in an at-risk patient

SEE ALSO 13 Hemiparesis and the stroke short case 19 Headache short case 24 Drugs, substances, pedigree, and the questions they ask

24 · Drugs, substances, pedigree, and the questions they ask

VIVA · TOPIC 24

What in the drug, substance and family history changes your neurological diagnosis?

LONG CASE HISTORY; VIVA TABLE ON IATROGENIC NEUROLOGY

THE SHAPE OF THE ANSWER

1 **Drug chart**	Class by class, with the syndrome each one causes
2 **Substances**	What they cause acutely, and on chronic use
3 **Development**	Perinatal events, then motor and language milestones
4 **Pedigree**	The inherited disorders that mimic a drug effect
5 **Answer the stem**	Give the line the examiner is listening for

The drug chart is part of the neurological examination. Read it class by class.

- **Concept.** Antipsychotics give acute dystonia, drug-induced parkinsonism, akathisia and tardive dyskinesia.
- **Concept.** Antidepressants give serotonin syndrome with hyperreflexia and clonus, plus tremor and a lowered seizure threshold.
- **Concept.** Metronidazole produces sensory peripheral neuropathy, and ciprofloxacin can induce seizures.
- **Trap.** Anticoagulants raise the hazard of spontaneous intracerebral haemorrhage, and of chronic subdural haematoma after trivial trauma.

The developmental and family history is where the syndromic diagnoses hide, so ask in four directions.

- **Concept.** Perinatal hypoxia, birth trauma, prematurity and low birth weight.
- **Concept.** Antecedents of cerebral palsy, structural epilepsy and intellectual disability, with the timing of motor and language milestones.
- **Concept.** Past meningitis or encephalitis, predisposing to mesial temporal sclerosis and focal epilepsy.
- **Concept.** Atrial fibrillation, diabetes, chronic kidney disease and vitamin B12 deficiency.

QUESTION	THE LINE THEY ARE LISTENING FOR
Why document handedness at the outset?	**It identifies the dominant hemisphere, contralateral to the preferred hand in most individuals, and so decides whether a speech disturbance is dysphasia or part of a non-dominant spatial syndrome**
How do you differentiate dysarthria from dysphasia?	**Dysarthria is motor with comprehension, grammar and syntax intact; dysphasia disrupts comprehension, word formulation, repetition or naming, and writing exposes it**
What is Uhthoff sign and what does it signify?	**Transient worsening of a deficit on raised body temperature, signifying demyelination through temperature-dependent conduction block**

QUESTION	THE LINE THEY ARE LISTENING FOR
A patient on antipsychotics develops choreiform movements. What else do you ask?	**Family history and psychiatric trajectory, because an inherited neurodegenerative disorder is misattributed to the drug, and the pattern differs from the stereotypic bucco-linguo-masticatory pattern of tardive dyskinesia**

TRAP Two attribution errors recur. Dismissing subacute confusion, psychomotor slowing or fluctuating headache in an alcohol-dependent patient as withdrawal or delirium, and so missing a surgical subdural haematoma. And diagnosing dissociative convulsions or psychiatric agitation in a patient with nocturnal frontal lobe seizures, whose bizarre hypermotor behaviour and brief recovery is the whole presentation.

HOLD THIS

The drug chart, the substance history and the pedigree are three separate examinations, and each has caught a case the bedside missed. Ask them in every neurological history, in that order, before reaching for an investigation.

WHAT EARNS THE MARKS

- **The chart read by class**: the syndrome is named with the agent that caused it
- **The pedigree taken in a movement disorder**: it is what catches the inherited mimic
- **Development asked for in an adult**: perinatal history reframes a late presentation
- **The stem answered in one line**: the examiner is scoring a sentence, not a list

SEE ALSO 15 Tremor, chorea and Wilson's disease 17 Fatigable and flaccid weakness 22 The history frame, and episodic loss of consciousness

Quick review

The whole picture in one table	
THE LADDER	Cortex disproportionate and keeps company; capsule proportionate and alone; brainstem crossed; cord has a level and a normal jaw jerk; junction fatigable; muscle proximal with reflexes preserved.
EXAMINATION ORDER	Attention before cognition. Fluency, comprehension, repetition, with handedness first. Repetition separates perisylvian from transcortical. Forehead separates upper from lower motor neuron facial palsy.
TONE AND REFLEXES	Spasticity velocity-dependent and clasp-knife; rigidity velocity-independent and lead-pipe or cogwheel; paratonia rises with your force. Clonus beyond 5 beats and an extensor plantar make the upper motor neuron case.
SENSORY PATTERNS	Suspended cape sparing the legs is syringomyelia; bilateral thermoalgesic loss with intact vibration is anterior cord; bilateral vibration and position loss is posterior column; preserved vibration in a hemisensory deficit is cortical, not thalamic.
CEREBELLUM AND GAIT	Romberg is not a cerebellar test. Pendular knee jerk beyond 4 swings, and vermal lesions leave finger-nose and heel-shin normal in bed.
CORD AND BRAINSTEM	Brown-Sequard: ipsilateral motor and dorsal column, contralateral pain and temperature 1 to 2 segments below. Wallenberg spares power; Dejerine takes it and deviates the tongue.
STROKE	Proportionate with no cortical sign is a capsular lacune. Ask for aphasia, neglect, a field cut and gaze deviation explicitly.
PARKINSON'S DISEASE	Bradykinesia with decrement plus tremor or rigidity. Established disease needs 2 supportive criteria and no red flags. Early falls, early autonomic failure, downgaze palsy, cerebellar signs and symmetry take it away.
TREMOR AND COPPER	Re-emergent after 5 to 10 seconds is parkinsonian; immediate is essential. Valproate tremor does not track the serum level.
WASTING AND FATIGUE	Brisk reflexes in a wasted fasciculating limb with normal sensation is motor neuron disease. Absent ankle jerks with bilateral extensor plantars is B12.
EPILEPSY	Five minutes is status; lorazepam 4 mg, repeated once; then levetiracetam 60 mg/kg or valproate 40 mg/kg or phenytoin 20 mg/kg. Awareness impaired at any point makes a focal seizure focal impaired awareness.
HEADACHE	Movement aggravates migraine and not tension-type. The cluster patient paces. Overuse thresholds are 10 days for triptans, ergots, opioids and combinations, 15 for simple analgesics, and chronic daily headache on analgesia is two diagnoses.

The whole picture in one table	
DEMENTIA WITH FOCAL SIGNS	Gait, then cognition, then continence, with an acute callosal angle and the DESH pattern. A negative tap test does not exclude it.
SPOT SIGNS	Eyelid closure separates facial grade III from IV. Adie is unilateral, dilated and tonic; Argyll Robertson is small, irregular and bilateral.

HOLD THIS, LAST

Say where before you say what, name the discriminator you used, and name the gap when there is one. Those three habits carry a neurology short case further than any additional fact, because they are what the examiner is scoring.

PART V

V

OSCE and OSPE

Every OSCE station as a script an examiner ticks: opening, content, closing, and the trap.

01 How stations are marked

02 Communication stations

03 Skill stations

04 Professionalism stations

05 Quick review

OSCE and OSPE

Four sections · 21 stations · one complete set of station scripts

A station is not a conversation, it is a rubric being filled in by someone who is watching your hands and your sentences. The candidate who scores is the one whose structure is visible from the examiner's chair: named and introduced, agenda screened, content delivered in chunks with comprehension checked, emotion answered before logistics, plan negotiated, safety net stated, closed on time.

Every script below is built in that order, because that is the order the marks are printed in. Where a station carries a legal duty, the Indian statute is named in an India box, because a foreign section number scores nothing here.

HOW TO USE THIS SET

Each station gives **four things in the same sequence**: the opening line you say out loud, the content the checklist is looking for, the closing that earns the last marks, and the trap that fails candidates who knew the content.

Grids carry the one discriminator the examiner listens for. Where a value could not be sourced, the question is flagged for verification instead of a number being supplied.

How stations are marked

01 · What a station is built from

HOW STATIONS ARE MARKED · TOPIC 01

What is an OSCE station actually made of, and how does the format decide what you are scored on?

OSCE VIVA TABLE, OPENING QUESTION

THE SHAPE OF THE ANSWER

1 **The definition**	A circuit of short standardised tasks, one competency each
2 **Four documents**	Candidate sheet, patient brief, examiner rubric, station setup
3 **Two formats**	Observed with a patient, unobserved with data or a report
4 **What it changes**	Answer the task sentence, not the topic it sits in

Know the machinery and you can predict the marks. An objective structured clinical examination is a circuit of short, standardised tasks, each blueprinted against a competency rather than a topic. Every station is assembled from four documents, and each one tells you something about how you will be judged.

1. **The candidate instruction sheet.** Clinical stem, patient demographics, the setting (outpatient clinic, emergency service, consultation-liaison ward), the single task, and the time, typically 5 to 10 minutes.

2. **The standardised patient brief.** History, presenting affect, the exact opening statement, disclosures that are released only on open questioning, a hidden concern, and boundary rules such as becoming anxious if interrupted, or admitting suicidal intent only on direct and empathic inquiry.
3. **The examiner rubric.** An itemised checklist of observable actions, a global performance scale, and designated critical fail criteria, for example failing to assess immediate lethal intent in a severe depressive episode.
4. **The setup.** Table, privacy screen, props, prescription charts.

Two station formats run in the same circuit, and the format tells you which competency is being sampled.

- **Concept.** In an **observed station** you interact with a simulated patient, role-player or caregiver while the examiner scores silently, in the room or behind a one-way mirror, without intervening.
- **Concept.** That format samples interview technique, mental state examination, risk assessment, diagnostic disclosure and shared management planning.
- **Concept.** In an **unobserved station** there is no patient. You read an electroencephalogram or a scan, audit a psychotropic chart, formulate a diagnosis from a written vignette, or draft a statutory report.
- **Concept.** The two are often **paired**: an observed station followed by a linked unobserved one in which you write post-encounter orders or defend the formulation in a short structured viva.

RULE **The hidden disclosure is the station.** The patient brief is written so that the decisive fact appears only on an open, empathic question. A closed symptom checklist walks past it, and the critical fail criterion is usually attached to it.

TRAP Treating the instruction sheet as a topic rather than a task. "Counsel this patient on lithium" is marked on counselling behaviours, not on a recital of pharmacology; content earns its marks only as it is delivered, checked and negotiated.

HOLD THIS

Four documents, two formats, one task per station. Read the task sentence twice, name the setting, and answer the task that is written rather than the topic it belongs to.

WHAT EARNS THE MARKS

- **Names the four documents**: it shows you know where the marks are printed.
- **Separates observed from unobserved**: each format samples a different competency.
- **Restates the task in your own words**: the rubric scores the written task, not the subject.
- **Asks the open question that releases the hidden fact**: the critical fail is usually attached to it.

SEE ALSO 02 What the checklist is actually scoring 03 Calgary-Cambridge and Kalamazoo, and the marks inside them 04 Reliability, the pass mark, and the ten-minute clock

02 · What the checklist is actually scoring

HOW STATIONS ARE MARKED · TOPIC 02

In a psychiatry station, are the marks for communication or for clinical content?

OSCE VIVA TABLE, EXAMINER CALIBRATION QUESTION

79 PERCENT OF ITEMS ARE COMMUNICATION ITEMS

THE SHAPE OF THE ANSWER

1 The finding	Psychiatry checklists are mostly communication items
2 The twist	Those communication items mostly score mastery of content
3 The discriminating row	Mastery section mean 89 percent against 51 percent
4 What it changes	Say the formulation out loud, in the patient's words

Communication is marked through content, not instead of it. The commonest misconception is that a psychiatry station rewards manner while the knowledge marks sit elsewhere. Calibration of interdepartmental circuits against the 17-item MAAS-Global standard shows the opposite: psychiatry checklists are mostly communication items, and those communication items are mostly about mastery of clinical content.

The calibration studied 380 candidates across 3 academic terms, and divided clinical communication into three sections.

- Key number. **Consultation phases**, 7 sequential items.
- Key number. **General communication skills** across the consultation, 6 items.
- Key number. **Mastery of medical content** during the consultation, 4 items.

DIMENSION	PSYCHIATRY STATIONS	GENERAL PRACTICE STATIONS
Communication items overall	79%	69%
Technical and other non-communication items	21%	31%
Section 1, consultation phases	1% to 16% (mean 6%)	11% to 19% (mean 15%)
Section 2, general communication skills	0% to 7% (mean 5%)	29% to 35% (mean 31%)
Section 3, mastery of medical content	77% to 96% (mean 89%)	41% to 57% (mean 51%)
Station duration	10 minutes	5 minutes
Reviewer calibration agreement (kappa)	0.99	0.83

Table: what a psychiatry checklist is made of. The discriminating row is the third section, because it says the communication marks are awarded for how well clinical substance is conveyed.

Within the phase items, psychiatry checklists draw almost exclusively on **item 5, diagnosis**, and within the general skills items on item 10, information giving.

- **Concept.** So the two behaviours most reliably present on the sheet in front of your examiner are: **did the candidate give the diagnostic picture**, and did the candidate give information the patient could use.

- **RULE** **Say the substance out loud, in the patient's words.** An unspoken formulation earns nothing. The examiner marks the explanation you gave, checked and summarised, not the reasoning you kept to yourself.

HOLD THIS

Psychiatry stations are communication stations whose communication items measure content. Warmth without substance and substance without delivery both lose the same marks.

WHAT EARNS THE MARKS

- **Quotes the mastery-of-content row**: it is the row that answers the question asked.
- **Names item 5 diagnosis and item 10 information giving**: those two behaviours sit on almost every sheet.
- **Gives the explanation aloud and checks it**: an unspoken formulation earns nothing.
- **Avoids warmth without substance**: both halves lose the same marks.

SEE ALSO 01 What a station is built from 03 Calgary-Cambridge and Kalamazoo, and the marks inside them 04 Reliability, the pass mark, and the ten-minute clock

03 · Calgary-Cambridge and Kalamazoo, and the marks inside them

HOW STATIONS ARE MARKED · TOPIC 03

Which frameworks do communication rubrics come from, and what does each one contribute?

COMMUNICATION STATION VIVA, OSCE TABLE

THE SHAPE OF THE ANSWER

1	Calgary-Cambridge	Five sequential stages on a continuous relationship thread
2	The illness framework	Ideas, concerns, expectations and effects, with the emotion named
3	Kalamazoo	Seven essential tasks, which are the rubric lines themselves
4	The three instruments	Original checklist, adapted Likert version, multi-rater gap version
5	Use it aloud	Signpost each transition so the examiner can tick

Two frameworks, one behaviour set. Rubrics in this territory descend from the Calgary-Cambridge guide, which gives the consultation its spine, and the Kalamazoo consensus statement, which gives the itemised tasks. Learn both as checklists you can hear yourself completing.

Calgary-Cambridge, formulated by Kurtz, Silverman and Draper, sets out 5 sequential stages held together by a continuous relationship-building thread: initiating the session, gathering information, providing structure, explanation and planning, and closing the session.

1. **Initiating.** Rapport, your name and role, consent, physical comfort, and the reason for the consultation.
2. **Gathering.** The disease framework (symptoms, signs, diagnostic criteria) and the illness framework in parallel.
3. **Providing structure.** Overt signposting, periodic summary, attention to pace and logical flow.
4. **Explanation and planning.** Information in digestible increments, comprehension checked, the patient's view incorporated, plan negotiated.
5. **Closing.** Decisions summarised, a collaborative contract for next steps, contingency safety net, mutual understanding confirmed.

The illness framework is elicited as **ICE plus feelings**: *ideas* about origin and nature (somatic weakness, a spiritual crisis), *concerns* that generate the anxiety (admission, dependence on drugs, loss of employment), *expectations* of this consultation (sleep tonight, a certificate, validation), and *effects* on relationships, work and routine with the underlying emotion named out loud.

The Kalamazoo consensus statement was formulated in 1999 by 21 educational leaders and published in 2001. It defines 7 essential communication tasks, and they map directly onto rubric lines.

KALAMAZOO TASK	WHAT THE EXAMINER IS WAITING TO HEAR	DISCRIMINATOR
Builds a relationship	Respect and interest in the person; supportive statements; eye contact, posture, tone and pace aligned	**Empathy spoken aloud, not merely felt**
Opens the discussion	The opening statement heard without interruption, the full agenda elicited, priorities negotiated	**"Is there anything else?" asked explicitly**
Gathers information	Open prompts first, then focused clarification, with periodic summary offered back	**The cone: open to closed, not closed throughout**
Understands the perspective	Life events, social context, beliefs, concerns, expectations, values acknowledged	**The patient's idea reappears in your plan**
Shares information	Baseline knowledge and desired detail established; plain words; chunk and check; questions invited	**Comprehension probed after each chunk**
Reaches agreement	Choice offered to the degree wanted, adherence barriers explored, resources identified	**"What might make this hard to take regularly?"**
Provides closure	Decisions summarised, outstanding concerns checked, follow-up and emergency contingency specified	**A named safety net with a named contact**

The three instruments the viva asks about. Name which one you are describing, because the tasks are the same and the scoring is not.

- **Key number.** The **original checklist** scores the 7 competencies across 24 sub-competencies on 4 categorical options, done well, needs improvement, not done, not applicable, with internal consistency of 0.88.
- **Key number.** The **adapted version** keeps the same 7 competencies and 24 sub-competencies, rated on a 5-point Likert scale from poor to excellent, which gives finer gradation.
- **Key number.** The **gap version** is built for multi-rater use, with parallel forms for faculty, patients and families drafted at a sixth-grade reading level, peer facilitators and self.
- **Key number.** It expands to 9 dimensions by adding demonstrates empathy and communicates accurate information, with Likert ratings plus 2 forced-choice items and 2 free-text boxes, and a discrepancy analysis between self and observer ratings.

INSTRUMENT	DIMENSIONS	SCORING	RELIABILITY	DISCRIMINATOR
Kalamazoo essential elements checklist	7 competencies, 24 sub-competencies	4 categorical options	Cronbach alpha 0.88	**Categorical, single faculty rater**
Adapted checklist	7 competencies, 24 sub-competencies	5-point Likert, 1 poor to 5 excellent	Good internal consistency	**Graded rating, same 7 tasks**
Gap version	9 dimensions, adding empathy and accurate information	Likert plus 2 forced-choice and 2 free-text	Alpha 0.844 to 0.880; ICC 0.830 to 0.89 across 118 conversations	**360-degree, self versus observer gap analysis**

TRAP Asking about ideas and concerns as a formula and then ignoring the answer. The distinction between a pass and a merit is that the superior candidate builds the patient's stated idea and concern into the options proposed, so the plan visibly belongs to the patient.

MNEMONIC

ROGUSAC

The seven Kalamazoo tasks in order: **R**elationship, **O**pen the discussion, **G**ather, **U**nderstand the perspective, **S**hare, **A**gree, **C**lose. If you can say the seven, you can narrate any communication station out loud while you do it.

HOLD THIS

Calgary-Cambridge gives the five stages, Kalamazoo gives the seven tasks, and the rubric is a list of the seven. Signpost each transition and the examiner can tick without guessing.

WHAT EARNS THE MARKS

- **Says the seven tasks in order**: the rubric in front of the examiner is that list.
- **Builds the patient's stated idea into the plan**: it separates a pass from a merit.
- **Names which instrument you are describing**: the three differ in scoring, not in tasks.
- **Signposts each transition out loud**: a tick the examiner has to guess at is a tick lost.

SEE ALSO 02 What the checklist is actually scoring 04 Reliability, the pass mark, and the ten-minute clock 12 Behaviour change and explaining a therapy

04 · Reliability, the pass mark, and the ten-minute clock

HOW STATIONS ARE MARKED · TOPIC 04

How reliable is an OSCE circuit, and where does the pass mark come from?

OSCE VIVA TABLE, ASSESSMENT THEORY QUESTION

THE SHAPE OF THE ANSWER

1	**Generalisability**	Reliability rises with more stations, not longer ones
2	**The numbers**	0.54 to 0.73 at four or five stations, higher at fifteen
3	**Standard setting**	Borderline regression on examiner global ratings sets the cut

4 The clock

Four phases in order, with closure protected at the end

Why circuits are long and cut-offs are not round numbers. Station scores are only as dependable as the sampling, and the pass mark is derived from examiner behaviour rather than chosen in advance. Both facts are examinable and both explain the format you will sit.

Generalisability theory measures how dependably a circuit ranks candidates.

- **Key number.** Across psychiatry stations of 10 minutes duration, a circuit of 4 to 5 stations produced a G-coefficient between 0.54 and 0.73.
- **Key number.** Decision studies lifted that to 0.72-0.84 at 10 stations, and to 0.79-0.89 at 15 stations.
- **Concept.** **Reliability rises with the number of stations**, not with the length of any one station.

Standard setting. Modern circuits do not impose a fixed pass mark. They use the **borderline regression method**: total checklist scores are regressed on the examiners' global ratings, clear fail, borderline, clear pass.

The cut-score is the total predicted for a candidate performing at the borderline standard. So the pass mark adjusts for station difficulty and for examiner stringency, and a station a whole cohort found hard moves its own cut-off down.

> **WHY THIS MATTERS AT THE TABLE**
>
> Because the global rating drives the cut-score, the impression you leave on the examiner's overall judgment is not decoration on top of the checklist, it is the variable the checklist is calibrated against. Visible structure, spoken empathy and an explicit close move the global rating and therefore the mark.

The clock. The station runs to a single countdown, and the primary source for an Indian circuit gives the working window as an 8- to 10-minute station. Per-phase minute allocations are not sourced, so rehearse the *order* of phases and keep an eye on the clock rather than memorising minute cut-offs.

PHASE, IN ORDER	TASKS THAT CARRY MARKS	FRAMEWORK ALIGNMENT
Open	Greet, name and role, verify identity, consent, physical comfort. Take the opening statement without interrupting. Screen the agenda: "Is there anything else?" Negotiate the focus.	**Initiating; builds relationship, opens discussion**
Gather	Open to closed cone. Symptoms, psychopathology, baseline functioning, risk. Elicit ideas, concerns and expectations. Offer an interim summary.	**Gathering (disease and illness); understands perspective**
Share	Diagnostic picture in plain words after checking baseline knowledge. Options without jargon. Chunk and check. Acknowledge the emotional reaction.	**Providing structure, explanation and planning; shares information**
Agree and close	Negotiate the plan around the patient's preferences, name adherence barriers, summarise, state the safety net and emergency contact, invite final questions.	**Closing; reaches agreement, provides closure**

The micro-behaviours that are literally on the sheet. Six of them, each one a line the examiner can tick.

- **Concept. Facilitation**: nodding, forward lean, brief continuers such as "go on".
- **Concept.** An **uninterrupted first minute**, then the explicit agenda screen.
- **Concept. Overt signposting**: "now that we have talked about your mood, I would like to ask about sleep and physical health".
- **Concept.** An **empathic statement out loud** when distress appears: "I can see how overwhelming it has been to manage this alone".
- **Concept. Periodic summary**: "let me check I have understood so far".
- **Concept. Chunk and check**: "how does this plan sound to you so far?".

FAILURE MODE	WHAT IT LOOKS LIKE	WHAT CONVERTS IT TO A MARK
Interrogator	Rapid closed criteria, no eye contact, cues missed	**Open prompt, then name the emotion you see**
Premature advice	Psychoeducation in the first minutes, before beliefs are known	**Ask what they already know, then share**
Jargon	Anhedonia, affective lability, neurotransmitter imbalance	**Plain phrase plus a comprehension check**
Ignored agenda	Only the opening complaint addressed	**"Is there anything else?" before focusing**
Monologue	Uninterrupted explanation of the plan	**Chunk, check, invite a question**
Abandoned closure	Time expires deep in history	**Reserve the last stretch for summary and safety net**

TRAP Running out of clock inside the history. The closure items (summary, negotiated plan, follow-up, emergency contingency) are discrete checklist lines; a candidate who does not reach them forfeits them regardless of how good the history was.

HOLD THIS

Reliability comes from more stations, and the pass mark comes from the borderline regression against examiner global ratings. Protect the last phase of the clock: closure marks are the easiest marks in the circuit to lose.

WHAT EARNS THE MARKS

- **Says reliability comes from station number**: it is the single fact this question tests.
- **Explains borderline regression in one sentence**: it shows the cut-score is derived, not chosen.
- **Links the global rating to the cut-score**: visible structure moves the mark through it.
- **Reserves the last stretch for closure**: those are discrete lines and the easiest to forfeit.

SEE ALSO 01 What a station is built from 02 What the checklist is actually scoring 03 Calgary-Cambridge and Kalamazoo, and the marks inside them

Communication stations

05 · Breaking bad news, and the dementia family meeting

COMMUNICATION STATIONS · TOPIC 05

Break the diagnosis of dementia to this patient and her son, and answer their questions.

COMMUNICATION STATION, SIMULATED PATIENT WITH A RELATIVE

THE SHAPE OF THE ANSWER

1 **Set up, then ask** Privacy, who should be in the room, what they understand

2 Invitation and knowledge	Permission, warning shot, small chunks, plain words
3 Emotions	Name the emotion and its reason, then wait
4 Strategy	Concrete plan, carer support, safety, legal planning
5 Driving	Open it early, on-road assessment, no co-piloting

Open with the ask, not the news. Bad news is any information that adversely and seriously affects a person's view of their future.

Its impact sits **in the eye of the beholder**: the same result relieves someone who feared imminent death and devastates someone who expected a benign answer. The station is marked on a protocol, so name the protocol and walk it.

The interview has four objectives that the six steps deliver: gather information about what the person already knows and can bear, transmit facts at their level of comprehension, provide support that reduces isolation, and elicit collaboration on a plan.

STEP	WHAT YOU DO AND SAY	DISCRIMINATOR THE EXAMINER LISTENS FOR
S: setting up	Privacy or curtains drawn. Ask whom they want present; with a large family ask the patient to choose one or two representatives. Sit down with no desk between you. Silence the phone. Tissues on the table before you start.	**Sitting, and asking who should be in the room**
P: perception	"Before you tell, ask." "What have you been told about your condition so far?" "What is your understanding of why we did the scan?" Note misinformation, denial and unrealistic expectation without confronting them.	**No diagnostic label spoken before this step**
I: invitation	"Would you like me to go through the full details of the results, or would you prefer the overall picture while we focus on the plan?" Respect a wish not to know and offer to speak to a named relative instead.	**Permission asked, not assumed**
K: knowledge	Warning shot first: "Unfortunately the results show something more serious than we had hoped." Plain words (memory difficulty, shrinkage) not radiological jargon. Small chunks, pause, "does this make sense so far?" Name what is preserved and what can be treated.	**Warning shot, then chunk and check**

STEP	WHAT YOU DO AND SAY	DISCRIMINATOR THE EXAMINER LISTENS FOR
E: emotions	The four-step empathic response: observe the emotion, name it internally (or ask "could you tell me what is going through your mind?"), identify its reason, then make the connecting statement: "I can see how upsetting this is when you were hoping the lapses were just tiredness." Validate, then wait.	**Information stops until distress settles**
S: strategy and summary	Check readiness for next steps. Summarise. Check for misunderstanding. Follow-up appointment, staging investigations or baseline bloods, written leaflet, multidisciplinary supports.	**A concrete plan reduces the anxiety you just created**

Dementia is disclosed as a triad, not a dyad. Ordinary disclosure assumes a cognitively intact patient. A cognitive diagnosis needs three people in the room.

- **Concept.** The **care partner** is part of the unit: they give the collateral, provide the containment, and carry out the plan.
- **Concept.** Disclose the **syndrome and its stage first**, and the suspected pathology second.
- **Concept.** Calibrate depth and pace to cognitive capacity and emotional stamina.
- **Concept.** Disclose **early enough to plan**, while the person can still take part in legal and financial decisions and advance decisions.

> **CLINICAL ANCHOR**
>
> Under-disclosure is the norm rather than the exception: fewer than 50% of people with mild cognitive impairment are told their diagnosis, usually because of therapeutic nihilism or fear of causing distress. Say the words, then hold the room while the distress happens.

When deferral is defensible. Disclosure is the default once the assessment concludes. Withholding or deferring is justifiable in four situations.

- **Concept.** An **acute exacerbation** of severe depression, panic or active suicidal ideation triggered by disclosure. A stable mood disorder is not by itself a contraindication.
- **Concept.** An **explicit wish not to know**.
- **Concept.** **Advanced impairment or frailty** where comprehension has gone and disclosure produces intense distress without altering management.
- **Trap.** A **family request to conceal** is not simply agreed to. Explore their fears, assess the patient's capacity, and aim at supported, graded disclosure.

RULE **Screen for depression and suicide risk as part of disclosure in any chronic, life-limiting illness.** Separate expected, fluctuating existential grief from a depressive syndrome; treat the syndrome actively with medication and supportive psychotherapy alongside symptom relief.

The relative's agenda in the dementia station. Once the diagnosis lands, the checklist moves to safety, care and driving. Separate **anosognosia** (a neurobiological failure of self-monitoring from frontoparietal pathology) from **psychological denial** (an active defence against anxiety, with awareness retained underneath). Confronting anosognosia with reality checks produces agitation, paranoia and catastrophic reactions, not insight.

DOMAIN	WHAT YOU ADVISE THE RELATIVE	DISCRIMINATOR
Home safety	At least 1 sturdy handrail on every flight, non-skid strips and high-contrast tape on step edges, a sturdy shower chair, rugs and cords cleared, bright glare-free lighting, busy geometric patterns removed, socket plugs and cabinet latches, chemicals and matches locked away.	**Patterned carpets and curtains provoke misperception**
Communication	Short sentences, gentle eye level, binary choices, state your identity calmly, do tasks together step by step.	**Do not quiz: "don't you remember me?" is a harm**
Behaviour	Treat agitation, shouting, pacing, shadowing and repeated questions as unmet needs. Check pain, thirst, hunger, constipation, infection and fatigue, then noise, cold, overload and boredom, before any drug.	**Physical causes screened before prescribing**
Carer	Respite and a family rota, a daily micro-break, sleep and nutrition for the carer, support groups and formal therapy when distress surfaces.	**The carer is screened, not just advised**

Driving. Start the conversation early, while capacity is intact and the room is calm, not after a collision. Mild dementia does not by itself mean immediate revocation, but progression makes cessation inevitable, so plan for it.

- **Concept. Warning signs on the vehicle**: new dents and scrapes, near misses, hitting curbs, 2 or more traffic tickets, rising insurance premiums.
- **Concept. Warning signs in control**: confusing brake and accelerator, drifting across lanes, inappropriate speed, delayed braking.
- **Concept. Warning signs in navigation**: getting lost on familiar routes, missing signals, and passengers who no longer feel safe.
- **Key number.** The standard of assessment is an **on-road test** by an occupational therapy driving rehabilitation specialist, with off-road cognitive and visual screening first.
- **Key number.** If driving continues, review at least 6-monthly, and run the medical review every 6 months or sooner.

TRAP Offering a relative as a co-pilot. Verbal navigation from the passenger seat is not recognised safe practice: it adds dual-task interference and cannot prevent pedal confusion or a split-second steering error. Say so explicitly, because the examiner is listening for it.

TRAP Reaching for key confiscation first. Work the ladder: the agreed driving contract, then a written "do not drive" instruction on your letterhead so the family can deflect hostility onto the medical order, then trusted family elders, and only then physical measures such as hiding keys or disabling the car, reserved for severely lost insight with imminent public danger. Pair every step with alternative transport so the person is not isolated.

INDIA CONTEXT

Legal planning belongs in this station: an advance directive and a nominated representative under the Mental Healthcare Act 2017 (the sections are set out in the lithium and the consent topics), testamentary capacity for a will, joint accounts with an either-or-survivor mandate, and limited guardianship under the disability legislation.

TRAP The examiner will ask for the exact statute year and section for the will, for guardianship, and for the motor vehicle medical fitness form. The library marks those citations as unsourced, so verify them against the primary statute before the exam rather than quoting a number here.

HOLD THIS

Before you tell, ask; then warning shot, small chunks, and the connecting statement before any logistics. In dementia the unit of disclosure is the triad, and the driving conversation starts on the day of diagnosis.

WHAT EARNS THE MARKS

- **Asks before telling**: no diagnostic label is spoken before their understanding is known.
- **Gives a warning shot, then chunks and checks**: it is the scored shape of the disclosure.
- **Makes a connecting statement before logistics**: information stops until the distress settles.
- **Treats the care partner as part of the unit**: a cognitive diagnosis is disclosed as a triad.

SEE ALSO 06 Explaining schizophrenia, and expressed emotion 13 Consent and capacity as a bedside skill 15 Cognitive testing at the bedside

06 · Explaining schizophrenia, and expressed emotion

COMMUNICATION STATIONS · TOPIC 06

Explain this young man's schizophrenia to his family and answer their questions about relapse.

COMMUNICATION STATION WITH AN EXTENDED FAMILY

THE SHAPE OF THE ANSWER

1 **Their model first**	Ask what the family already believes, without correcting it
2 **Name the symptoms**	Plain words for voices, beliefs, disorganisation, negative symptoms
3 **Remove blame**	Vulnerability interacting with stress, not upbringing or weakness

4 Expressed emotion	Criticism, hostility and over-involvement adverse, warmth protective
5 Close with the plan	Four-phase family work, early warning list, named contacts

Explanatory model first, then the brain. The station usually seats an extended family. Identify the chief caregiver and the main decision-maker while keeping the patient central, ask what the family already believes (stress, supernatural causes, astrological explanations are common), acknowledge the caregiving struggle, and only then offer the medical account. Keep tone calm, respectful and free of blame.

Explain the illness as a long-term brain condition affecting thinking, perception and emotional expression. A usable analogy: the brain normally filters incoming information, decides what matters and organises thought; here the filter and the organising go wrong, so the brain misreads reality and produces experiences that feel entirely genuine.

PHENOMENON	WORDS FOR THE FAMILY	DISCRIMINATOR
Auditory hallucination	Hearing sounds or voices when nobody is present and nothing in the room accounts for it	**The brain registers them, so the person is not pretending**
Delusion	A belief held firmly that others do not share and reality does not support, such as neighbours intending harm	**Argument does not shift it; do not debate in the room**
Thought disorder	Difficulty keeping thoughts in order, so speech comes out jumbled	**A speech problem, not rudeness**
Passivity	The sense that actions, thoughts or feelings are being directed from outside	**Frightening rather than dramatic**
Negative symptoms	Reduced emotional expression, motivation and social contact	**Illness, not laziness; this line relieves the most conflict**
Lack of insight	The person genuinely cannot see that the experiences come from the illness	**A symptom, not obstinacy**

Give the diagnostic threshold if asked. The ICD-11 requirement is at least two symptoms present most of the time for at least one month, including at least one core symptom: delusions, hallucinations, disorganised thinking, or experiences of influence, passivity or control.

DSM-5 requires continuous disturbance for at least six months, including at least one month of active-phase symptoms.

Relieving guilt is a scored behaviour. Say plainly that the condition is **not caused by upbringing**, weakness of will or family quarrels.

- **Concept.** Then give the **stress-diathesis account**: a neurobiological vulnerability interacting with environmental stress.

- **Concept.** Excess dopamine transmission in mesocorticolimbic pathways maps to **positive symptoms**, and reduced mesocortical dopamine activity to negative symptoms, with serotonin and glutamate also involved.
- **Concept. Risk raisers**: pregnancy and birth complications, in utero viral infection, maternal malnutrition, neonatal hypoxia, and adolescent cannabis use.

RELATIONSHIP	RISK QUOTED	USE IN THE ROOM
General population	Approximately 1 in 100 people (1%)	**The baseline the family compares against**
Both parents affected	About 40%	Highest familial figure the family may have read
Monozygotic twin	50% concordance	**Not 100%, which is the point: genes are not destiny**
Dizygotic twin	15%	Contrast that carries the heritability argument
Adopted-away offspring of an affected parent	12%	**Risk travels with genes, not with the household**
Paternal age over 55 years	Contributes increased risk	Answers the "was it something we did" question

Course, physical health and suicide. Four numbers carry this part of the conversation.

- **Key number.** **Peak onset 15 to 35 years**, earlier in men, 18 to 25 years, than in women, 25 to 35 years.
- **Concept.** Course may be episodic with relapses, continuous, or rarely a single episode.
- **Key number.** **Life expectancy is reduced by roughly 15 to 25 years**, driven by premature cardiovascular disease, metabolic syndrome and late diagnosis of physical illness.
- **Key number.** Substance misuse affects up to a third of patients, and lifetime suicide risk is about 5%, which is why hopelessness and suicidal ideation are asked about at each review.

Medication counselling. Give the family the mechanism, the choice and the two emergencies, in that order.

- **Concept.** Antipsychotics act mainly as **dopamine D2 antagonists**.
- **Concept.** Second-generation agents, olanzapine, risperidone, quetiapine, aripiprazole, generally carry **less extrapyramidal burden** than first-generation agents such as haloperidol and chlorpromazine.
- **Concept. Long-acting injections**, given every 2 weeks to 3 months, are the option when oral adherence is unreliable.
- **Concept.** Inadequate response to at least two antipsychotics, one of them atypical, is what moves the case to **clozapine**.
- **Concept.** Teach the family to watch for **movement effects**, parkinsonism, akathisia, acute dystonia and tardive dyskinesia.
- **Concept.** Then the **metabolic and cardiac set**: weight gain, dyslipidaemia, impaired glucose tolerance, postural hypotension, QT prolongation and hyperprolactinaemia.
- **Trap.** Leaving out the **two emergencies**, neuroleptic malignant syndrome and clozapine-related neutropenia. Name both out loud.

Expressed emotion. The construct came out of work by Brown and colleagues at the Maudsley in the 1950s, when discharged patients returning to live with parents or spouses relapsed more often than those living elsewhere.

- **Concept. Five components.** Critical comments, hostility and emotional over-involvement on the adverse side; warmth and positive remarks on the protective side.
- **Concept.** Measurement is by the **Camberwell Family Interview**, with the five-minute speech sample, the level of expressed emotion scale, the family attitude scale and the family emotional involvement and criticism scale as alternatives.

DOMAIN	ODDS RATIO FOR RELAPSE	95% CONFIDENCE INTERVAL
High expressed emotion, early relapse (12 months or less)	**4.87**	3.22 to 7.36
High expressed emotion, late relapse (beyond 12 months)	2.13	1.36 to 3.35
High critical comments	2.22	1.16 to 4.26
Warmth	**0.35, protective**	0.15 to 0.85

Table: the meta-analysis figures worth quoting, from 33 prospective cohorts and 2284 patients.

INDIA CONTEXT

The Chandigarh studies by Leff, Wig and colleagues, 1987 and 1990, found substantially lower rates of high expressed emotion in urban and rural Indian families than Western cohorts, which fits caregiving shared across a joint family. Where high expressed emotion is present, its association with relapse holds.

Statutorily: the right to information sits in Section 22 of the Mental Healthcare Act 2017, confidentiality with its listed exceptions in Section 23, and access to records in Section 25.

The nominated representative sits in Section 14 with duties in Section 17, the service entitlement in Section 18, and supported admission in Section 89, limited to thirty days.

Reporting to the Board runs within three days for a woman or a minor, and within seven days for any other person.

The family intervention you offer at the close. Four phases, and the examiner wants them in order.

- **Concept. Engagement.** Build the alliance, validate caregiver stress, assign no blame.
- **Concept. Psychoeducation.** Diagnosis, biology, what treatment can be expected to do, and relapse, reframing avolition and poor self-care as illness.
- **Concept. Communication training.** Brief, clear, low-arousal sentences, criticism replaced by neutral requests, and face-to-face contact hours regulated when tension is high.
- **Concept. Problem solving.** An early warning checklist and named emergency contacts.

TRAP Telling the family that high expressed emotion causes the illness. It is a psychosocial stressor and a predictor of relapse, not the cause. Equally, do not scold relatives for criticism: high expressed emotion reflects burden, frustration and missing information, which is what the intervention supplies.

TRAP Reading emotional over-involvement as love. Intrusive, overprotective, infantilising self-sacrifice raises stress; warmth is calm validation and protects. Those two are scored on opposite sides.

INDIA CONTEXT

Do not present a key-worker care programme as Indian practice; it is a National Health Service administrative structure with no Indian statutory equivalent. The Indian counterpart is multidisciplinary follow-up in the treating unit with District Mental Health Programme linkage, which operates in 655 districts, and community facilities that Section 18(4) and Section 19(3) make statutory rather than optional.

HOLD THIS

Ask the family's explanatory model, remove blame, then name the symptoms in plain words. Warmth is protective, criticism is a relapse risk, and the negative symptoms conversation prevents most household conflict.

WHAT EARNS THE MARKS

- **Elicits the explanatory model before teaching**: the account you give has to land inside theirs.
- **Says the negative symptoms are illness**: that one line prevents most household conflict.
- **Separates warmth from emotional over-involvement**: they are scored on opposite sides.
- **Calls high expressed emotion a relapse predictor**: calling it the cause is a scored error.

SEE ALSO 05 Breaking bad news, and the dementia family meeting 08 Clozapine counselling 09 Lithium counselling, and the bipolar relapse plan

07 · Explaining ECT and taking consent

COMMUNICATION STATIONS · TOPIC 07

This patient has been advised electroconvulsive therapy. Explain it and take consent.

COMMUNICATION STATION, CONSENT STATION, STATUTE VIVA

THE SHAPE OF THE ANSWER

1 Capacity first	Understand, retain, weigh, communicate, then their fears
2 Describe it plainly	Anaesthesia, relaxant, measured current, brief therapeutic seizure
3 Indications and workup	Named indications, relative cautions, fasting, investigations
4 Memory, said accurately	About an hour either side, and the longer-term picture
5 The statute	Section 95 bars unmodified use, Section 94 bars emergency use

Consent begins with capacity, not with the leaflet. Establish that the patient can understand the treatment, retain the information, weigh risks and benefits against alternatives, and communicate a decision. Then elicit ideas, concerns and expectations, because the fear in the room usually comes from cinema and from unmodified practice rather than from the procedure you are proposing.

Describe it without euphemism and without menace: a controlled procedure under general anaesthesia in which a small, measured electrical current produces a brief therapeutic seizure. Give the leaflet, allow at least 24 hours to decide, and state that consent can be withdrawn at any point in the course.

1. **Indications you name.** Severe depression with high suicide risk, marked psychomotor retardation, food refusal or severe self-neglect; treatment-resistant depression; psychotic depression; catatonia of any cause; severe or mixed mania unresponsive to drugs; treatment-refractory agitation in dementia; severe depression in pregnancy where drugs pose fetal risk; and the frail older adult who cannot tolerate medication.
2. **Contraindications.** There are no absolute medical contraindications. Relative ones needing stabilisation and anaesthetic clearance: recent myocardial infarction or unstable cardiac disease, recent stroke, raised intracranial pressure, active internal bleeding, retinal detachment.
3. **Workup and fasting.** History, examination, cognitive assessment with baseline orientation and memory documented, basic bloods, electrocardiogram. Nil by mouth for at least 6 hours or from midnight; cardiac and antihypertensive drugs with sips of water; dentures, jewellery, contact lenses and hair accessories removed; bladder emptied.
4. **What happens in the suite.** Anaesthetist assessment and a short-acting anaesthetic producing unconsciousness of roughly 5 to 10 minutes; suxamethonium for neuromuscular blockade to prevent fracture and dislocation; oxygen by mask or tube through the transient apnoea, with routine intubation not required; padded mouthguard after relaxation; continuous electrocardiogram, oximetry, blood pressure and two-channel electroencephalogram.

PARAMETER	BITEMPORAL	RIGHT UNILATERAL
Placement	One electrode over each temple	Both electrodes over the non-dominant hemisphere
Efficacy	Standard response rate 70 to 80 percent (60 percent in one secondary source)	Can be slightly lower, still therapeutically effective
Cognition	More short-term confusion and retrograde amnesia	**Less cognitive disruption, less retrograde impairment**
Choose it when	Life-threatening illness, severe catatonia, high suicidality, or unilateral has failed	**Pre-existing memory deficit, or cognition must be protected**

Seizure adequacy and its two thresholds. The stimulus itself lasts about 5 seconds. Two durations decide what happens next.

- Key number. **Adequate** is a motor seizure over 15 seconds, typically 20 to 50 seconds, with generalised paroxysmal spike-and-wave discharges followed by post-ictal suppression.
- Key number. If the motor or electroencephalographic seizure is under 15 seconds, restimulate at a higher charge up to 2 further times, a **maximum of 3 stimulations** in one session. If the third fails, abandon the session and titrate the threshold upward next time.
- Key number. **Prolonged** is beyond 180 seconds, and is terminated with an intravenous benzodiazepine.

Course. Twice weekly in the primary guideline, with some protocols using two to three times weekly.

- **Key number.** An acute course runs **6 to 12 sessions**, with improvement often visible within 2 weeks or after about 6 treatments, and mental state and cognition reassessed after each session.
- **Key number.** Maintenance treatments taper from weekly to fortnightly to monthly, with continuation re-evaluated after 6 months.

Memory, said accurately. Four effects, each with its own time course, and no promises about any of them.

- **Concept.** **Post-ictal confusion** lasts minutes to a few hours, and is more noticeable in older patients.
- **Concept.** **Anterograde** difficulty holding new information persists through the course and settles over weeks.
- **Key number.** **Retrograde amnesia** typically covers events about 1 hour before and 1 hour after each treatment, such as the journey in.
- **Key number.** **Autobiographical gaps** for discrete events from the preceding weeks or months occur in about 10 percent of patients. Long-term irreversible loss is rare.
- **Key number.** Objective cognitive performance at **2 months** often improves on baseline, because the depression that was slowing cognition has lifted.
- **Key number.** **Activity restrictions.** No driving, machinery, work or major legal and financial decisions for 1 to 2 weeks after an acute course, or at least 24 hours after an outpatient maintenance treatment.

INDIA CONTEXT

The Mental Healthcare Act 2017 received assent on 7 April 2017 and came into force on 29 May 2018, repealing the 1987 Act.

Section 95 prohibits electroconvulsive therapy without muscle relaxant and general anaesthesia, so direct or unmodified treatment is unlawful.

Section 95 also prohibits it for minors under 18 years by default. Where the treating psychiatrist judges it necessary it requires both the guardian's informed consent and prior permission of the Mental Health Review Board, a quasi-judicial body of 6 members for a district or group of districts.

Section 94 prohibits it as emergency treatment, which is the opposite of the position candidates import from United Kingdom textbooks.

Rights sit in Chapter 5, Sections 18 to 28, and a valid advance directive binds the treating team unless altered by the Board.

TRAP Saying that emergency treatment can be given without consent to save life in a stuporous catatonic patient. Under Section 94 electroconvulsive therapy is not available as emergency treatment in India; the route is supported admission with the nominated representative's consent under the standard statutory procedure.

TRAP Confusing the two seizure numbers, or guaranteeing an outcome. Adequacy is a motor seizure over 15 seconds; prolonged is beyond 180 seconds. Do not promise cure and do not promise absence of memory effects; give the 1 hour window, the roughly 10 percent figure for autobiographical gaps, and the improvement seen at 2 months.

TRAP The examiner will ask which intravenous induction agent is used in Indian practice and what premedication is given before the stimulus, with the rationale. The library records the induction agent and any premedication as unsourced, so verify both against a cited Indian practice guideline before the exam; do not offer a drug name from memory in the station.

HOLD THIS

Modified means anaesthesia plus relaxant together, and in India Section 95 makes that the law and Section 94 bars emergency use. Adequate seizure over 15 seconds, prolonged beyond 180 seconds, memory gap about 1 hour either side.

WHAT EARNS THE MARKS

- **Defines modified treatment as anaesthesia plus relaxant**: it is what Section 95 requires.
- **Gives the two seizure durations correctly**: adequacy and prolonged are different numbers.
- **Describes memory effects without promising anything**: a promise here is a scored error.
- **Offers the leaflet and time to decide, with withdrawal**: consent is a process, not a signature.

SEE ALSO 08 Clozapine counselling 09 Lithium counselling, and the bipolar relapse plan 13 Consent and capacity as a bedside skill

08 · Clozapine counselling

COMMUNICATION STATIONS · TOPIC 08

Counsel this patient and his nominated representative before starting clozapine.

COMMUNICATION STATION, PRESCRIBING VIVA

THE SHAPE OF THE ANSWER

1 Why it is worth it	Most effective in resistance, fewer admissions, less suicidality
2 Baseline and titration	Counts, cardiac markers, metabolic set, slow upward steps
3 Monitoring	Green, amber and red counts on a fixed schedule
4 The red flags	Fever, chest symptoms, constipation, dizziness on standing
5 Dose variables	Smoking, infection, and a break beyond forty-eight hours

Lead with why it is worth the blood tests. Clozapine is the **most effective option in treatment-resistant illness** and reduces suicidal thinking and admissions. That is the sentence the patient and the nominated representative need before any monitoring burden is described.

- **Concept. Treatment resistance** means an unsatisfactory response to adequate doses of at least 2 antipsychotics, at least 1 of them a second-generation agent, for an adequate duration.
- **Concept.** Other indications: **recurrent suicidal behaviour** in schizophrenia or schizoaffective disorder, severe intolerable neurological adverse effects with other agents, and psychosis in Parkinson disease where standard treatment has failed.

Baseline workup, in four groups.

- **Key number. Haematology.** Full blood count with absolute neutrophil count less than 10 days old, with white cells 3.5 x 10^9/L or above and neutrophils 2.0 x 10^9/L or above.
- **Key number. Cardiac.** Electrocardiogram, troponin I, C-reactive protein, pulse, and lying and standing blood pressure.

- **Key number.** **Metabolic and organ.** Fasting glucose, HbA1c, lipids, weight, body mass index, waist circumference, liver and renal profile.
- **Key number.** **The easily forgotten set.** A documented baseline bowel habit and laxative history, abnormal involuntary movement screening, a pregnancy test where relevant, and smoking status.

Titration. Admission is advised for initiation, because orthostatic hypotension, sedation, tachycardia and syncope peak early. Community initiation needs daily visits for at least 2 weeks and an overnight support person in the first week.

- **Key number.** **Adults aged 18 to 59 years.** 12.5 mg at night on the first night, 25 mg on the second, 37.5 mg on the third, then 50 mg held for 3 days, then increases of 50 mg every 3 days.
- **Key number.** Above 200 mg daily, **split the dose unevenly** with the larger part at night.
- **Key number.** The formulary maximum is 900 mg daily, and a typical minimum target is 300 mg to 350 mg.
- **Key number.** **From 60 years**, start 12.5 mg at night, increase by 25 mg every 3 days, and hold a lower target of 100 mg to 300 mg.

CATEGORY	COUNTS	ACTION
Green	White cells 3.5 x 10^9/L or above and neutrophils 2.0 x 10^9/L or above	Continue unchanged
Amber	White cells 3.0 to 3.5 x 10^9/L or neutrophils 1.5 to 2.0 x 10^9/L	**Continue, but repeat the count twice weekly until green; look for viral illness and marrow-suppressing co-medication**
Red	White cells below 3.0 x 10^9/L or neutrophils below 1.5 x 10^9/L	**Stop immediately, daily counts until 2 consecutive green results, watch for infection; a confirmed red result precludes re-exposure**

Schedule, primary standard: **weekly counts for the first 18 weeks**, fortnightly from weeks 19 to 52, monthly thereafter, and weekly for 4 weeks after stopping.

- **Trap.** The United States framework quoted in secondary sources is weekly for the first 6 months, fortnightly for months 7 to 12, then monthly. **Name which one you are using.**
- **Key number.** Monitoring reduces agranulocytosis risk roughly 20-fold, from an unmonitored 0.7% to 0.03%, and the peak risk period is the **first 6 months**, independent of dose.

PROBLEM	NUMBERS THAT MATTER	DISCRIMINATOR
Benign drug fever	Up to 20% of patients in the first 3 weeks; an isolated temperature of 38 degrees C or above	**Not a reason to stop, provided septic screen, counts and cardiac markers are clear**
Myocarditis	Incidence 0.1% to 3.0%; within the first 28 days, peak 12 to 16 days; C-reactive protein rises about 5 days before troponin; sinus tachycardia over 120 bpm or a rise over 30 bpm	**Stop permanently if C-reactive protein is over 100 mg/L or troponin I exceeds twice the upper limit**
Cardiomyopathy	Average latency 14.4 months; ejection fraction under 25% carries poor prognosis; screen for heart failure at least 4 times a year; QT over 500 ms	**Late, with heart failure symptoms and a raised natriuretic peptide**
Gastrointestinal hypomotility	Constipation in 14% to 60%; admission if no bowel movement for 5 days despite laxatives	**Kills more patients than agranulocytosis does**
Seizures and levels	Half-life about 12 hours, norclozapine about 23 hours; trough drawn 10 to 14 hours post-dose in 2 mL of EDTA blood; response threshold 0.35 mg/L to 0.42 mg/L, up to 0.5 mg/L; expected clozapine to norclozapine ratio about 1.32	**A low ratio suggests induction or missed doses, a high ratio saturation or poor metabolism**

Cardiac surveillance you say out loud. Two schedules and one threshold pair.

- **Key number.** **Vital signs** before the dose and 1 hour after it, through the first 7 days.
- **Key number.** **Full blood count, C-reactive protein and troponin I** at baseline and then at 7, 14, 21 and 28 days.
- **Key number.** With C-reactive protein 50 to 100 mg/L or troponin at or below twice the upper limit, continue with daily markers and request an **urgent echocardiogram**. Above those thresholds, stop and refer to cardiology.

Smoking and the missed dose, the two questions that decide the station. Both are about induction of the same enzyme, and both change the dose.

- **Concept.** Polycyclic aromatic hydrocarbons from **burning tobacco or cannabis induce CYP1A2**. Nicotine itself, and nicotine replacement, do not.
- **Key number.** So smokers need higher doses. Targets are given as 250 mg daily for a female non-smoker, 350 mg for a male non-smoker, 450 mg for a female smoker and 550 mg for a male smoker.
- **Trap.** **Abrupt cessation on a smoke-free ward** removes the induction within days and pushes levels up towards sedation, myoclonus and seizures. Infection and sepsis lower CYP1A2 expression and do the same.
- **Key number.** A break longer than **48 hours** means tolerance to alpha-1 and cholinergic blockade has gone. Do not resume the previous dose: restart at 12.5 mg at night and re-titrate.
- **Key number.** **Reset the haematological monitoring too.** A gap over 3 days on weekly monitoring returns to weekly for 18 weeks. On fortnightly or monthly monitoring, a gap over 3 days but under 4 weeks means weekly for 6 weeks, and over 4 weeks means starting again as a new patient.

CLINICAL ANCHOR

The red-flag script for the patient and the nominated representative: fever, sore throat or rigors; chest pain, breathlessness or a racing heart; severe constipation, no stool or flatus, abdominal swelling or vomiting; dizziness on standing or fainting. Add the bowel regimen with fluids, fibre and prophylactic laxatives taken as prescribed, and the instruction not to double up after a missed dose but to telephone the clinic.

TRAP Naming agranulocytosis as the main lethal hazard. With monitoring in place, gastrointestinal hypomotility (ileus, toxic megacolon, ischaemia, perforation) causes more deaths, and patients with schizophrenia often under-report abdominal pain until they are septic. Ask about bowels at every contact.

TRAP Stopping the drug for an isolated fever, or restarting a full dose after a three-day gap. Both are scored errors: investigate the fever, and restart at 12.5 mg.

INDIA CONTEXT

There is no statutory national manufacturer registry here of the kind that governs monitoring elsewhere, so governance rests on the treating psychiatrist and the hospital dispensary: a named monitoring record, a nominated representative who knows the red flags, and a documented consent discussion.

TRAP Hypersalivation is on the counselling list the primary guideline requires you to cover, alongside constipation, sedation, weight gain and urinary incontinence, but its mechanism and drug management are marked unsourced in the library. Verify the pharmacological management from a prescribing reference before the exam instead of quoting an agent and dose here.

HOLD THIS

Green, amber, red on the counts; the first 28 days for myocarditis; bowels at every visit; and 48 hours means re-titrate from 12.5 mg. Smoking status is a dosing variable, and stopping smoking is a dose reduction.

WHAT EARNS THE MARKS

- **Leads with the benefit before the burden**: it is what makes the monitoring acceptable.
- **Gives the green, amber and red actions**: the count alone is not the answer.
- **Names bowel symptoms as the lethal hazard**: hypomotility outranks agranulocytosis under monitoring.
- **Treats smoking status as a dosing variable**: stopping smoking acts as a dose increase.

SEE ALSO 06 Explaining schizophrenia, and expressed emotion 09 Lithium counselling, and the bipolar relapse plan 16 AIMS and the other movement scales

09 · Lithium counselling, and the bipolar relapse plan

COMMUNICATION STATIONS · TOPIC 09

Counsel this patient starting lithium, then build a relapse prevention plan with her.

COMMUNICATION STATION, PRESCRIBING VIVA, STATUTE VIVA

THE SHAPE OF THE ANSWER

1	**Baseline and brand**	Renal, thyroid, calcium, count, tracing, pregnancy test
2	**Levels**	Trough twelve hours after the dose, target set by indication
3	**Nuisance against toxicity**	Fine tremor tolerable, coarse tremor and vomiting mean stop
4	**Fluid and salt**	Steady salt, more fluid in heat, fever and fasting
5	**The relapse plan**	A signature of four to six items, two tiers, three contacts

Brand, level, hydration, toxicity: the four things that decide this station. Work through indication, contraindications and baseline before any counselling line.

- **Concept. Confirm the indication**: acute mania, maintenance prophylaxis, augmentation in recurrent depression, or aggressive and self-harming behaviour.
- **Concept. Screen the contraindications**: significant renal impairment, untreated hypothyroidism, cardiac rhythm disturbance including Brugada syndrome, Addison disease, low sodium states.
- **Concept. Baseline bloods.** Urea, electrolytes, creatinine and eGFR; free T4 and thyroid-stimulating hormone; calcium; full blood count; urine dipstick.
- **Concept. Baseline, the rest.** A 12-lead electrocardiogram where there is cardiovascular disease or risk, weight and body mass index, and a pregnancy test in women of childbearing potential.

Hand over the treatment card, the information booklet and the monitoring record, and make the brand point explicitly: preparations are not bioequivalent, so the prescription names the brand, which matters where retail substitution is common.

ITEM	NUMBER TO QUOTE	DISCRIMINATOR
Starting dose	400 mg once at night in adults, 200 mg at night in older adults, adjusted in steps of 100 mg to 200 mg	**Night dosing allows a true 12-hour trough**
Sampling	Exactly 12 hours post-dose, just before the next dose	**A sample at 4 to 6 hours reads the peak and fakes toxicity**
Early checks	7 days after starting, 7 days after each change, then weekly until stable	Kinetics are linear: 400 mg giving 0.4 mmol/l predicts 0.6 mmol/l at 600 mg
Target, first manic presentation	0.6 to 0.8 mmol/l	**Refractory mania 0.8 to 1.0 mmol/l**
Target, maintenance, older adults, augmentation	0.4 to 0.6 mmol/l	Overall reference window 0.4 to 1.0 mmol/l
Stable monitoring	Level every 3 months for the first 1 year, then every 3 to 6 months; renal, thyroid, calcium and weight every 3 to 6 months; urinalysis and blood pressure every 12 months	**Formal review after 2 to 5 years, with treatment planned for at least 2 to 3 years**
Intensified monitoring	At least 3 monthly from 65 years, on interacting drugs, with falling eGFR, thyroid or parathyroid disease, shifting sodium or fluid intake, poor control or doubtful adherence, or a last level of 0.8 mmol/l or higher; and in pregnancy	**The last level itself is a risk factor**
Half-life	20 hours in healthy individuals	Explains why a single missed dose is not a crisis

Nuisance versus toxicity, taught as a contrast. Three tiers, and the patient needs to be able to tell them apart.

- **Concept.** **Dose-related nuisances**, managed by small dose adjustment: fine hand tremor, mild nausea, loose stool, dry mouth, a mild metallic taste, mild polyuria and polydipsia, ankle oedema, weight gain.
- **Concept.** Lithium-induced hypothyroidism is treated with **levothyroxine while the lithium continues**.
- **Key number.** **Early toxicity**, above 1.0 mmol/l in one guideline and above 1.5 mmol/l in others: anorexia, persistent vomiting, worsening diarrhoea, a coarse tremor of the hands and lower jaw, muscle weakness, lethargy, ataxia, dysarthria, tinnitus, blurred vision.
- **Key number.** The action there is to **withhold the drug**, take a stat level and renal panel, and seek specialist advice the same day.
- **Key number.** **Severe neurotoxicity**, above 2.0 mmol/l, brings confusion and delirium, muscle hyperirritability, choreoathetoid movements, convulsions, syncope, arrhythmias, stupor and coma. That is a hospital emergency.
- **Trap.** Neurotoxicity can occur with levels **inside the target range**, and persistent headache with visual disturbance is investigated for raised intracranial pressure.

- **RULE** **Fine tremor is a nuisance, coarse tremor of hands and jaw is a warning.** Give the patient one instruction they can act on alone: if vomiting, diarrhoea, a coarse tremor or unsteadiness appear, stop the tablets and attend the nearest emergency facility.

Diet, fluid and the Indian summer. Lithium is reabsorbed in the proximal tubule alongside sodium, so **salt intake is a dosing variable**.

- **Concept.** A sudden drop in dietary salt, crash dieting or a low-salt regimen drives **retention and toxicity**. Keep salt intake steady.
- **Concept.** **Dehydration cuts clearance sharply**, so fluids matter during fever, gastroenteritis, vomiting, diarrhoea, heavy exertion, long air travel and hot weather, where heat waves and high sweat rates are a real hazard.
- **Concept.** **Religious and traditional fasting** needs supervision and a planned fluid schedule.
- **Key number.** Avoid alcohol in the first 1 week, then limit it to no more than 1 to 2 units daily.
- **Key number.** A **missed dose** is taken only if remembered within 3 hours, otherwise skipped, and the dose is not doubled. Tablets are swallowed whole.

INTERACTION	MECHANISM AND SIZE	MANAGEMENT
Thiazide diuretics	**Reduced clearance, up to a 4-fold rise within 7 to 10 days**	Avoid; if unavoidable, reduce the lithium dose and monitor closely
ACE inhibitors and angiotensin receptor blockers	Reduced filtration and excretion, up to a 4-fold rise, with risk of renal failure	Avoid; intensify level and renal checks if required
NSAIDs	**Prostaglandin inhibition, reduced renal blood flow, rise within days**	Warn against over-the-counter ibuprofen and diclofenac; if essential, fixed dosing with level checks rather than as-needed use
Loop diuretics	Retention, greatest risk in the first 1 month	Monitor during initiation
Carbamazepine	Neurotoxicity despite normal levels	Monitor clinically, not just biochemically

Pregnancy, in the order the examiner asks. Counsel before conception, weighing fetal exposure against maternal relapse, and advise against stopping contraception without discussion.

- **Key number.** In **sustained remission with mild illness**, lithium may be tapered over 4 weeks. In severe recurrent illness or previous postpartum psychosis, continuing lithium or switching to an antipsychotic is considered.
- **Key number.** **First-trimester exposure** carries an absolute risk of congenital cardiac anomaly of up to 2.25%, with right ventricular outflow tract obstruction at 0.6%.
- **Trap.** The historical association with a **specific tricuspid anomaly** has not been confirmed by later systematic study, so do not quote it as established.
- **Key number.** Risk is **dose-related**, with malformations increased above 900 mg daily in a single study. No dose is established as safe, and exposure is not in itself a medical ground for termination.
- **Key number.** **Monitoring.** Levels every 4 weeks until the 36th week and weekly thereafter, delivery in hospital with strict fluid balance in labour, the maternal level within 24 hours of delivery, and the neonatal level shortly after birth.

- **Key number.** Watch the newborn for **floppy baby syndrome**, hypothermia, poor feeding, cyanosis, tremor or seizures. Breastfeeding is not recommended on lithium.

HAEMODIALYSIS CRITERION	THRESHOLD
Life-threatening dysrhythmia or severe neurological features (seizures, coma)	**Dialyse irrespective of the serum concentration**
Renal impairment	Above 4.0 mmol/l
Asymptomatic	Above 5.0 mmol/l
Acute overdose on long-term therapy	Above 4.0 mmol/l
Chronic toxicity	**Above 2.5 mmol/l**

Emergency sequence, in order.

- **Key number.** **Stop the drug**, assess airway, breathing and circulation with a 12-lead electrocardiogram, and take a stat level with renal panel, calcium, thyroid function and full blood count.
- **Key number.** Give **intravenous saline** to restore volume and promote elimination. Consider whole bowel irrigation above 4 g ingested. Forced diuresis is contraindicated.
- **Key number.** Then apply the **dialysis criteria** in the grid above.
- **Key number.** **Medical assessment is mandatory** after ingestion of 50 mg/kg or more of lithium carbonate, or 100 mg/kg of the citrate, in any intentional overdose, from 65 years of age, or where interacting drugs are involved.
- **Key number.** Restart only once levels have stayed within or below target for at least 24 hours.

The relapse plan the bipolar station is really asking for. Four words, distinguished before you start.

- **Concept.** An **inter-episode symptom** is any mood symptom between episodes.
- **Concept.** A **prodrome** is the build-up in number, frequency and severity just before an episode.
- **Concept.** **Early warning symptoms and signs** are that person's own subjective and observable features.
- **Concept.** A **relapse signature** is the idiosyncratic set chosen to trigger action.

The evidence is worth one sentence, and it is a sentence with numbers in it.

- **Key number.** In a randomised controlled trial of 69 patients, teaching people to detect early manic symptoms and seek routine care **increased the time to the next manic episode 4-fold** and produced a 30% reduction in manic relapses over 18 months.
- **Key number.** Depressive relapses were unchanged in that first trial. Across 6 randomised controlled trials and 468 patients the approach is **established for both poles**.
- **Key number.** Family-focused work that covers early warning signs in the first 7 of 18 sessions also protects against **depressive recurrence**.

1. **Session 1, retrospective mapping.** Reconstruct the onset of the most recent and the first or most severe episodes, identify the moment insight was lost, cross-check against a prodrome checklist, and set a mood diary of at least 7 days.
2. **Session 2, diary review and card sorting.** Remove the person's baseline inter-episode symptoms from the list, write each candidate symptom on a card with one card reading "loss of insight", shuffle, and have the person order them forward to insight loss or backward from it, repeating with a second well-remembered episode and filling gaps from family or records.

Select a signature of 4 to 6 symptoms, signs or situations, split into warning level and danger level.

3. **Session 3, the action plan.** Agree the two tiers, write the person's own motivational statement, list adaptive coping and the habits to avoid, name 3 healthcare contacts with at least 1 offering 24-hour access, agree the clinical response pathway, and have the patient, clinician, responsible consultant and primary caregiver sign it. Condense the key sections onto a wallet card.

Timing is the teachable part, and it splits in two.

- **Key number.** The **early prodrome** appears up to 4 weeks before insight is lost, commonly 2 to 4 weeks, and carries 2 or 3 warning symptoms. That is when monitoring moves from weekly to daily and personal coping starts.
- **Key number.** The **late prodrome** escalates over days to hours and triggers immediate service contact.
- **Key number.** Carers recognise manic signs roughly **1 week later** than the patient does, which is why both are trained, and services aim to review within 1 week of an early warning.

POLE	COPING TO PRESCRIBE	HABITS TO PROHIBIT
Manic prodrome	Deliberate slowing, low-stimulus activities, the agreed extra dose, prioritising and shedding commitments, delaying major financial and interpersonal decisions, reality-testing with a trusted confidant, taking leave from work	**Stopping the mood stabiliser, alcohol, cannabis or stimulants, new projects, and calling acceleration good health**
Depressive prodrome	A structured daily schedule, organised routines, actively mobilising family and friends, socialising rather than withdrawing, behavioural distraction, testing catastrophic assumptions	**Abruptly stopping or doubling medication, alcohol, and prolonged rumination**

Sleep is both a sign and a trigger. Teach the family to separate the two pictures before they act on either.

- **Concept. Reduced need for sleep in mania**: short sleep, waking energised, more nocturnal goal-directed activity.
- **Concept. Insomnia in depression or anxiety**: long latency, broken or early waking, waking unrefreshed and distressed.
- **Concept.** Then **fix the rhythm**: the same bedtime and rising time whatever the fatigue, regular meals, regular social contact.
- **Concept.** Dim the light and drop stimulating screens as latency widens, and agree a **rescue step** in advance if sleep falls below the agreed baseline on two consecutive nights.

INDIA CONTEXT

Carry the action plan into statute. Section 5 gives every person who is not a minor the right to make an advance directive in writing, stating how they wish and do not wish to be treated and naming the nominated representative.

It is invoked only when capacity to make treatment decisions is lost, a later capacitous decision over-rides it, and one made contrary to law is void from the start.

Section 6 sets the manner, Section 7 puts the online register of directives on every Board, and Section 10 makes it the duty of the medical officer and treating psychiatrist to treat in accordance with a valid directive, subject to Section 11.

Section 11(4) lets a legal guardian make one for a minor, and the nominated representative is appointed in writing under Section 14.

Where admission is needed and the person accepts they have a mental illness and wants admission, the route is independent admission under Section 86 rather than supported admission under Section 89.

TRAP Reciting a generic textbook list as the relapse signature. Prodromes are idiosyncratic and the signature has to be mapped from that person's own episodes by card sorting; a borrowed list gives a plan nobody recognises in time.

TRAP Offering psychoeducation as an alternative to maintenance pharmacotherapy. It is an adjunct; stopping the mood stabiliser in favour of intermittent treatment increases relapse morbidity.

TRAP Quoting the expanded multifamily group format as the version that carries the trial evidence. The randomised data (35 families, 29 completers, children aged 8 to 11, and improved ability to obtain services in 82% of intervention families against 20% of wait-list families, with parental knowledge gains held at 6-month follow-up) tested the original six-session, seventy-five-minute groups; the current 8-session, 90-minute format is a later clinical iteration with no separate outcome figures. The individual family version runs 16 sessions.

HOLD THIS

Trough at 12 hours, target by indication, and the patient's one rule: coarse tremor, vomiting or unsteadiness means stop and attend. The relapse plan is a signature of 4 to 6 items with two tiers, three contacts and four signatures.

WHAT EARNS THE MARKS

- **Prescribes by brand and says why**: preparations are not bioequivalent.
- **Gives the patient one rule they can act on alone**: coarse tremor, vomiting or unsteadiness means stop and attend.
- **Maps the signature from her own episodes**: a borrowed list is recognised too late.
- **Offers psychoeducation as an adjunct**: dropping the mood stabiliser for it raises relapse morbidity.

SEE ALSO 06 Explaining schizophrenia, and expressed emotion 08 Clozapine counselling 13 Consent and capacity as a bedside skill

10 · Starting an antidepressant, stopping a benzodiazepine

COMMUNICATION STATIONS · TOPIC 10

Start an antidepressant with this patient, then taper another patient's long-term benzodiazepine.

TWO COMMUNICATION STATIONS, PRESCRIBING VIVA

THE SHAPE OF THE ANSWER

1 **Set the frame**	Shared decision, their beliefs, the relative who holds the strip
2 **The seven contents**	Indication, dose, lag, early effects, contacts, duration, stopping
3 **The activation window**	Supervised dispensing, crisis contact, an early review date
4 **Hyperbolic tapering**	Proportional steps, not a fixed milligram cut
5 **The benzodiazepine**	Stabilise, cross over, decelerate, hold rather than escalate

One station starts a drug, the other unpicks one, and both are marked on the same behaviours. Set the frame with **shared decision-making**, and ask what the patient already believes.

- **Key number.** Involve the family member who will **hold the strip**: between 40% and 80% of what is said in a consultation is forgotten immediately, and adherence in depression is low.

Cover, in order: indication, dosing schedule, the lag to benefit, early adverse effects, crisis contacts, planned duration, and discontinuation precautions. Pharmacotherapy is indicated for moderate to severe depression, for illness persisting 2 years or more, and for mild depression persisting longer than 3 months.

QUESTION THE PATIENT ASKS	ANSWER TO GIVE	DISCRIMINATOR
When will it work?	The highest rate of symptom improvement often falls in the first 2 weeks, while noticeable subjective and functional recovery usually takes 2 to 4 weeks to consolidate	**Adverse effects arrive before benefit does**
What if nothing changes?	No measurable improvement after 3 to 4 weeks at an adequate dose makes that regimen unlikely to succeed, so the dose is raised or the drug changed	**A review date, not an open-ended wait**
How long will I take it?	After remission of a single episode, continue at the dose that produced remission for at least 6 months to 9 months	**The full dose, not a token dose**
And if it has happened before?	With 3 or more episodes, maintenance runs at least 2 years and longer in severe or frequently recurring illness	**Episode count drives duration**
Anything besides tablets?	Combining psychotherapy reduces relapse when the drug is stopped, though fewer than 20% of patients receive it; exercise 3 times a week or more protects against recurrence	Adds an intervention the patient controls

Early effects, each with a behavioural fix. Give the fix in the same breath as the effect.

- **Concept. Nausea**: take with food, smaller frequent meals, cool water.
- **Concept. Sedation**: move the dose to bedtime, short naps, no driving until it settles.
- **Concept. Insomnia**: move the dose to the morning, cut late caffeine, finish exercise well before bed.
- **Concept. Dry mouth**: sips, sugar-free gum, nasal breathing, brush twice daily.
- **Concept. Constipation**: fluids, fibre, activity, a softener.
- **Concept. Postural dizziness**: slow transitions, hydration, bedtime dosing.
- **Concept. Sexual dysfunction**: name it before it happens, then consider timing intimacy before the dose, switching agent, or an adjunct.
- **Concept. Cardiac risk**: baseline and periodic electrocardiogram where QTc prolongation matters.

CLINICAL ANCHOR

The activation window is the safety-critical part of this station. In the first weeks, rising synaptic monoamines can produce restlessness, panic surges and agitation, and in vulnerable people that can raise suicidal ideation or supply the energy to act on a plan.

So the station expects three explicit safeguards: supervised dispensing by a family member holding the strip, a named 24-hour emergency contact, and a follow-up date inside the activation window.

Separate akathisia, which is being unable to sit still, from worsening depression, because the response is dose reduction rather than escalation. Then screen for a bipolar diathesis if racing thoughts, extreme energy or reduced sleep need appear.

TRAP The examiner will ask for the decriminalisation provision for attempted suicide and for the national crisis helpline number. The library marks that section number and those helpline digits as unsourced, so name the Mental Healthcare Act 2017 and the principle (a person who attempts suicide is presumed to have been under severe stress, is not prosecuted, and is owed care and rehabilitation) and verify the section and the number before the exam rather than quoting them here.

Serotonin syndrome, because it follows activation in the viva. An idiosyncratic disorder from **overstimulation of serotonergic receptors** by agents such as selective serotonin reuptake inhibitors, monoamine oxidase inhibitors and tricyclics.

- **Concept. Autonomic**: labile blood pressure, sweating, dyspnoea, fever, tachycardia.
- **Concept. Cognitive and behavioural**: confusion, hypomania, agitation, insomnia.
- **Concept. Neurological**: akathisia, hyperreflexia.
- **Trap.** Its management is marked **unsourced** in the library. Name the triad, say you would manage it as a medical emergency, and read the management from a prescribing reference before the exam.

FEATURE	DISCONTINUATION SYNDROME	RELAPSE OR RECURRENCE
Onset	**Within 1 to 2 days of the change, or hours after a missed dose (delayed days to weeks with fluoxetine)**	Insidious over weeks to months
Core symptoms	Dizziness, electric shock sensations, nausea, headache, ataxia, derealisation, vivid dreams, akathisia	Pervasive low mood, anhedonia, guilt, psychomotor slowing, vegetative change
Somatosensory signs	**Present: zaps, vertigo, flu-like aches, visual trailing**	Absent
Response to rechallenge	**Resolves within hours to a few days**	Needs 2 to 4 weeks of re-titration
Course	Transient over weeks, occasionally months	Progressive without treatment

Incidence to quote: **33% to 50% of patients** experience discontinuation symptoms on stopping or reducing, in the primary guidance. A secondary summary gives 15%, which is the discrepancy

the examiner may test.

- **Key number.** **Highest risk tracks the shortest half-life.** Venlafaxine 5 hours, clearing 99% in 1 day; duloxetine and desvenlafaxine 12 hours, clearing in 2.5 days; paroxetine 24 hours, clearing in 4.4 days.
- **Key number.** **Medium risk.** Sertraline 26 hours and 5.4 days, escitalopram 27 to 32 hours and 6.1 days, citalopram 36 hours and 7.3 days, bupropion 21 hours and 4.4 days, fluoxetine 4 to 6 days clearing in 25 days.
- **Key number.** By class: **mirtazapine highest risk**, amitriptyline and clomipramine medium, vortioxetine low, agomelatine lowest.

Hyperbolic tapering, explained the way the examiner wants. Transporter occupancy follows a hyperbolic curve, so a **fixed milligram cut removes far more occupancy at low doses** than at high ones.

- **Key number.** Reduce by a proportion of the current dose, **10% to 50% every 2 to 4 weeks**.
- **Key number.** On long treatment or high-risk agents, use **5% to 10% steps** down to doses as low as 2% of the original before stopping.
- **Key number.** **Practical methods.** Oral liquids diluted and drawn up in 1 ml, 5 ml or 10 ml syringes, with citalopram drops 8 mg equivalent to a 10 mg tablet; quartering round tablets; counting or weighing beads from extended-release capsules.
- **Key number.** Also dispersing a tablet in 100 ml of water for fractional dosing, and a single 20 mg dose of fluoxetine after stopping a short-acting agent as an **autotaper**.

Addiction versus dependence, the sentence that reassures. Addiction involves craving, compulsive self-administration, escalation and a high; antidepressants produce none of those. Physical dependence is neurobiological adaptation, so abrupt cessation produces somatic and affective withdrawal that a slow taper prevents. Using the phrase "discontinuation symptoms" rather than "withdrawal" keeps the conversation clinical.

The benzodiazepine station. Beyond the recommended 2 to 4 weeks, continued prescribing usually **outlasts the indication** without sustained benefit, because of receptor tolerance.

- **Concept.** **Harms to name.** Cognitive impairment with a prospective dementia association, psychomotor slowing with injurious falls and hip fracture in older people, impaired driving, increased mortality.
- **Key number.** **Fatal overdose risk** rises with opioids or alcohol, and alprazolam has risen more than 8-fold in prescribed quantity while being disproportionately toxic in overdose.
- **Trap.** **Abrupt cessation** can produce grand mal seizures 1 to 12 days after stopping, delirium, psychosis and autonomic instability, and a protracted syndrome can persist 18 to 24 months or longer after rapid unmonitored cessation.

PRIOR DURATION OF USE	MINIMUM TAPER LENGTH
2 to 8 weeks	At least 2 weeks
8 weeks to 6 months	At least 4 weeks, slowing in the second half
6 months to 1 year	At least 8 weeks
More than 1 year	**6 to 18 months**

Before any reduction, **stabilise interdose withdrawal** by spreading the same daily dose more evenly.

- **Key number.** Alprazolam 4 to 5 times daily, lorazepam 3 to 5 times daily, clonazepam 2 to 3 times daily, diazepam 2 to 3 times daily, with a diary of exact times and doses.
- **Key number.** **Structural safeguards.** One prescriber, one pharmacy, staged dispensing of 1 to 2 weeks at a time.
- **Key number.** Begin with a **test reduction** using the smallest cut the formulation allows, such as a quarter tablet, because rapid 25% cuts fail in 32% to 42% of long-term users and precipitate withdrawal in 90%.

Why diazepam, and how to cross over. Ashton's principles came out of 14 years of a dedicated withdrawal clinic reporting a **90% success rate**.

- **Key number.** Diazepam and its active metabolites have a half-life of **20 to 80 hours**, up to 200 hours, which smooths the decline and removes interdose troughs.
- **Key number.** Scored 2 mg, 5 mg and 10 mg tablets allow decrements of **1 mg or 0.5 mg**.
- **Concept.** Substitute **stepwise, one dose at a time starting at night**, because diazepam needs several days to accumulate and an abrupt full switch leaves a trough.
- **Key number.** Then decelerate: **5% to 10% of the current dose every 1 to 2 weeks**. From a 40 mg start that means 2 to 4 mg steps down to 20 mg, 1 to 2 mg steps down to 10 mg, 1 mg steps down to 5 mg, and 0.5 mg steps below that until cessation.

RULE **Hold, do not escalate, and do not give as-needed rescue doses.** Symptoms wax and wane; a flare is not failure. Hold the current dose for weeks until it settles, then resume, and keep the trajectory one-directional.

TRAP Equivalence quoted from one table as if it were universal. Lorazepam 1 mg is equated to 10 mg of diazepam in one guideline and to 5 mg in another; cross-tolerance is incomplete, so titrate to prevent withdrawal without daytime sedation before any reduction starts, and say which table you are using.

TRAP Substituting diazepam in hepatic impairment, or reading expected metabolites as diversion. In liver failure diazepam accumulates through oxidative metabolism, so taper the original agent (lorazepam, oxazepam and temazepam are conjugated and less dependent on liver function) by up to a tenth of the daily dose every 1 to 2 weeks. Finding oxazepam and temazepam in the urine of someone tapering on diazepam is its expected metabolism, not misuse.

INDIA CONTEXT

Benzodiazepines are dispensed here under the restricted schedules of the drug rules, which means a documented prescription register and controlled supply; the exact schedule labels are recorded as unsourced in the library, so name the mechanism and verify the schedule letter before the exam.

HOLD THIS

Both stations turn on proportional steps: 5% to 10% of the current dose, not a fixed milligram cut, because receptor occupancy is hyperbolic. Discontinuation appears within 1 to 2 days and resolves on rechallenge; relapse takes weeks and does not.

WHAT EARNS THE MARKS

- **Names the three activation safeguards**: it is the safety-critical part of the station.
- **Separates akathisia from worsening depression**: the response is reduction, not escalation.
- **Explains hyperbolic occupancy in one sentence**: it justifies proportional steps.
- **Separates dependence from addiction**: it is the sentence that reassures the patient.

SEE ALSO 09 Lithium counselling, and the bipolar relapse plan 11 Risk stations: suicide, agitation, and the liaison call 12 Behaviour change and explaining a therapy

11 · Risk stations: suicide, agitation, and the liaison call

COMMUNICATION STATIONS · TOPIC 11

Assess this patient's suicide risk, manage the agitated patient next door, then hand over by telephone.

RISK STATION, AGITATION STATION, HANDOVER STATION

THE SHAPE OF THE ANSWER

1 Frame the question	Seen alone, confidentiality with its limit, positive framing
2 Work the four windows	Presenting event, recent weeks, past, immediate and future
3 Intent against lethality	Ask what they expected the act to do
4 Safety plan	Six steps with means restriction, not a contract
5 Agitation and handover	Verbal first, oral before parenteral, then a recommendation

Ask the question in the direction that permits an answer. Interview alone: ask accompanying relatives to step out, introduce yourself by name and role, and validate the distress.

- **Concept.** Set the **confidentiality frame with its limit** before the sensitive questions start, in one sentence: everything here is confidential, but if there is imminent risk of serious harm to you or to someone else, such as a child, I will share what is needed with my senior team to keep you safe.
- **Concept.** Then move in with **praise and normalisation** rather than a jump.

GATEWAY QUESTION	WHAT PATIENTS DO WITH IT	CONSEQUENCE
Negatively framed ("no thoughts of harming yourself?")	66% answer no, 9.7% answer yes, 24.2% give an ambivalent narrative	False-negative denials rise, and the clinician moves on without further assessment in 60% of encounters
Positively framed ("do you feel that life is not worth living?")	42.9% answer no, 38.1% answer yes, 19% give a narrative	Disclosure becomes possible without inflating reported suicidality

In recorded outpatient consultations 75% of gateway questions were framed negatively and only 25% positively. About 75% of people who die by suicide minimise or deny ideation at their last contact with services, which is the reason the framing is scored.

The validity techniques, with a script each. Six of them, and the examiner is listening for the wording.

- **Concept. Normalisation.** "When people are under this kind of financial strain and humiliation, thoughts of ending life are common; have you had thoughts like that?"

- **Concept. Gentle assumption.** "What other methods have you considered?" Used cautiously with suggestible patients.
- **Concept. Shame attenuation.** "With all the pain you have been carrying, it is understandable to want it to stop; have you had thoughts of suicide to make it stop?"
- **Concept. Symptom amplification.** "On the worst days, how much of the day do the urges take, ten hours, twelve hours?"
- **Concept. Behavioural incident.** "Did you put the rope around your neck?", "exactly how many tablets came out of the strip?", "what happened straight after that?"
- **Concept. Denial of the specific.** Hanging, then overdose, then jumping, asked one at a time with an answer taken for each, rather than offered as a list.

1. **Presenting event.** The crisis from beginning to end: trigger, thought content, substances, setting, actions, whether alone, precautions against discovery, and the feeling on surviving, relief or regret.
2. **Recent events.** The preceding 8-week window, where planning and weighing happen: methods contemplated, steps taken to secure means, time spent ruminating, how close they came.
3. **Past events.** The most lethal attempt in the lifetime, the approximate number of attempts, medical intervention required, and the triggers then.
4. **Immediate and future.** Ideation in the room now, hopelessness, what returning home will feel like, and what they will do if the thoughts come back today, with likelihood over the next 24 to 48 hours.

Intent against lethality. Medical lethality is the damage the method objectively did; intent is what the person expected it to do.

- **Concept.** A small ingestion taken in the firm belief that it was fatal carries **high intent**. A larger impulsive ingestion with no expectation of death can cause more harm with less intent.
- **Concept.** So ask it directly: **"did you think it would kill you?", "did you want to die?"**
- **Concept. Markers of high intent**: extensive planning rather than impulse, final acts such as a will, a note, giving away possessions or closing contracts, precautions against discovery, avoiding or resisting rescue, and regret at surviving.
- **Trap.** Reading **non-suicidal self-injury** as an attempt. It is done to relieve unbearable affect or to end dissociation, so ask whether they hoped to die or were trying to cope, while remembering that repeated self-injury is itself a risk factor for later attempts.

FACTOR	REPORTED INCREASE
Previous suicide attempt	**40-fold, and the strongest single predictor**
Anorexia nervosa	25-fold
Severe depression	20-fold
Recreational opiate misuse or dependence	14-fold
Haemodialysis	14-fold
Alcohol dependence	6-fold
Demographic and social	Male, age greater than 45 years, widowed, separated, single or divorced, living alone or isolated, unemployed or low income

Protective factors and the scaling pair. Ask what has kept them from acting so far: faith, children, family responsibility, future goals, a pet.

- **Key number.** Then **scale twice**: intensity now from 1 to 10, and likelihood of acting over the next 24 to 48 hours from 1 to 10.
- **Concept.** If they say four, ask **both anchors**: what keeps it from being five or six, which gives the protective factors, and why not two or three, which gives the modifiable risks and triggers.

RULE **A safety plan replaces a no-suicide contract.** Contracting for safety is unproven, indefensible and hazardous because it manufactures reassurance and suppresses disclosure. Write six steps instead: recognise warning signs, internal coping, social distraction, trusted contacts, professional and crisis contacts, and means restriction. Then ask the safety question: "do you believe you need help right now to keep yourself safe?"

STRATUM	PICTURE	DISPOSITION
Imminent risk	Active persistent ideation with intent, a concrete plan, access to means, severe hopelessness or active psychosis	**Emergency admission with constant observation and not left unattended**
Raised risk without imminence	Ideation with some intent but protective factors intact, no immediate lethal plan, support available	**Safety plan reinforced, means restricted, review preferably within 72 hours and a telephone check within 48 hours**
Low risk	Passive death wishes without intent, plan or preparation, strong protective factors	Crisis contacts, coping strategies, routine outpatient follow-up

TRAP Chasing a yes-or-no answer when the patient gives a narrative. Ambivalence ("I just want the pain to stop") is the disclosure; stay with the conflict between wanting to die and wanting relief. The other common failure is spending the station on distant attempts in chronic suicidality: take enough past history to inform disposition and protect time for the recent window, present intent and the safety plan.

TRAP Indian epidemiology and the method profile. Every prevalence figure and risk multiplier available here comes from United Kingdom, United States or global data, and the Indian method distribution is marked unsourced, so do not recite a foreign method profile for an Indian patient when means restriction depends on it. Name the national crime records source and verify the figures before the exam.

The agitated patient, second half of the same territory. Early agitation has **four features**: inability to remain calm, motor or verbal hyperactivity, emotional tension, and difficulty communicating.

- **Concept. Secure the environment first**: an unobstructed exit, heavy objects and potential weapons removed, no enclosed isolated space.
- **Concept. Screen reversible causes in parallel**, hypoglycaemia, hypoxia, intoxication, withdrawal and metabolic derangement, with vital signs and a bedside glucose as soon as engagement allows.

Verbal de-escalation is first-line, with relaxed posture, open visible hands, eye level where feasible, personal space preserved and an even tone. The CALMER sequence gives it structure.

- **Concept. C, calm yourself** before anything else.
- **Concept. A, acknowledge the grievance** by agreeing with a fact, "I agree, you have waited a long time", or a principle, "I agree, everyone deserves prompt and respectful attention".
- **Concept. L, listen** with open prompts, and altercast a positive identity: "you sound like a parent who wants the best for your family".
- **Concept. M, mirror** the primary emotion under the anger.
- **Concept. E, empower** with concrete non-coercive choices: water, food, a blanket, tablet or liquid, a telephone call to a relative.
- **Concept. R, take responsibility** by committing to one specific action with a time attached.

Rapid tranquillisation is the administration of a benzodiazepine, an antipsychotic or both by the parenteral route, or repeated oral dosing within 60 minutes.

- **Concept.** A single as-needed oral dose is **not** rapid tranquillisation, and the goal is calm with preserved consciousness and conversation, not sedation to stupor.
- **Key number.** Offer **oral first** and allow about 1 hour to judge the response.
- **Concept.** Reserve the **intravenous route** for a general hospital setting with a cannula in situ, airway-skilled personnel present, consultant approval, and flumazenil prescribed at the bedside.

DRUG	ADULT DOSE	OLDER ADULT	REPEAT INTERVAL AND 24-HOUR MAXIMUM	DISCRIMINATOR
Lorazepam, oral or intramuscular (dilute 1:1)	1 to 2 mg	0.5 to 1 mg	30 to 60 minutes; adult 4 to 8 mg, older adult 2 mg	**First-line; no active metabolites; keep flumazenil available**
Haloperidol with promethazine	5 mg, with promethazine 50 mg	0.5 mg (1 mg if not frail), with promethazine 25 mg	60 minutes; adult 10 to 20 mg, older adult 2.5 to 5 mg	**Baseline electrocardiogram needed; avoid in Lewy body disease and Parkinson disease**

DRUG	ADULT DOSE	OLDER ADULT	REPEAT INTERVAL AND 24-HOUR MAXIMUM	DISCRIMINATOR
Olanzapine, intramuscular	10 mg (range 5 to 10 mg)	2.5 to 5 mg	2 hours; adult 20 mg; maximum 3 injections in 24 hours and 3 consecutive days	**Not within 1 hour of a parenteral benzodiazepine**
Aripiprazole, intramuscular	9.75 mg (range 5.25 to 15 mg)	5.25 mg	2 hours; 30 mg for adult and older adult	**May be combined with a benzodiazepine**
Midazolam, intramuscular	5 to 7.5 mg (2.5 mg approximates 1 mg lorazepam)	2.5 mg	Adult 15 mg, older adult 5 mg	Reserve for when intramuscular lorazepam is unavailable

Cardiac gate for haloperidol. Upper limits of normal QTc are 440 milliseconds in men and 470 in women, and any QT-prolonging psychotropic is withheld above 500 milliseconds.

- **Key number.** If no normal tracing exists within the preceding 1 month, or an electrocardiogram cannot be obtained, choose **intramuscular lorazepam or olanzapine** instead.
- **Concept.** **Promethazine** is added for sedation and for central anticholinergic protection against acute dystonia.
- **Concept.** Drugs are **not mixed in one syringe**, and the prescription states single dose, route, minimum interval and 24-hour ceiling.

After administration, observations every **15 minutes for at least 1 hour** and until the patient is stable and alert, with continuous oximetry if asleep.

- **Key number.** **What you record.** Consciousness on the alert-confused-voice-pain-unresponsive scale, respiratory rate, pulse and rhythm, blood pressure, oxygen saturation, temperature and hydration.
- **Key number.** **Cover for the ward.** Resuscitation equipment within 3 minutes, a doctor able to attend within 30 minutes, and senior review within 24 hours of any episode.
- **Key number.** **Escalate** at a respiratory rate below 10 per minute or saturation below 94%, an orthostatic diastolic drop of 30 mmHg, systolic below 90 mmHg or diastolic below 50 mmHg, pulse of 140 or more or below 50.
- **Key number.** Temperature above **38 degrees C with rigidity** means withholding antipsychotics and checking creatine kinase.
- **Key number.** **Flumazenil** is 200 micrograms intravenously over 15 seconds, then 100 micrograms every 60 seconds as needed, to a usual 300 to 600 micrograms and a maximum of 1 mg in 24 hours. It is contraindicated in epilepsy maintained on benzodiazepines.
- **Key number.** **Acute dystonia**: benztropine 1 to 2 mg intramuscularly or intravenously, or procyclidine 5 to 10 mg, 2.5 to 5 mg in adolescents and older patients.

INDIA CONTEXT

Physical restraint needs a coordinated team of at least 5 trained staff, 1 at the head managing airway and neck and 1 for each of the 4 limbs, with prone restraint avoided because of positional asphyxia.

Section 97(2) of the Mental Healthcare Act 2017 sets the standard: physical restraint is not to be used for a period longer than is absolutely necessary to prevent the immediate risk of significant harm.

It is not a sanction, a convenience or a substitute for staffing, and it requires medical authorisation, continuous monitoring and documented justification. Scottish and other foreign statutes have no standing here.

- **TRAP** Giving intramuscular olanzapine within 1 hour of intramuscular lorazepam or midazolam. The combination can produce profound central depression, hypotension, bradycardia and cardiorespiratory arrest. If a benzodiazepine has been given, choose aripiprazole or haloperidol with promethazine, or wait the interval. Two further scored errors: haloperidol alone without promethazine and without a recent electrocardiogram, and using zuclopenthixol acetate for an acute unheralded crisis, where its slow onset and long action make it unsuitable and it is contraindicated in neuroleptic-naive patients.

The liaison call. The same territory produces a handover station: you telephone a medical colleague about a delirious inpatient.

- **Concept.** Use **SBAR**, which came from naval handoffs and entered healthcare in 2003 at Kaiser Permanente, and which levels the hierarchy that otherwise produces hinting and hoping.
- **Key number.** Its force is in the evidence: in an analysis of 936 sentinel events in 2015, **communication failure was the root cause in more than 70%** of serious adverse events.
- **Key number.** Across 46 articles describing 24 handoff mnemonics, this one accounted for about **69.6% of the literature**.

STAGE	WHAT YOU SAY
Situation	Your name, grade and unit, the patient, the bed, resuscitation status, and the reason you are calling now, with the abnormal observation that triggered it
Background	Admission date and diagnosis, procedures, relevant medical and psychiatric history, baseline cognition, current drugs and allergies
Assessment	Consolidated observations, bedside mental state and cognitive findings, and the suspected cause, or a clear statement of concern if the cause is unclear
Recommendation	**Named, time-bound requests: the tests you want now, the nursing placement, the drug cautions, and when you will review**
Readback	**The receiver repeats the situation, assessment and verbal orders before the call ends**

For a delirium call the **recommendation block has a fixed shape**, and it is four requests and a review time.

- **Concept. Investigations now**: bedside glucose, electrolytes, renal panel and arterial gas.
- **Concept. Environment**: a well-lit room near the nursing station, a family attendant, reorientation cues, glasses and hearing aids, daytime light, minimal night-time interruption, hydration, nutrition, mobilisation, and bowel and bladder care.
- **Concept. Restraint avoided** unless there is imminent danger to life or to lines.
- **Concept. Medication review** for anticholinergics, sedatives, steroids and polypharmacy.
- **Concept.** If agitation threatens essential care, **low-dose haloperidol** with the baseline QT interval checked, with benzodiazepines avoided unless this is alcohol or sedative withdrawal.
- **Concept.** Then give your **review time** and ask for the readback.

TRAP Omitting the recommendation, which is missing in 50% of real handoffs, or dumping the psychiatric history instead of a headline. The other failure is prescribing before investigating: an oxygen saturation of 88 per cent, a blood pressure of 85/50 mmHg or an unmeasured glucose is the problem, not the agitation.

HOLD THIS

Positively framed question, behavioural detail, then a written safety plan with means restriction. In agitation, verbal first, oral before parenteral, lorazepam first-line, and no intramuscular olanzapine inside 1 hour of a parenteral benzodiazepine. On the phone, end with a recommendation and a readback.

WHAT EARNS THE MARKS

- **Frames the gateway question positively**: negative framing produces denial and abandonment.
- **Takes behavioural detail rather than a yes or no**: ambivalence is the disclosure.
- **Writes a safety plan with means restriction**: contracting for safety is indefensible.
- **Ends the telephone call with a recommendation and readback**: the recommendation is the part most often missing.

SEE ALSO 09 Lithium counselling, and the bipolar relapse plan 10 Starting an antidepressant, stopping a benzodiazepine 14 Eliciting the mental state on a simulated patient

12 · Behaviour change and explaining a therapy

COMMUNICATION STATIONS · TOPIC 12

Raise this man's drinking with him, and explain cognitive behavioural therapy to the next patient.

ALCOHOL STATION, INSOMNIA STATION, EXPLAINING-THERAPY STATION

THE SHAPE OF THE ANSWER

1 Spirit and processes	Partnership, acceptance, compassion, evocation across four processes
2 OARS	Open questions, affirmations, reflections, summaries that invite correction
3 Evoke change talk	Use a ruler, then ask why it is not lower
4 Insomnia	Diagnose it, formulate with the three P model, then components
5 Explaining therapy	Ask expectations, give the frame, state the confidentiality limits

These three stations are marked on restraint. The alcohol station, the insomnia station and the explaining-therapy station all fail the candidate who instructs. The behaviours that score are eliciting, reflecting and negotiating, with information given only after permission.

Motivational interviewing is a **directive, person-centred style for resolving ambivalence**, in which resistance is read as a mismatch between the clinician's pace and the person's readiness rather than as a trait.

- **Concept.** Its spirit is **PACE**: partnership between two experts, acceptance, compassion, and evocation of what is already there.
- **Concept. Acceptance** unpacks into absolute worth, accurate empathy, autonomy support and affirmation.
- **Concept.** The four early principles map onto the four current processes: express empathy becomes **engaging**, develop discrepancy becomes focusing, with agenda mapping across 3 to 5 concerns, roll with resistance becomes evoking, and support self-efficacy becomes planning.

STAGE	WHAT THE PERSON SAYS	YOUR GOAL IN THIS STATION
Precontemplation	No intention to change; harm minimised	**Build rapport and raise doubt without arguing**
Contemplation	Acknowledges harm, feels two ways	**Explore ambivalence and tip the balance**
Preparation	Decided, planning steps	Choose an achievable goal and troubleshoot barriers
Action	Changing patterns and triggers	Bolster self-efficacy
Maintenance	Change held for at least 6 months	Stabilise and restructure lifestyle
Recurrence	Return to previous drinking	**Frame as a learning point and re-enter the cycle, not as failure**

OARS, and the reflections that earn marks. Open questions ("what part does alcohol play in your evenings?"), affirmations of attributes rather than your own approval ("you showed persistence coming back after last week"), reflections, and summaries that end with "did I miss anything?".

- **Concept. Simple** restatement of what was said.
- **Concept. Complex feeling**: "you feel frustrated because the watching makes you feel untrusted".
- **Concept. Complex meaning**: "you value being a reliable parent, and the routine is cutting across that".
- **Concept. Double-sided**, joining both sides with "and" rather than "but": "drinking helps you unwind, and you are worried about the liver results".
- **Concept. Amplified**, echoing sustain talk without sarcasm so the person argues the other side.
- **Concept. Reframing**, for example reading high tolerance not as strength but as a lost warning signal.

Change talk is sorted as **DARN** preparatory talk, desire, ability, reasons and need, and **CAT** mobilising talk, commitment, activation and taking steps.

- **Concept.** Evoke it with an **importance or confidence ruler**: "on a scale of 0 to 5, with 5 the highest, how important is changing your drinking?"

- **Concept.** Follow it immediately with the question that forces change talk: **"what makes it a 5 and not a 0?"**
- **Concept.** Then **querying extremes**, looking back and looking forward, and contrasting values with behaviour.

The **decisional balance** runs in a fixed order that keeps resistance low: the good things about drinking first, then the less good things, then the downsides of changing, then the benefits of changing, closing with a double-sided summary that makes the discrepancy audible.

For a brief intervention in a general setting, use **FRAMES**: feedback on the screening score and physical markers, responsibility left with the person, advice only after permission through elicit-provide-elicit, a menu of options, empathy, and self-efficacy.

MNEMONIC

Five traps

Expert, labelling, question-and-answer, premature focus, blaming. Each one is a scored failure: taking over, insisting on the word alcoholic, interrogating, pushing cessation before the person's agenda is known, and arguing about whose fault it was.

Evidence for the viva: **motivational enhancement therapy** is the manualised variant of 3 to 4 sessions adding structured assessment and personalised normative feedback.

- **Key number.** A Cochrane review of **93 trials with 22,776 participants** found reduced substance use against no intervention, standardised mean difference 0.48, 95% CI 0.07 to 0.89, and at 1 year 0.12, 95% CI 0.05 to 0.20.
- **Key number.** Beyond 1 year results were **similar to controls**, 0.12, 95% CI 0 to 0.25, with outcomes comparable to active treatments such as cognitive behavioural therapy for less clinical time.
- **Key number.** A group trial in **118 veterans** lowered heavy drinking episodes, relative risk 0.74, 95% CI 0.59 to 0.91, and drinking days, 0.79, 95% CI 0.67 to 0.94, at 3-month follow-up.
- **Key number.** Delivery is **1 to 4 sessions of 15 to 45 minutes** with a minimum useful dose of 20 minutes, and skills decay after workshops unless practice is coded with 3 to 4 coaching sessions over a 6-month period.

The insomnia station. Diagnose before you advise.

- **Concept. The criterion set.** Dissatisfaction with sleep quantity or quality, with difficulty initiating sleep, maintaining it, or early waking with inability to return to sleep.
- **Concept.** It causes **distress or impairment**, occurs despite adequate opportunity, at least 3 nights per week for at least 3 months, and is not explained by another sleep-wake disorder, substance or comorbid condition.
- **Trap.** Missing **reduced sleep need in hypomania**, where the person sleeps briefly, wakes energised and wants no more sleep, while the person with insomnia pursues sleep and suffers for the lack of it.

Multicomponent cognitive behavioural therapy for insomnia is first-line, delivered across 4 to 8 sessions of 30 to 60 minutes, weekly or every 2 weeks.

- **Key number.** Improvement is usually visible inside **6 to 8 weeks**, with meaningful benefit in up to 80% of those treated and hypnotics reduced or stopped in up to 90%.
- **Key number.** Gains **persist after treatment ends**, unlike pharmacotherapy with its tolerance and rebound.

Formulate with the 3P model: predisposing traits, precipitating stressors, and the perpetuating adjustments (more time in bed, naps, evening alcohol, stimulating activity in the bedroom) that turn an acute disturbance into conditioned arousal, which is why the patient is exhausted on the couch and "tired but wired" in bed.

COMPONENT	THE INSTRUCTION YOU GIVE	DISCRIMINATOR
Stimulus control	Go to bed only when sleepy rather than fatigued; bed for sleep and intimacy only; if sleep has not come in about 15 to 20 minutes, leave the bedroom for a quiet dimly lit activity and return only when sleepy, repeating as often as needed; fixed rising time 7 days a week; naps avoided, or limited to 15 to 30 minutes about 7 to 9 hours after rising	**Sleepiness is struggling to stay awake; fatigue is low energy**
Sleep restriction	Match prescribed time in bed to the diary-derived total sleep time, fix the rising time and count the bedtime backwards, then add 15 minutes per week once sleep efficiency reaches 85%	**The absolute floor is 5 hours in bed**
Cognitive restructuring	Test the catastrophic predictions about a sleepless night and reframe awakenings as manageable	Reduces the pre-sleep arousal that hygiene cannot touch
Relaxation	Progressive muscle relaxation, diaphragmatic breathing, autogenic training, imagery	**Exercises longer than 15 minutes are done outside the bedroom**
Sleep hygiene	Same rising time daily, only as much time in bed as needed, daytime exercise but not late, a dark quiet cool room, alcohol limited and caffeine avoided after midday, no forcing sleep	**Adjunct only; advised against as sole treatment**

The Indian adaptation of hygiene advice. Late family dinners, heavy evening tea and coffee, noisy multi-generational households and long afternoon siestas are the practical targets, and they are worth naming because generic advice about a dark quiet bedroom often does not survive the household.

TRAP Prescribing sleep restriction without screening the contraindications. Sleep loss can precipitate a manic switch in bipolar disorder, lowers the seizure threshold in epilepsy, and is unsafe for commercial drivers and machinery operators. And the floor of 5 hours in bed is not negotiated downward however short the reported sleep.

Explaining a therapy. Ask first what the patient expects from therapy, because many arrive expecting passive listening, moral guidance or advice.

- **Concept.** Then give the frame in plain words: a **structured, time-limited treatment** in which we look at how thoughts, feelings, physical sensations and actions keep each other going, test those patterns against what actually happens, and learn tools you can use.
- **Concept.** Name the **three principles**: some of the problem lies in unhelpful thinking, some in learned unhelpful behaviour, and better coping can be learned.
- **Concept.** Say that the focus is the **present maintaining cycle** rather than an extended tour of the past.

QUESTION	WHAT YOU TELL THE PATIENT
How long?	Typically short-term, approximately 5 to 20 sessions, often across a few months, with the exact number and the session length agreed together
What happens in a session?	Mood check, homework review, agenda set together, skills work or problem solving, new homework agreed, then feedback on the session
Homework?	**Designed together, not set; self-monitoring, thought records, behavioural experiments, graded approach to what is avoided, reading, and calming practice**
Will it work?	It may not remove an illness or a painful situation, but it builds skills that change how you cope; progress needs practice and is not linear
Could it make me worse?	**Examining painful material and facing avoided situations raises distress temporarily, which settles as tolerance develops; if there is no progress after several sessions the formulation and plan are reviewed**
And my tablets?	Therapy runs alone or alongside medication; drugs can reduce symptom severity enough to engage with the work, and changes are coordinated with the treating psychiatrist

Collaborative empiricism is the phrase to use out loud: the two of you are co-investigators who turn beliefs into testable hypotheses and design real-world experiments, so that the patient ends up able to do this without you.

Close by stating confidentiality and its limits: explicit intent or plans to end one's life, clear plans to harm another person, disclosure or reasonable suspicion of abuse or neglect of a child or a vulnerable adult, and inability to meet basic survival needs safely.

TRAP Three failures in one station: lecturing a definition instead of checking comprehension, promising a cure, and drifting into childhood conflict and unconscious defences when the model being explained is about present maintaining factors. A fourth loses the same marks: leaving out the confidentiality exceptions.

HOLD THIS

Elicit, provide, elicit: information is offered only after permission, in all three stations. A ruler followed by "why not lower?" produces change talk; sleep hygiene alone is not treatment; and homework is designed with the patient, not assigned to them.

WHAT EARNS THE MARKS

- **Asks permission before giving information**: elicit, provide, elicit is the scored pattern.
- **Reads resistance as a pace mismatch**: arguing the other side loses the station.
- **Diagnoses insomnia before advising on it**: hygiene alone is not treatment.
- **Designs homework with the patient**: assigned homework is not collaborative empiricism.

SEE ALSO 10 Starting an antidepressant, stopping a benzodiazepine

11 Risk stations: suicide, agitation, and the liaison call 17 Relaxation and behavioural techniques on demand

Skill stations

13 · Consent and capacity as a bedside skill

SKILL STATIONS · TOPIC 13

Assess whether this inpatient has capacity to refuse admission, and take consent for the procedure.

SKILL STATION, BEDSIDE CAPACITY ASSESSMENT, STATUTE VIVA

THE SHAPE OF THE ANSWER

1	Five disclosure elements	Diagnosis, procedure, risks, alternatives, consequences of refusal
2	Two legal standards	The professional standard and the reasonable patient standard
3	Diagnostic stage	Is there an impairment or disturbance of mind or brain
4	Functional stage	Understand, retain, weigh and communicate this decision now
5	Indian furniture	Advance directive, nominated representative, Form CR-A, the Board

Consent is a conversation with five contents, and capacity is a two-stage test. Informed consent is an interactive process ending in voluntary authorisation for a specific intervention; treating without it is battery or actionable negligence. The disclosure has to be in lay language and to cover five elements.

ELEMENT	WHAT IS DISCLOSED
Diagnosis	The clinical diagnosis, including any diagnostic uncertainty
Nature and purpose	What the intervention is, its steps and its intended goal, in plain words
Risks and consequences	**Commonly occurring risks and serious morbidity or mortality; remote minor risks need not be itemised**
Alternatives	Medically viable options, including ones outside your own preference
Prognosis without treatment	**What happens if the person declines**

Add the practical details: who will be involved, expected recovery, and follow-up.

- **Key number.** Courts test adequacy against **2 standards**. The professional standard is what a prudent practitioner would customarily disclose, and it needs expert testimony.
- **Key number.** The **reasonable patient standard** is what a reasonable person in that position would consider material, where expert testimony is not strictly required.
- **Concept.** They also examine **proximate cause**: whether a reasonable person would have declined had disclosure been complete.
- **Concept.** Indian jurisprudence has adopted a **balanced-information standard** aligned with the professional test: normal risks and alternatives, not an exhaustive catalogue of remote contingencies.

The capacity station itself. Prepare before the bedside.

- **Concept. Read the record** for functional baseline, current medical instability, recent sedatives or anticholinergics, and previous assessments.
- **Concept. Identify the precise decision**: consent for a cannula, refusal of admission.
- **Concept. Pick the window** that avoids post-sedation drowsiness.
- **Concept. Open properly**: hand hygiene, your full name and role, identity confirmed with 2 identifiers, the purpose in plain language, privacy, and the sensory aids or a neutral interpreter if needed.

Then the two stages. First the diagnostic stage: is there an impairment or disturbance of mind or brain, progressive (dementia), permanent (brain injury) or temporary (delirium, intoxication)? Second the functional stage: does that impairment stop this decision being made at this time?

ABILITY	HOW YOU TEST IT OUT LOUD
Understanding	"Could you tell me in your own words why the team has recommended admission?" "What might happen if the infection is not treated?"
Retaining	"A moment ago we went through the plan; could you recap the main risks and benefits?"
Weighing up	**"How did you balance the risk of serious complications against wanting to go home today?"**
Communicating	"Having thought about it, what is your decision?" Speech, writing, signing or clear gesture all count

- **RULE** **Capacity is presumed, decision-specific and time-specific, and an unwise decision is not an incapacitous one.** If the person understands the risks including death, retains the information, reasons through their own values and communicates a choice free of undue influence, the refusal stands.

The agitated or delirious inpatient who wants to leave. Work a stepped protocol, and say the steps in order.

- **Concept.** A **structured cognitive screen** including orientation and attention, months of the year backwards, and a brief validated delirium screen.
- **Concept.** **Verbal de-escalation** that names the real worry, the pets, the locked house, and offers a pragmatic compromise.
- **Concept.** **Supportive delirium measures** with lighting, repeated reorientation and sensory aids, plus collateral from family.
- **Key number.** **Proportionate restraint or low-dose pharmacotherapy as a last resort**, with haloperidol 0.5 to 1 mg used cautiously, avoided in parkinsonian disorders, and the baseline QT interval checked.
- **Trap.** **Benzodiazepines in the older delirious patient**, which are generally avoided because of paradoxical worsening.
- **Concept.** Then **senior and liaison escalation**, with documentation and a reassessment time.

INDIA CONTEXT

Advance directives sit in Chapter 3 of the Mental Healthcare Act 2017, which runs from Sections 5 to 13, that is 9 sections.

A directive is made in writing by a person who is a major, at 18 years, with capacity, and past illness or admission does not disqualify them. A legal guardian may make one for a minor.

Form CR-A is the prescribed format, the nominated representative signs to confirm willingness, 2 witnesses attest signature in the maker's physical presence, and registration with the Board carries no fee.

The electronic register under Section 91 clause (a) is to receive the directive within 14 days. A directive is invoked only on loss of capacity and lapses when capacity returns, and amendment or cancellation follows the same procedure as making it.

A treating psychiatrist cannot set a directive aside unilaterally. The application goes to the Board, which alone may alter, modify or cancel it, and emergency treatment under Section 103 is the stated exemption.

Where no representative has been appointed, Section 14(4) deems, in order: the person named in the advance directive, a relative, a care-giver, a suitable person appointed by the Board, and failing all of those the Director of the Department of Social Welfare or a designated representative.

A registered organisation's representative can serve as a temporary nominated representative pending appointment, and for a minor the legal guardian is the representative unless the Board orders otherwise.

Research consent, which is a different animal. Treatment consent aims at the patient's own benefit and rests on **beneficence**. Research consent aims at generalisable knowledge and rests on

protection against exploitation, with unconditional withdrawal at any stage without prejudice to ordinary care.

- **Concept.** The Indian ethical guidelines require consent as an **ongoing dialogue rather than a signature**, with a participant information sheet and consent form in simple language in the participant's own language.
- **Concept.** **What is disclosed**: objectives, methods, randomisation and placebo, risks, direct benefits, confidentiality measures, compensation for study-related injury, and freedom to withdraw, with ethics committee approval before enrolment.
- **Concept.** **Vulnerability is the examinable word**: psychiatric disorder, dementia or delirium affecting capacity; minors under age 18, who give assent while a guardian consents; institutionalised people under hierarchical control; and economically disadvantaged, non-literate or subordinate participants.
- **Concept.** **Protections**: research on them only when scientifically indispensable, capacity assessed before enrolment, a legally authorised representative where capacity is impaired, and an impartial witness signature for non-literate participants.
- **Trap.** Leaving **therapeutic misconception** unaddressed, which is the belief that research procedures were designed for that individual's benefit.

Therapeutic privilege and informed refusal. Privilege is a narrow exception for clear evidence that disclosure itself would inflict severe harm on an unusually fragile person. It is not cover for paternalism, not a route around a predicted refusal, and it requires a documented rationale.

- **Concept.** **Informed refusal is the mirror image of consent**: explore the concerns, explain the risk of non-treatment, offer alternatives, avoid abandonment, and document.
- **Key number.** Documentation follows **8 principles**, of which four get asked.
- **Concept.** Explain terms in **lay language**, and list the commonly occurring risks.
- **Concept.** State that **no outcome is promised**, and do not call a procedure simple or minor.
- **Concept.** Take **timed and dated signatures** from both parties, with a witness who attests that the discussion happened.

TRAP Importing capacity law from another jurisdiction. Deprivation of liberty safeguards, lasting powers of attorney and independent advocates have no place in an Indian answer; the Indian instruments are the nominated representative, the Mental Health Review Board, Form CR-A and the electronic register. Two more scored errors: treating a diagnosis as incapacity, and using euphemisms when testing understanding. Examiners expect the word death to be said out loud when serious complications are explained.

TRAP Believing a signed form is consent. The signature is evidence; if the dialogue omitted risks and alternatives, the consent is invalid, and a general admission form covers routine non-invasive care only, not an invasive procedure, a psychotropic trial or electroconvulsive therapy. Keep competence (a legal status a court decides) separate from capacity (a clinical determination for one decision at one time).

HOLD THIS

Five disclosure elements, two legal standards, two stages and four abilities. Capacity is presumed and decision-specific; an unwise decision by a capacitous person is respected; and the Indian statutory furniture is the advance directive, the nominated representative and the Board.

WHAT EARNS THE MARKS

- **States that capacity is presumed and decision-specific**: an unwise decision is not an incapacitous one.
- **Says the word death out loud**: euphemism when testing understanding is a scored error.
- **Names the Indian instruments, not foreign ones**: imported safeguards have no standing here.
- **Treats the signature as evidence, not consent**: an undisclosed risk invalidates the form.

SEE ALSO 07 Explaining ECT and taking consent 15 Cognitive testing at the bedside 20 Confidentiality, the duty to warn, and a decision model

14 · Eliciting the mental state on a simulated patient

SKILL STATIONS · TOPIC 14

Elicit the mental state of this patient with a first episode of psychosis, in front of the examiner.

SKILL STATION, SIMULATED PATIENT, PHENOMENOLOGY VIVA

THE SHAPE OF THE ANSWER

1	Safety and rapport	Introduce, state the purpose, set the confidentiality frame
2	Open before structured	Sample thought stream, affect and cooperativeness first
3	Ownership and agency	Insertion, made thoughts, withdrawal and broadcasting, each with a script
4	Obsession against delusion	Resistance and ownership settle the distinction
5	Finish on risk	Suicidality, then the question about the children

This station is scored on your verbatim questions. Safety first, then rapport: introduce yourself, state the purpose, and set the confidentiality frame as in any clinical encounter.

- **Concept.** If others are present, ask whether the patient would rather speak with them there or alone, which **hands back some control**.
- **Concept.** Start **open**, to sample thought stream, affect and cooperativeness before any structured probe.

With a guarded or suspicious patient, **check comfort first**: "do you feel safe sitting here with me, or does having other people present make you uncomfortable?"

Then normalise the transition: "this period may feel frightening, and people who come to see us often describe hearing unusual voices or sensing strange things around them; has anything like that happened to you?"

TARGET	THE QUESTION YOU SAY	WHAT YOU ARE SEPARATING
Referential ideas	"When you walk down the street or enter a room, do you sense that strangers are watching you, whispering about you, or hinting about what you have done?"	Self-reference before persecution
Media reference	"Does it seem as though television programmes, the radio or newspapers carry messages meant specifically for you?"	A reliable opener the patient recognises

TARGET	THE QUESTION YOU SAY	WHAT YOU ARE SEPARATING
Persecutory belief	"Do you believe particular people or groups are watching you, following you, or trying to harm you?" then "how certain are you that it is happening this way, and could there be another explanation?"	**Conviction, preoccupation and insight, without argument**
Delusional perception	"Have ordinary sights or everyday events suddenly carried a hidden meaning meant just for you?" then anchor it: "when you saw that car pass your gate, what went through your mind at that exact moment? Did the car do anything unusual, or did you realise instantly, just from seeing it, that your life was in danger?"	**The two-stage structure: a normal percept, then an unintelligible meaning**
Auditory hallucination	"When you are sitting quietly alone, do you hear sounds or voices when nobody is nearby and nothing in the room accounts for them?"	External acoustic space against inner speech
Third person and running commentary	"Do the voices speak to you as 'you', or do two or more voices speak about you as 'he' or 'she'? Do they argue about you?" and "does a voice describe what you are doing as you do it?"	**Schneiderian form rather than content**
Thought echo	"Do you experience your own unspoken thoughts being spoken aloud or repeated so that you can hear them?"	An auditory experience, not a belief about broadcasting

Thought interference, the discrimination examiners chase. Disturbances of possession run in **two directions across the ego boundary**. Inward gives thought insertion and made thoughts; outward gives withdrawal and broadcasting.

- **Concept. Insertion has two components**: the boundary is intruded upon, and ownership of the thought is alien, with mental agency bypassed so that another entity did the thinking.
- **Concept.** In **made thoughts** the process feels manipulated from outside, but the thought remains the patient's own.
- **Trap.** The term **thought alienation** has been used for both in the older literature, and is best avoided.

PHENOMENON	OWNERSHIP AND AGENCY	SCRIPT
Thought insertion	**Ownership alien, agency bypassed**	"Do you find thoughts in your head that are not your own, put there from outside? Whose are they, and how do they reach you?"
Made thoughts	**Ownership retained, agency influenced**	"Does an outside force play on your mind and force you to think certain things, even though the thoughts are still yours?"
Thought withdrawal	Ownership personal, thoughts removed	"Do thoughts get taken out of your head by an outside power, leaving your mind blank?"
Thought broadcasting	Ownership personal, thoughts escape	"Do your thoughts leave your head so that people around you know what you are thinking without you speaking?"

Specificity matters when the examiner asks whether first-rank symptoms are pathognomonic.

- **Key number.** **Thought insertion** is elicited in 19% of patients with schizophrenia, 7.2% with mood disorders, 8% with brief reactive psychoses and 6% with atypical psychoses.
- **Key number.** **Made thoughts** occur in 35.8%, 27.7%, 32% and 18% of those same groups respectively.
- **Concept.** So they are **symptoms of psychosis across diagnoses**, not markers of one. Under ICD-10, thought insertion sustained for at least 1 month in the absence of affective or organic disorder suffices for the diagnosis.

Passivity phenomena. One script for each, and the wording is what is scored.

- **Concept.** **Made feelings**: "do you get sudden feelings, grief or rage, that do not feel like your own, as though they were manufactured and put into your chest from outside?"
- **Concept.** **Made impulses**: "do you get a sudden urge to act where the urge itself was planted in you?"
- **Concept.** **Made acts**: "do your limbs or body move as though an outside operator were working them, with you watching?"
- **Concept.** **Somatic passivity**: "do you feel physical sensations, vibrations, burning or pinpricks, that you know are being directed into you from outside?"

Obsessions, and the line against psychosis. Screen with "do you find you have to do things in an exact order, or check locks and appliances repeatedly, or struggle with upsetting thoughts that keep coming back?"

- **Concept.** Then ask about **intrusiveness and resistance**.
- **Concept.** Then **ownership and origin**: "do you recognise these as products of your own mind, even though they are unwelcome?"
- **Concept.** Then the **neutralising compulsions** and the feared consequence of not performing them, and insight into senselessness.

FEATURE	OBSESSION	DELUSION	THOUGHT INSERTION
Ownership	**Own mind, recognised as such**	Own, and believed	**Alien, belongs to another agency**
Mental agency	Intact	Intact	Bypassed
Resistance	**Resisted, egodystonic**	Not resisted, incorporated into reality	Not resisted, experienced as intrusion
Insight	Usually recognised as excessive	Absent, conviction unshakeable	Held as a real external violation

Mood, and the two risk questions that finish the station. Ask pervasive mood over the past month, anhedonia, worthlessness, guilt and the view of the future.

- **Concept.** Then step through suicidality: **passive** ("have you had thoughts that you would rather not wake up?"), active, then plan, preparation and means, then protective factors.
- **Concept.** Nearly half of patients who end up with a diagnosis of schizophrenia **present initially with depressive symptoms** meeting criteria for major depression, and depressive symptoms in early psychosis raise suicidal ideation and acts. So this is not an optional limb.
- **Concept.** In a patient with young children, add the **protective homicide question** gently and directly: "do you ever feel the world is so dangerous or painful that you need to protect your children, even if that meant taking them away from this world with you?"

Numbers for the follow-on viva, in three groups.

- **Key number. Course.** Most first-episode patients present within the first 2 years of onset, the first 2 to 3 years are clinically unstable, and maximum symptomatic improvement occurs in the first 6 to 12 months of treatment.
- **Key number. Outcome.** Over 80% relapse, about two-thirds have moderate to poor long-term outcomes, and one-third a favourable course.
- **Key number. Workup.** Full blood count, electrolytes including sodium, renal and liver panels, thyroid screen, syphilis and retroviral serology, a supervised urine drug screen, and neuroimaging if focal signs appear.
- **Key number. Follow-up.** A stable patient reviewed in the community is seen after 1 week to judge efficacy and tolerability, and treatment continues up to 1 year after full remission.

TRAP Quoting the older manual's criteria as current. The criteria set available in the library is the DSM-IV-TR table, including its single-symptom shortcut for bizarre delusions or a commenting voice or two voices conversing. Name the edition you are quoting, and read the current DSM-5-TR wording from the manual before the exam; it is marked unsourced here.

TRAP Calling an intrusive obsession thought insertion, and calling thought echo broadcasting. Ownership settles the first: in obsessional illness the patient knows the thought is theirs and fights it. Modality settles the second: echo is heard, broadcasting is believed.

HOLD THIS

Ask about form, not just content, and let the patient locate ownership and agency for you. Every psychosis station ends with suicide risk, and in a parent it ends with the question about the children.

WHAT EARNS THE MARKS

- **Uses verbatim questions the examiner can hear**: this station is scored on your wording.
- **Lets the patient locate ownership and agency**: that is what separates insertion from made thoughts.
- **Normalises before probing unusual experiences**: a guarded patient discloses to a normalised question.
- **Closes on suicidality in a parent with the homicide question**: leaving it out forfeits the safety marks.

SEE ALSO 06 Explaining schizophrenia, and expressed emotion 11 Risk stations: suicide, agitation, and the liaison call 15 Cognitive testing at the bedside

15 · Cognitive testing at the bedside

SKILL STATIONS · TOPIC 15

Perform a bedside cognitive assessment on this patient, then interpret and defend the score.

SKILL STATION, OSPE, SCORING VIVA

THE SHAPE OF THE ANSWER

1 Choose the instrument	Suspected pathology and literacy decide which screen
2 Administer to script	Permission first, no hints, a fixed number of repeats
3 Score the awkward items	Backwards spelling by order, subtraction errors, the figure
4 Adjust	Prorate omitted items, add the education and age points
5 The Indian caveat	Literacy-loaded items push the score falsely low

Choose the instrument before you administer one. Selection turns on the suspected pathology, cortical against subcortical or frontal, and on literacy. The examination-relevant contrast is simple: the older screen is weighted to orientation, language and verbal memory and misses mild impairment, while the newer one samples executive function, visuospatial skill and abstraction and catches it.

INSTRUMENT	BEST USED FOR	LIMITATION THE EXAMINER WANTS NAMED
Mini-mental state examination	General dementia screening and tracking progression	**Sensitivity for mild cognitive impairment only 18% to 25%, with a marked ceiling effect**
Montreal cognitive assessment	Mild cognitive impairment, vascular and subcortical presentations; validated for ages 55 to 85 years	**Educational bias on the standard version, and it needs literacy**
Hindi mental state examination	Low-literacy and rural screening, with literacy-dependent items replaced	**Total and cut-offs are unsourced here; verify before quoting**
Addenbrooke's cognitive examination	Detailed profiling and dementia subtyping in a memory clinic	**Total, cut-offs and subscale weights are unsourced here; verify before quoting**

The mini-mental state examination has **11 items across 7 cognitive domains for a maximum of 30 points.**

- **Key number.** **Orientation** to time 5, orientation to place 5.
- **Key number.** **Registration** of 3 unrelated objects named at 1-second intervals, 3.
- **Key number.** **Attention and calculation** 5, by serial sevens or spelling a five-letter word backwards, then delayed recall of the 3 words without cues, 3.
- **Key number.** **Language** 8: naming 2, repetition of a phonetically dense phrase 1, a three-stage command 3, reading and performing a written instruction 1, writing a sentence with a subject and verb 1.
- **Key number.** **Copying intersecting pentagons**, 1.

Administration rules that carry marks. Seat the patient facing you with glasses and hearing aids on.

- **Concept.** **Ask permission**: "would it be all right to ask you some questions about your memory?" It avoids a catastrophic reaction.
- **Key number.** **Score zero immediately** for a wrong answer, without hinting or shaking your head, and repeat an instruction a maximum of 3 times if asked.
- **Concept.** Deflect "what is this for?" with a neutral line about **explaining once you are finished**.
- **Key number.** Standardised administration reduces intra-rater variability by 86% and inter-rater variance by 76%, lifting the **intraclass correlation from 0.69 to 0.90**, which is why the script is fixed.

SCORING QUESTION	THE RULE
Serial sevens	**Allow 5 subtractions (93, 86, 79, 72, 65) uninterrupted; penalise only the erroneous step, so 93, 85, 78, 71, 64 scores 4 out of 5 because each later subtraction correctly removes 7 from the patient's own previous answer**
Spelling backwards	**Score order, not sequence: write the correct reversal, place the patient's letters beneath, join identical letters with non-crossing lines, and score the maximum number of non-crossing lines; this single item carries 17% of the total**
Pentagons	**One point needs 10 angles and an overlap forming a four-sided figure with 2 points of intersection; tremulous or curved lines still score if the geometry is there**
Writing and reading	An intelligible sentence with subject and verb scores, spelling and grammar errors notwithstanding; reading the instruction aloud without closing the eyes scores zero
Self-correction	Immediate spontaneous self-correction is credited across items

Adjustments, the part candidates skip. Where a physical or sensory disability prevents an item, **omit it and prorate**: the adjusted score is the actual score multiplied by 30 and divided by the maximum obtainable.

- **Key number.** A **blind patient** who cannot read, write a sentence or copy the figure loses 3 items, so the maximum obtainable is 27, and a raw score of 15 becomes 15 multiplied by 30 divided by 27, which is 16.66 and rounds to 17.
- **Key number.** Fractions of **0.5 or more round up**.
- **Concept.** For a **unilateral amputation**, adapt the three-stage command to crumpling and dropping the paper with one hand.
- **Key number.** **Demographic points.** Add 1 point for fewer than 12 years of formal education, and 1 point from 75 or 80 years of age.

BAND OR POPULATION	SCORE	NOTE
No impairment	24 to 30	Ceiling effect lives here: 71.4% of healthy and mildly impaired individuals score 28 to 30
Mild impairment	18 to 23	Cut-off 23/24 is the most widely supported for dementia
Moderate impairment	12 to 17	Alternative frameworks give 11 to 20
Severe impairment	0 to 11	**Floor effect: 29% of severe cases cluster at 0 to 6, so decline can no longer be tracked**
Schooling-adjusted cut-offs	**Illiterate 21 (sensitivity 93%, specificity 82%), low education 22 (87%, 82%), middle 23 (86%, 87%), high 24 (81%, 87%)**	The answer when the patient has had no schooling and only this instrument is available
Parkinson disease dementia	26 of 30 identified as optimal, with 26 giving sensitivity 79.9% and specificity 74.0% in one study	Annual decline averages 2.3 points, and a fall of 1 to 3 points is clinically meaningful

The montreal cognitive assessment totals **30 points with sensitivity around 90%** for mild cognitive impairment.

- **Key number.** **The domains.** Visuospatial and executive 5, with alternating trails, cube and clock; naming 3; attention 6, with digit span 2, vigilance 1 and serial sevens 3; language 3, with repetition 2 and phonemic fluency 1; abstraction 2; delayed recall of 5 words; orientation 6.
- **Key number.** **The clock** scores 1 each for contour, numbers and hands, and numbers arranged in a circle without a drawn contour score zero for contour.
- **Concept.** **Calculation is mental**, with no pencil, paper or finger counting.
- **Key number.** **Exactly 2 learning trials** are given, and alternate versions exist for retesting within 3 months to limit practice effects.
- **Key number.** **Add 1 point** for 12 years of formal education or fewer, counting years after kindergarten, and not beyond the maximum of 30.

INTERPRETATION OR ADAPTATION	RULE
Normal cognition	26 and above
Mild cognitive impairment	18 to 25, averaging 22 with a range of 19 to 25
Moderate then severe impairment	10 to 17, then 0 to 9
Separating impairment from dementia	**A score of 18 is the dividing line, with mild Alzheimer disease averaging 16, range 11 to 21**
Blind or telephone version	**Visual items omitted, total 22 points, normal from 19; prorate as raw multiplied by 30 divided by 22, so 19 of 22 becomes 25.9 and rounds to 26**
Basic version for low education	A cut-off below 24 of 30 gave 81% sensitivity and 86% specificity for mild cognitive impairment
Hemiplegia	**Written items omitted, scored out of 25, prorated as raw multiplied by 30 divided by 25, so 22 of 25 becomes 26.4 and rounds to 26**

INDIA CONTEXT

Administering a literacy-loaded screen to an illiterate or semi-literate patient produces a falsely low score, because spelling, reading, writing and the intersecting figure all depend on schooling.

Say that out loud and offer the alternative: the Hindi mental state examination, or the low-education version of the newer screen, with orientation items using local seasons and administrative units, village, panchayat, taluk, district, state, and familiar household objects for registration.

If only the standard instrument is to hand, use the schooling-adjusted cut-off of 21 for illiteracy rather than the usual 24.

TRAP Cueing during delayed recall, adding an education point to a perfect score, or counting kindergarten as a year of schooling. Each one is a mechanical error the examiner can see. The clinical version of the same mistake is dismissing memory complaints in an educated patient scoring 28 of 30, where sensitivity for mild impairment is as low as 18%.

TRAP The examiner will ask for the Hindi screen's total and cut-offs, the detailed battery's total, its two cut-offs and its five subscale weights, and the composition and cut-off of the briefest bedside screen. All of these are marked unsourced in the library, so verify them against a primary reference before the exam instead of quoting a number in the station.

HOLD THIS

Score order for the backwards spelling, penalise only the erroneous subtraction, and prorate omitted items rather than scoring them zero. The standard screen has a ceiling, so a normal score in an educated patient with complaints means moving to the executive instrument.

WHAT EARNS THE MARKS

- **Asks permission before testing memory**: it avoids a catastrophic reaction and is on the sheet.
- **Prorates an omitted item instead of scoring zero**: the arithmetic is the examinable part.
- **Declines to cue during delayed recall**: cueing is a mechanical error the examiner can see.
- **Moves to the executive screen when the basic one is normal**: the older screen has a ceiling.

SEE ALSO 05 Breaking bad news, and the dementia family meeting 13 Consent and capacity as a bedside skill 16 AIMS and the other movement scales

16 · AIMS and the other movement scales

SKILL STATIONS · TOPIC 16

Examine this patient on long-term antipsychotics for abnormal involuntary movements and score the scale.

SKILL STATION, OSPE, MOVEMENT DISORDER VIVA

THE SHAPE OF THE ANSWER

1 Set the room	Hard chair without arms, mouth empty, dentures asked about
2 Observe, then activate	Unobtrusive observation first, then the activation manoeuvres
3 Score	Highest severity seen, minus a point if only on activation
4 Read the total	Sum the anatomical items and apply the screen threshold
5 Separate the syndromes	Dyskinesia, akathisia, parkinsonism, acute dystonia

Set the room up before you touch the patient. The abnormal involuntary movement scale is a **12-item clinician-rated instrument from 1976** that records involuntary movements, chiefly tardive dyskinesia, and tracks severity over time in anyone exposed to dopamine-blocking drugs.

- Key number. Administration takes about **5 minutes**, and it needs a hard firm chair without arms, because armrests hide swaying, pelvic shifts and unsupported limb posturing.
- Concept. **Observe unobtrusively at rest** before or after the structured examination.
- Concept. Ask them to **remove gum or sweets** from the mouth, ask about the state of the teeth, and ask whether dentures are worn and currently uncomfortable.
- Concept. Ask whether they **notice the movements**, and whether these interfere with daily life.

STEP	INSTRUCTION	PURPOSE
1 to 3	Clear the mouth, ask about teeth and dentures, ask about awareness and interference	Remove mechanical confounders and record subjective distress
4	Sit with hands on knees, legs slightly apart, feet flat	Whole body at rest in a defined posture
5	Sit with hands hanging unsupported	**Unmasks digital, wrist and choreic hand movements**
6 and 7	Open the mouth wide, then protrude the tongue, each twice	Tongue at rest inside the cavity, then in movement

STEP	INSTRUCTION	PURPOSE
8	Tap thumb with each finger as fast as possible for 10 to 15 seconds, right then left	**Activation: unmasks facial grimacing and leg movements**
9	Examiner passively flexes and extends each arm	Tone and rigidity
10	Stand up	Posture in profile, spine and hips
11	Extend both arms forward, palms down	**Activation: unmasks trunk, leg and mouth movements**
12	Walk a few paces, turn and return, twice	**Activation: unmasks distal choreoathetosis and gait change**

The 12 items fall into **five sections**.

- Key number. **Facial and oral**: muscles of facial expression, lips and perioral area, jaw, tongue.
- Key number. **Extremities**, upper then lower, and **trunk**, neck, shoulders and hips.
- Key number. **Global judgments**: overall severity, incapacitation, and the patient's awareness and distress.
- Key number. **Dental status**: current teeth or denture problems, and whether dentures are usually worn.
- Key number. **Scoring.** Items 1 to 10 are rated 0 none, 1 minimal or possibly extreme normal, 2 mild, 3 moderate, 4 severe, with item 10 anchored from no awareness to awareness with severe distress. Items 11 and 12 are yes or no.

RULE **Rate the highest severity you observed, then apply the activation discount.** If a movement appeared only during finger tapping, arm extension or walking and was absent spontaneously, subtract 1 point from that item. Forgetting this inflates apparent spontaneous dyskinesia.

The **anatomical total is the sum of items 1 to 7**, and it is kept for monitoring rather than announced to the patient.

- Key number. Research practice operationalises **probable tardive dyskinesia** as a score of 3 or more on at least 1 of the first 7 items, or 2 or more on 2 or more of them.
- Concept. **No single number dictates intervention.** Management follows functional impairment and distress, and may mean dose reduction, switching to a lower-risk agent, or a vesicular monoamine transporter inhibitor.
- Key number. Screening is recommended at baseline and **every 3 to 12 months** during maintenance antipsychotic treatment.

SCALE	STRUCTURE	SCORING AND CUT-OFF
Simpson-Angus scale, from 1970	10 items: gait, arm dropping, shoulder shaking, elbow rigidity, wrist rigidity, head rotation, glabella tap, tremor, salivation, akathisia	**Each item 0 to 4; the total is the sum divided by 10 to give a mean item score, and a mean above 3 indicates drug-induced parkinsonism**
Barnes akathisia rating scale	4 items: objective restlessness, subjective awareness of restlessness, subjective distress, and a global clinical judgment	**First three scored 0 to 3, global 0 to 5; a global score of 2 or more defines akathisia, with 3 or more used as the trial threshold**

Course figures for **akathisia**, which the examiner asks for as a pair of percentages.

- **Key number.** It develops in up to **50% of cases within 1 month** and 90% within 3 months of starting an antipsychotic.
- **Key number.** First-generation rates are **15% to 40% in trials**, 21% to 75% in general practice, against 0% to 12% with second-generation agents.
- **Key number.** **Pseudoakathisia**, objective restlessness without subjective awareness or distress, occurs in about 24% of mixed inpatient cohorts on first-generation drugs.

FEATURE	TARDIVE DYSKINESIA	AKATHISIA	DRUG-INDUCED PARKINSONISM	ACUTE DYSTONIA
Phenomenology	Involuntary non-rhythmic choreoathetoid movements	Inner urge to move with semi-purposeful restlessness	Bradykinesia, rigidity, rest tremor	Sustained painful contraction
Site	**Orofacial first: tongue, lips, jaw, then trunk and distal limbs**	**Lower limbs: shuffling, pacing, weight shifting**	Upper limbs, axial muscles, facial masking	**Craniocervical: torticollis, retrocollis, trismus, oculogyric crisis**
Subjective state	Often unaware, distress variable	Severe inner tension and dysphoria	Heaviness, slowness, stiffness	Pain and panic
Effect of activation	**Provoked or increased by voluntary movement elsewhere**	Pacing gives temporary relief	Activated by contralateral motor tasks	Unaffected
Anticholinergic response	**Often worsens or becomes unmasked**	Variable, mainly if parkinsonism coexists	Improves substantially	Resolves rapidly, which is also diagnostic

TRAP Scoring tremor under the upper extremity item. The instruction excludes tremor, which is regular and rhythmic and belongs to the parkinsonism scale. Two more mechanical errors: penalising inability to sustain tongue protrusion when the item rates only increased movement, and summing the parkinsonism scale instead of dividing by 10.

TRAP Reading oral movements in an edentulous mouth or under loose dentures as dyskinesia, which is why the dental items exist. And clinically, reading akathisia as worsening psychosis: raising the antipsychotic dose then worsens the akathisia and the suicide risk that goes with it.

HOLD THIS

Hard chair without arms, three activation manoeuvres, and a 1-point discount for movements seen only on activation. Sum items 1 to 7 for the anatomical total; 3 or more on one item, or 2 or more on two, is a positive screen.

WHAT EARNS THE MARKS

- **Uses a hard chair without arms and says why**: armrests hide the movements the scale is looking for.
- **Applies the activation discount**: forgetting it inflates apparent spontaneous dyskinesia.
- **Keeps tremor out of the extremity item**: tremor is rhythmic and belongs to the parkinsonism scale.
- **Checks the mouth and dentures first**: an edentulous mouth mimics dyskinesia.

SEE ALSO 06 Explaining schizophrenia, and expressed emotion 08 Clozapine counselling 15 Cognitive testing at the bedside

17 · Relaxation and behavioural techniques on demand

SKILL STATIONS · TOPIC 17

Teach this patient progressive muscle relaxation, then set up her activity schedule.

SKILL STATION, DEMONSTRATION STATION

THE SHAPE OF THE ANSWER

1	**Mechanism**	Tension and arousal run together, so discrimination is the target
2	**Prepare aloud**	Quiet room, supported posture, loosened clothing, centring breaths
3	**The cycle**	Tense, release suddenly, observe, and name your protocol
4	**Desensitisation**	Hierarchy, relaxation, graded images, erase the screen
5	**Activation**	Routine, necessary and pleasurable, scheduled with the four Ws

You will be asked to do it, not to describe it. Progressive muscle relaxation rests on a **bidirectional link between muscle tension and autonomic arousal**: sustained contraction maintains sympathetic activation, and deliberate release recruits parasympathetic activity.

- **Concept.** The therapeutic target is **discrimination**, training the patient to tell a contracted muscle from a relaxed one, because people under sustained tension stop detecting their own baseline.

Prepare the setting **out loud**, as you would in clinic.

- **Concept.** A **quiet room** with subdued light and comfortable temperature.
- **Concept.** The patient **supported** in an armchair with arms resting and feet flat, or supine.
- **Concept.** Tight collars, belts, spectacles and footwear **loosened or removed**, and eyes gently closed with no straining.
- **Concept.** A **centring start** of 5 slow abdominal breaths, or 3 deep breaths in through the nose and out through the mouth.

PHASE	INSTRUCTION AND TIMING	DISCRIMINATOR
Tense	Inhale and contract the group firmly without straining for 5 seconds (some scripts allow 5 to 10 seconds), counting silently	**Firm, not painful; cramp and strain are errors**
Release	Exhale and let the contraction go abruptly and completely, so the limb goes limp	**Sudden release, not a gradual easing**
Observe	Rest 10 seconds (some scripts 10 to 20 seconds), attention on the contrast, warmth and heaviness	**Skipping this phase loses the whole mechanism**
Close	2 minutes of stillness, count backwards from 5 to 1, slow breaths, gentle stretch, eyes open, sit up slowly before standing	Prevents orthostatic dizziness

Work the groups in a **fixed order**, head to foot or foot to head: hands, biceps, triceps, forehead, eyes and central face, jaw and cheeks, tongue against the palate, posterior then anterior neck, shoulders, chest, abdomen, back, buttocks, thighs, calves, feet and toes.

- Key number. **Name the protocol you are demonstrating.** The shortened full-body script covers 14 major muscle groups, while the 20-minute hospital protocol used in Indian practice sets out 17 discrete tensing steps.
- Trap. A candidate who **quotes one count and performs the other** gets pulled up.
- Key number. Prescribe daily practice of **10 to 15 minutes**, once or twice a day while the technique is being learnt.

Indications, cautions, and the passive variant. Indications: generalised anxiety, panic disorder, insomnia, tension headache and migraine, essential hypertension, chronic pain including temporomandibular dysfunction, irritable bowel syndrome, and distress or anticipatory nausea in cancer care.

- Trap. **Withhold active tensing** with acute fractures, severe muscle tears or sprains, unstable spinal disc pathology, recent surgical incisions across the target region, and active severe spasm.
- Concept. In those cases use **passive relaxation**, dropping the contraction phase and guiding the patient to notice, release and soften existing tension.
- Concept. Where time is short, a **shortened protocol** targets 3 to 5 tension-holding zones such as shoulders, neck and jaw.

Diaphragmatic breathing, demonstrated in four lines. One hand on the chest and one over the abdomen above the navel, sitting upright with shoulders dropped.

- Concept. **Inhale slowly through the nose** into the belly, so that the lower hand rises while the chest stays still.
- Concept. Brief pause, then **release smoothly through the mouth** with a soft sigh, feeling the belly settle.
- Concept. Pair the exhalation with a **silent cue word**.

Systematic desensitisation. Wolpe's counterconditioning paradigm rests on **reciprocal inhibition**: if a response incompatible with anxiety, deep muscular relaxation, is elicited in the

presence of the anxiety-evoking stimulus, the conditioned link weakens.

- **Concept.** Later work showed the same fear reduction can be explained by **respondent extinction, habituation or improved self-efficacy**, which is worth saying because it shows you know the mechanism is contested.
- **Key number.** **Suitable candidates** have 4 phobic fears or fewer, can visualise vividly, and report clear discomfort when imagining the feared cue.

1. **Build the hierarchy.** Rate items on the subjective units of disturbance scale, which runs from 1, complete calm, to 100, the worst imaginable panic. Generate triggers together and vary distance, duration, intensity and social complexity, then order them from lowest to the ultimate feared situation.
2. **Induce relaxation.** Progressive muscle relaxation or diaphragmatic breathing first.
3. **Present the lowest unmastered item** and hold the image briefly, a few minutes.
4. **Manage breakthrough anxiety.** On the patient's signal: "erase the screen, wipe the image away completely, and bring your attention back to your breathing and your muscles."
5. **Re-establish and re-expose** until the scene evokes little or no anxiety, then move up one step. Expect to move to real-life exposure for full resolution.

Behavioural activation and activity scheduling. Four steps, and the examiner wants all four.

- **Concept. Identify activities across 3 categories.** Routine, such as dishes, cooking, laundry and basic housework; necessary, such as bills, appointments and official correspondence, the things that compound if postponed; and pleasurable, a friend, a walk, a hobby, something creative.
- **Concept. Rank them** easy, moderate and difficult.
- **Concept. Schedule them** in a weekly diary specifying the four Ws, what exactly, when and for how long, where, and with whom, with mood recorded immediately before and after, and mastery and pleasure rated in the classical cognitive behavioural version.
- **Concept. Implement**, review the diary weekly, name the obstacles, and step the activity up.

OBSTACLE	WHAT IS HAPPENING	THE FIX YOU OFFER
Too difficult to start	High initial friction, anticipatory fatigue	**The 5-minute rule: commit to 5 minutes and permit stopping after that**
Task feels too large	Executive overload, global defeatism	Break it into small distinct increments
No energy or motivation	Waiting for motivation before acting	**Switch from task-contingent to time-specific scheduling, and fix sleep and meal routines**
No time	Poor prioritisation, role conflict	Prioritise, and enforce the balance across the three categories
No mood improvement afterwards	Anhedonia, negative filtering	**Act from the outside in: perform the scheduled action regardless of how you feel rather than waiting to feel better**

TRAP Demonstrating passive relaxation or autogenic training when asked for the progressive method, which requires active isometric contraction before release. The timing errors cost the same marks: tensing far too long or to the point of pain, and rushing on without the 10-second observation phase that the whole technique depends on.

TRAP Confusing desensitisation with modern exposure. In desensitisation relaxation is used deliberately to inhibit anxiety and the image is withdrawn if anxiety breaks through; in prolonged exposure relaxation is omitted and withdrawal is avoided so that extinction and inhibitory learning can happen. Say which you are doing. And do not build an activity schedule made only of pleasant hobbies or only of overdue chores.

HOLD THIS

Tense 5 seconds, release suddenly, observe 10 seconds, and name the protocol you are demonstrating. A hierarchy is rated 1 to 100, breakthrough anxiety means erase the screen, and every activity schedule balances routine, necessary and pleasurable.

WHAT EARNS THE MARKS

- **Demonstrates active contraction before release**: the passive variant answers a different instruction.
- **Holds the observation phase after each release**: discrimination is what the technique trains.
- **Names the protocol and its group count**: quoting one count and performing another is caught.
- **Balances the activity schedule across three categories**: chores only, or hobbies only, loses the mark.

SEE ALSO 10 Starting an antidepressant, stopping a benzodiazepine 12 Behaviour change and explaining a therapy 14 Eliciting the mental state on a simulated patient

Professionalism stations

18 · Which code is in force, and the social media rules

PROFESSIONALISM STATIONS · TOPIC 18

Which code of professional conduct governs you today, and what does it say about social media?

PROFESSIONALISM STATION, ETHICS VIVA

THE SHAPE OF THE ANSWER

1 The dates	Notified in August 2023, in abeyance three weeks later
2 What governs now	The 2002 conduct, etiquette and ethics regulations
3 The digital directives	Eleven points, and still the examinable content
4 The four prohibitions	No individual advice, no images, no testimonials, no bought visibility

Answer the regulatory question with a date, not an impression. The professional conduct regulations for registered medical practitioners were **notified in the Gazette on August 2, 2023**.

- **Concept.** They introduced a mandate to **prescribe by generic name**, with licence suspension for up to one month as the penalty.

- **Concept.** **Regulation 35** barred practitioners from taking part in third-party educational events sponsored directly or indirectly by the pharmaceutical or allied healthcare industry.
- **Concept.** They also replaced the older oath with the **physician's pledge**.

After opposition from professional bodies over bioequivalence and drug quality standards, and the cancellation of scientific conferences, a further Gazette notification on **August 23, 2023 placed those regulations in abeyance** until further notice.

The regulator clarified that the professional conduct, etiquette and ethics regulations of **2002 stand reinstated** and remain in force with immediate effect. The parent statute, the commission's own Act of 2019, governs human medical practice.

INSTRUMENT	STATUS TO STATE	WHAT IT GOVERNS
Professional conduct regulations of 2023	**In abeyance since August 23, 2023**	Generic prescribing, Regulation 35 on sponsorship, social media directives
Professional conduct, etiquette and ethics regulations of 2002	**The operative legal standard**	Core ethical standards, prohibition of commissions, gifts and fee-splitting

The digital conduct directives, which are still the examinable content. Eleven points were framed alongside the 2023 regulations, and they are the answer to any social media station.

- **Concept.** Share **factual, verifiable educational material**, and avoid content that is deceptive, misleading or exploitative of vulnerability.
- **Concept.** Do not discuss **individual treatment** or prescribe on public platforms. Redirect an approach online into a structured telemedicine consultation or a clinic appointment.
- **Concept.** Do not post **patient photographs, scans or clinical imagery**.
- **Concept.** Keep interactions with **colleagues** within medical ethics and mutual respect.
- **Concept.** Do not **buy followers, likes or search rankings**, and do not register with platforms that sell patient leads or rating boosts.
- **Concept.** Do not solicit, circulate or publish **patient testimonials** or endorsements.
- **Concept.** Do not publish **procedural footage** or before-and-after images of treated patients.
- **Concept.** Keep public educational material inside **your own verified field**.
- **Concept.** Treat your **clinic website and institutional pages** under the same rules as social media.
- **Concept.** Maintain **decorum** and prevent boundary violations online.
- **Concept.** Treat direct or indirect **solicitation of patients** through social networks as misconduct.

TRAP Saying that the 2023 regulations are being enforced, or that patient consent makes a testimonial or an outcome video publishable. The regulations are in abeyance and the 2002 code governs, and the prohibition on testimonials, endorsements and outcome footage does not turn on consent.

HOLD THIS

Notified August 2, 2023, in abeyance from August 23, 2023, and the 2002 code is what you are answering under. No individual treatment advice, no patient images, no testimonials, no bought visibility.

WHAT EARNS THE MARKS

- **Gives both dates and the abeyance**: the question is asked to see whether you know the status.
- **States which code you are answering under**: answering under a suspended code is the trap.
- **Redirects an online approach into a proper consultation**: public advice on a platform is misconduct.
- **Says consent does not make a testimonial publishable**: the prohibition does not turn on consent.

SEE ALSO 19 Gifts, boundaries, and the internet 20 Confidentiality, the duty to warn, and a decision model 21 Safeguarding disclosures: domestic violence and POCSO

19 · Gifts, boundaries, and the internet

PROFESSIONALISM STATIONS · TOPIC 19

A patient offers you an expensive gift and sends a friend request. How do you handle each?

PROFESSIONALISM STATION, BOUNDARY VIVA

THE SHAPE OF THE ANSWER

1	Separate the two gifts	Industry gift is conflict of interest, a patient gift is a boundary
2	The three-factor test	Value, their motivation, and your own motivation
3	Crossing against violation	Intent and exploitation separate the two
4	The staged dilemmas	Friend request, chance encounter, physical contact
5	Prevention	Privacy controls, separate accounts, supervision, media disclaimer

Two different gift problems, and the examiner wants them separated. An **industry gift is a conflict of interest**: personal gifts, hospitality, travel, endorsement fees and referral commissions from healthcare manufacturers are misconduct under the operative code, and the suspended regulations went further by barring sponsored participation in continuing professional development.

- **Concept.** A **patient's gift is a boundary question** instead, usually arising from gratitude, cultural custom or transference.
- **Concept.** Weigh it on **three factors**: the monetary value, the patient's motivation for offering it, and your own motivation for accepting or declining.
- **Concept.** An inexpensive token at termination or during a festival can be a **benign crossing**. An expensive or intimate item, or a gift offered during acute symptom exacerbation, is explored tactfully and declined.

Boundaries rest on 4 ethical pillars: beneficence, nonmaleficence, respect for autonomy with justice, and fidelity or integrity. The distinction the station is built around is between a crossing and a violation.

PARAMETER	BOUNDARY CROSSING	BOUNDARY VIOLATION
Definition	Minor, non-exploitative deviation from convention intended to support care	Transgression that exploits the patient or serves the clinician at the patient's expense
Intent	**Clinical and benevolent**	**Self-serving: gratification, emotional reliance or gain**

PARAMETER	BOUNDARY CROSSING	BOUNDARY VIOLATION
Impact	Usually benign, and can strengthen the alliance when processed	Harmful, confusing, and corrosive of trust
Examples	An inexpensive festival token, brief non-intrusive self-disclosure to normalise distress, attending a major milestone	Sexual or romantic contact, business partnership, exploitative bartering, excessive intimate self-disclosure
Management	**Document the rationale, explore the meaning in session, take it to supervision**	**Stop immediately, supervision, institutional reporting, transfer of care**

The three dilemmas that get staged. A **friend request** from a patient creates a dual relationship and bidirectional access to personal life, so it is declined, personal and professional profiles are kept separate, and the reason is explained in session: the separation is what lets the patient disclose stigmatising material without social consequence.

- **Concept.** An **unplanned encounter** in a market, a concert or a restaurant is an inadvertent event rather than a violation. Protect confidentiality by not greeting or acknowledging the patient first unless they approach you, and avoid planned social meetings outside the professional setting.
- **Concept.** **Physical contact** carries a risk of misinterpretation, particularly with trauma histories, so a handshake or supportive gesture is offered with restraint and cultural awareness.

Privacy settings are part of professionalism now.

- **Key number.** An early study found **up to 80% of users** did not actively manage their settings, and an evaluation of medical trainees found only 37.5% used privacy safeguards.
- **Concept.** Where a patient discovers a personal profile or a dating profile, the revelation is addressed **analytically in the session**, exploring the patient's fantasies and curiosity, rather than defensively.

DIRECTION OF SEARCHING	STATUS	WHAT YOU DO
Patient searches the clinician	**Not a boundary violation, because patients are not bound by professional codes**	Respect autonomy rather than issuing rules, take your own reaction to supervision, and explore the meaning of the curiosity
Clinician searches the patient	**Not unethical in itself, but it risks rupture of trust**	Requires clinical justification or consent; reserve it for emergencies and risk assessment rather than routine, unannounced use

The preventive list. Nine recommendations are worth reciting in a boundaries viva.

- **Concept.** Activate **every available privacy control**, and search yourself periodically for inaccurate data or unauthorised images.
- **Concept.** Keep **confidential clinical material**, disparaging remarks about colleagues or patient groups, commentary on live legal proceedings and unprofessional photographs off your accounts.
- **Concept.** **Anticipate the clinical consequences** before searching a patient.
- **Concept.** Avoid online friendships and dual relationships by keeping **separate accounts**, and remember that pseudonymous comments remain traceable.

- **Concept. Teach digital professionalism** in residency, write institutional policy for online misconduct, and bring internet dilemmas into psychotherapy supervision.
- **Concept.** For **media work**, use an educational disclaimer, state that the commentary is not individual treatment, stay with evidence, and keep to the rule against offering a psychiatric opinion on a public figure you have not examined.

TRAP Calling every deviation a violation, or calling a patient's online search of you a violation. Benign, culturally syntonic crossings exist and can support the alliance when handled transparently, while the patient's search is autonomy and curiosity to be explored.

HOLD THIS

Intent and exploitation separate a crossing from a violation, and the test for a gift is value, their motive, your motive. Decline the friend request, keep the accounts separate, and explain why in the room.

WHAT EARNS THE MARKS

- **Weighs the gift on all three factors**: value alone does not settle it.
- **Declines the friend request and explains it in session**: the reason is what protects disclosure.
- **Calls a benign crossing a crossing**: labelling every deviation a violation is a scored error.
- **Treats the patient's search of you as material for the session**: it is curiosity to explore, not misconduct.

SEE ALSO 13 Consent and capacity as a bedside skill 18 Which code is in force, and the social media rules 20 Confidentiality, the duty to warn, and a decision model

20 · Confidentiality, the duty to warn, and a decision model

PROFESSIONALISM STATIONS · TOPIC 20

Your patient threatens a named neighbour. What do you disclose, and under what authority?

PROFESSIONALISM STATION, ETHICS AND STATUTE VIVA

THE SHAPE OF THE ANSWER

1 **The narrow grounds**	Imminent serious risk, statutory reporting, notifiable disease
2 **Name the domestic provision**	Section 23 and its listed exceptions, before any case
3 **The foreign case**	Cited for the concept of a duty to protect, not as authority
4 **The decision model**	Seven steps, said in order, with documentation

Confidentiality is a prerequisite, not an absolute. The grounds for breach are narrow and nameable: imminent life-threatening risk to the patient or another person, mandatory statutory reporting of suspected child abuse or vulnerable adult neglect, and notification of dangerous communicable disease. What matters in an Indian answer is naming the domestic provision before any foreign case.

INDIA CONTEXT

Confidentiality of mental health and treatment information sits in Section 23 of the Mental Healthcare Act 2017, and the provision carries its own exceptions.

Those exceptions include release to the nominated representative so that he can discharge his duties, and release where it is necessary to protect another person from harm or to prevent a threat to life.

Read that alongside Section 22, the right to information about the provision under which the person is admitted, the criteria for that admission, the right to apply to the Board for review, and the nature of the illness with the proposed treatment plan and its known side effects, in a language and form the person understands.

Then Section 25, the right to access basic medical records, which the professional in charge may withhold in part where disclosure would cause serious mental harm to the person or likely harm to others, the person then being told of the right to apply to the Board.

The duty to warn is **United States case law**, and it is cited for the concept rather than as binding authority here.

- **Concept.** It arose from the killing of Tatiana Tarasoff in **October 1969**: a graduate student disclosed his intention to kill her to his treating psychologist during outpatient therapy, the psychologist alerted the campus police, and neither she nor her family was told.
- **Concept.** The Supreme Court of California held that when a mental health professional determines that a patient presents a **serious and foreseeable danger of violence to an identifiable third party**, the clinician carries a duty to protect the intended victim.
- **Concept.** So the obligation of confidentiality sits **alongside a duty to protect**. Duty-to-warn and duty-to-protect statutes followed across United States jurisdictions.
- **Trap.** Presenting it as settled good practice. Critics note that **mandatory disclosure may deter volatile individuals** from seeking treatment.

TRAP Quoting the American case as Indian law, or treating the duty as licence for blanket disclosure to police or family. Name the domestic confidentiality provision first, and confine protective action to a credible threat of serious harm against an identifiable person. The often-quoted line from the judgment is not reproduced in the material available here, so paraphrase the principle rather than quoting it.

The model to reach for when the dilemma is genuinely hard. Seven steps, and saying them in order is itself a scored behaviour.

- **Concept. Identify the problem.**
- **Concept. Gather information** from the operative code, professional guidance, institutional rules, and a colleague or supervisor.
- **Concept. Identify the possible courses of action.**
- **Concept. Consider the consequences** of each for the patient, the therapeutic frame, the family and public safety.
- **Concept. Choose the course** that prioritises welfare, autonomy and nonmaleficence.
- **Concept. Implement it** with professional communication and contemporaneous documentation.
- **Concept. Review the outcome** and carry the lesson forward.

HOLD THIS

Name Section 23 and its exceptions before you name any foreign case. The duty to protect is triggered by a credible, foreseeable threat to an identifiable person, and the seven-step model is what you say out loud when the answer is not obvious.

WHAT EARNS THE MARKS

- **Names Section 23 first**: a foreign case quoted as Indian law is the trap.
- **Confines disclosure to a credible threat against an identifiable person**: it keeps the breach proportionate.
- **Reads the rights provisions together**: information, confidentiality and record access sit side by side.
- **Walks the seven steps out loud**: saying them in order is itself a scored behaviour.

SEE ALSO 13 Consent and capacity as a bedside skill 18 Which code is in force, and the social media rules 21 Safeguarding disclosures: domestic violence and POCSO

21 · Safeguarding disclosures: domestic violence and POCSO

PROFESSIONALISM STATIONS · TOPIC 21

One patient discloses violence by her husband; a child discloses abuse. What do you do in each?

PROFESSIONALISM STATION, SAFEGUARDING STATION, STATUTE VIVA

THE SHAPE OF THE ANSWER

1 Set the room	She is seen alone, with records kept off shared summaries
2 Ask behaviourally	Universal framing, neutral words, specific behaviours
3 Lethality	The five-item danger assessment and the homicide markers
4 The civil statute	Protection, residence, monetary relief and custody orders
5 The child	No private inquiry, report on knowledge, immunity in good faith

One station asks you to enquire safely; the other asks you to report without investigating. Both are marked on a statutory duty and on the words you choose.

For intimate partner violence, the enquiry happens **with the woman alone**, with partners, relatives, in-laws and friends outside the room, and with a professional interpreter rather than a family member if one is needed.

- Concept. Keep screening responses, diagnostic codes and referral details **off visit summaries, billing slips and any portal** an abusive partner can open.
- Concept. **Avoid the loaded words**, battered, abuse, rape, violence.
- Concept. Open with a **universal framing statement** that removes personal suspicion, then state confidentiality and its statutory limit before any question.

1. **Frame it.** "We have started talking to all of our patients about safe and healthy relationships, because it can have such a large impact on your health."
2. **State the limit.** Explain what you would have to pass on, naming the mandatory reporting duty for a child.

3. **Ask behaviourally.** "Has your current partner ever threatened you or made you feel afraid?" "Has your partner ever hit, choked or physically hurt you?" "Has your partner ever forced you to do something sexually that you did not want to do, or refused your request to use condoms?" "Does your partner support your decision about when or whether to become pregnant?" "Has your partner ever tampered with your birth control?" With a disabled patient, add whether a partner has withheld a wheelchair, cane or respirator, or threatened to withhold help with medicines, the bathroom, dressing or food.
4. **Assess lethality.** The five-item danger assessment: has the violence increased in frequency or severity over the past year; has the partner used or threatened a weapon; do you believe your partner is capable of killing you; has your partner ever tried to choke or strangle you; is your partner violently and constantly jealous. Non-fatal strangulation, weapon access, explicit threats to kill, recent estrangement or pending separation, and severe coercive control are the homicide markers.

SCREENING TOOL	STRUCTURE AND THRESHOLD	PERFORMANCE
Hurt, insult, threaten, scream	**4 items on a 5-point scale, range 4 to 20, abuse indicated at 11 or higher**	Sensitivity 86%, specificity 99%
Woman abuse screening tool	8 items on a 3-point scale, range 0 to 16, exposure indicated at 4 or higher	Sensitivity 47% to 88%, specificity 89% to 96%
Humiliation, afraid, rape, kick	4 items, dichotomous, positive on any affirmative answer	Sensitivity 81%, specificity 95%

Documentation is the medico-legal deliverable. Five things go into the record.

- **Concept.** **Verbatim statements in quotation marks**, avoiding editorial phrasing such as "claims" or "alleged".
- **Concept.** Injuries mapped on a **body diagram** with dimensions, shapes and colour changes.
- **Concept.** **Photographs with a scale** and an orientation landmark where protocol allows and written consent is documented.
- **Concept.** **Objective mental state observations.**
- **Concept.** The **communication channel she has agreed is safe**, because post, a text message or a call can each alert the abuser.

INDIA CONTEXT

The protective statute is the Protection of Women from Domestic Violence Act, 2005, a civil remedial law.

Section 3 defines domestic violence as any act, omission or commission that harms, injures or endangers the health, safety, life, limb or well-being, mental or physical, of the aggrieved person, that coerces her over unlawful dowry demands, or that threatens such harm.

It covers physical, verbal and emotional, economic and sexual abuse, including insults directed at a woman for not having a child or a male child.

Section 2(a) defines the aggrieved person and Section 2(q) the respondent, interpreted by the Supreme Court to include female relatives. Section 17 gives the right to reside in the shared household irrespective of title.

An application goes to the Magistrate under Section 12, with proceedings in camera on request.

The reliefs: Section 18 protection orders, Section 19 residence orders, Section 20 monetary relief for medical expenses, lost earnings, lost property and maintenance, and Section 21 temporary custody and visitation.

Breach of a protection order is cognizable and non-bailable under Section 31, punishable with imprisonment up to 1 year, a fine up to INR 20,000, or both.

Protection Officers are appointed in each district under Section 8, preferably women. They prepare the domestic incident report for the Magistrate, facilitate legal aid, coordinate medical examination and shelter, and execute monetary relief orders.

The criminal route on cruelty by a husband or his relatives runs in parallel and is listed among the aggrieved person's rights, so both can be invoked together.

For prevalence, quote the national family health survey finding that nearly one in three married women has experienced physical, emotional or sexual violence from a spouse, with almost 30% reporting some form of domestic violence while only a fraction seek formal help.

TRAP Reporting an adult woman's disclosure to the police without her consent. In a competent adult that breaches confidentiality and can provoke lethal retaliation; mandatory reporting attaches to minors and to vulnerable adults. Two more: reciting foreign prevalence figures as Indian data, and pressing a brochure or safety card into her hand to take home, where discovery can trigger assault. Offer a private institutional telephone and keep cards in private examination rooms or restrooms instead.

The child disclosure station is the opposite of discretion. Under the child protection statute of 2012 a child is anyone **below the age of 18 years**, and the reporting duty in Section 19(1) falls on any person, including the child, who has knowledge that an offence has been committed or apprehension that one is likely.

- **Concept.** Information goes to the **special juvenile police unit or the local police**.
- **Key number.** **Section 19(2)** requires the report to be given an entry number, recorded in writing, read over to the informant and entered in the register.

- **Key number.** **Sections 19(3) and 19(4)** require simple language, and an interpreter or translator where needed.
- **Key number.** **Section 19(5)** requires care and protection, shelter or hospital admission within 24 hours where needed, and Section 19(6) requires the police to report to the Child Welfare Committee and the Special Court within 24 hours.
- **Key number.** **Section 19(7)** gives complete civil and criminal immunity for information given in good faith.
- **Key number.** **Section 27(1)** allows the medical examination under the criminal procedure provision even before an information report is registered.

WHO FAILS TO REPORT	PROVISION	PENALTY
An individual, including a medical practitioner	Section 21(1)	**Imprisonment up to 6 months, or fine, or both**
The person in charge of an institution or hospital, for a subordinate's offence	Section 21(2)	**Imprisonment up to 1 year, and fine**
The child victim	Section 21(3)	Expressly exempt from prosecution

Three rulings settle the questions examiners ask.

- **Concept.** A clinician is **not required to conduct a roving investigation** or to actively gather knowledge, which requires information that appeals to the senses or a direct disclosure.
- **Concept.** **Credible information received directly from a child constitutes knowledge**, and neither a school nor a clinician may run an informal private inquiry or withhold reporting because physical signs of trauma are absent, since internal inquiries delay examination and compromise evidence.
- **Key number.** A senior specialist **cannot bypass age verification**: in one judgment an obstetrician of 35 years experience who treated a girl of 12 years and 11 months in haemorrhagic shock, on the family's claim that she was 18 years and 3 months old, failed to have the charge quashed.

Consensual adolescent pregnancy, where two statutes collide. The child protection statute **recognises no consent below 18 years**, so every sexual act with a minor is an offence, and there is no close-in-age exception of the kind other jurisdictions use.

- **Concept.** That **deters minors and guardians** from approaching registered practitioners for a lawful termination.
- **Key number.** The harmonised position from the Supreme Court in 2022 is that the practitioner **must still report under Section 19(1)**, but for the limited purpose of providing a safe termination.
- **Concept.** On the request of the minor and her guardian, the practitioner may **omit her name, identity and personal details** from that report, and is exempt from disclosing her identity in the resulting proceedings.
- **Concept.** So the **duty to report remains, and the identity is shielded**.

CLINICAL ANCHOR

Interview like a clinician, not an investigator: open-ended, neutral questions, no leading or suggestive statements, and no cross-examination for dates and sequences the child cannot supply.

Avoid repetition of interviews, which increases trauma and produces inconsistencies that defence counsel will use.

Then link: reporting triggers referral to the Child Welfare Committee within 24 hours, and a child helpline supports coordination.

Assess the non-offending caregiver's protective capacity, and remember that about 90 percent of perpetrators are people known and trusted by the child or family.

Trauma-focused cognitive behavioural therapy, eye movement desensitisation and reprocessing, play therapy and dialectical behaviour therapy are the named interventions after stabilisation.

TRAP Promising a 15-year-old that a disclosure stays between you, or conditioning the report on finding physical signs. The statutory duty overrides ordinary medical confidentiality, good-faith reporting carries immunity, and private verification has been held impermissible. Validate the distress, explain the duty transparently, and stay with the child through what follows.

HOLD THIS

For an adult woman: private enquiry, neutral words, lethality screen, verbatim documentation, and her consent before any report. For a child: no inquiry, no conditions, report to the police or the juvenile unit, and mask identity only in the termination scenario.

WHAT EARNS THE MARKS

- **Keeps an adult woman's disclosure under her own consent**: reporting her without it can provoke retaliation.
- **Runs the five-item danger assessment**: it is the part that changes the disposition.
- **Documents verbatim, without editorial words**: the record is the medico-legal deliverable.
- **Reports a child without conditions or inquiry**: private verification has been held impermissible.

SEE ALSO 11 Risk stations: suicide, agitation, and the liaison call 13 Consent and capacity as a bedside skill 20 Confidentiality, the duty to warn, and a decision model

Quick review

The whole practical in one table	
HOW STATIONS ARE MARKED	Four documents per station, observed and unobserved formats, one task. Psychiatry checklists are 79% communication items, of which a mean 89% score mastery of content. Seven Kalamazoo tasks over five Calgary-Cambridge stages. Reliability rises with station number (0.54 to 0.73 at 4 to 5 stations, 0.79-0.89 at 15); the cut-score comes from borderline regression on examiner global ratings.
BAD NEWS AND DEMENTIA	Before you tell, ask; warning shot, chunks, connecting statement, then strategy. Dementia is a triad with the care partner, syndrome disclosed before pathology. Fewer than 50% with mild impairment are told. Co-piloting is not safe practice; driving is an on-road occupational therapy assessment with review at least 6-monthly.
SCHIZOPHRENIA AND EXPRESSED EMOTION	Explanatory model first, blame removed, negative symptoms named as illness. Monozygotic concordance 50%, dizygotic 15%, adopted-away 12%, population 1%. High expressed emotion predicts early relapse (odds ratio 4.87) and warmth protects (0.35). Section 22 information, Section 23 confidentiality, Section 14 representative, Section 89 supported admission for thirty days.
ECT	Modified means anaesthesia plus suxamethonium. Adequate seizure over 15 seconds, prolonged beyond 180 seconds, up to 3 stimulations per session. Course 6 to 12 sessions twice weekly. Retrograde gap about 1 hour either side, autobiographical gaps about 10 percent, cognition often better at 2 months. Section 95 bans unmodified use and restricts minors under 18 to guardian consent plus Board permission; Section 94 bars emergency use.
CLOZAPINE	Resistance is failure of at least 2 antipsychotics, 1 atypical. Green, amber, red on white cells 3.5 and neutrophils 2.0, amber at 1.5 to 2.0, red below 1.5 with immediate cessation. Weekly for 18 weeks, fortnightly to 52, then monthly. Myocarditis in the first 28 days, peak 12 to 16 days, stop if C-reactive protein exceeds 100 mg/L. Hypomotility kills more than agranulocytosis. Break over 48 hours means restarting at 12.5 mg.
LITHIUM AND BIPOLAR PLAN	Trough at 12 hours. Acute mania 0.6 to 0.8, maintenance 0.4 to 0.6 mmol/l. Coarse tremor, vomiting and ataxia mean stop and attend. Dialysis above 2.5 mmol/l in chronic toxicity. Pregnancy: cardiac risk up to 2.25%, outflow tract 0.6%, levels every 4 weeks to the 36th week then weekly. Relapse signature of 4 to 6 items, warning window 2 to 4 weeks, review within 1 week, advance directive under Section 5.
ANTIDEPRESSANTS AND BENZODIAZEPINES	Improvement often in the first 2 weeks, review at 3 to 4 weeks, continue 6 to 9 months after a single episode and at least 2 years after 3 or more. Discontinuation in 33% to 50%, onset 1 to 2 days,

The whole practical in one table	
	resolves on rechallenge. Taper hyperbolically, 5% to 10% of the current dose. Benzodiazepine use over 1 year tapers over 6 to 18 months; 25% weekly cuts fail in 32% to 42%.
RISK STATIONS	Positively framed gateway question (negative framing yields 66% denials and abandonment in 60%). CASE covers presenting, recent 8-week window, past and immediate. Intent is not lethality. Safety plan, not a contract. Agitation: verbal first, oral before parenteral, lorazepam 1 to 2 mg first-line, no intramuscular olanzapine within 1 hour of a parenteral benzodiazepine, observations every 15 minutes for 1 hour, restraint under Section 97(2).
BEHAVIOUR CHANGE AND THERAPY	PACE, OARS, DARN-CAT, FRAMES, elicit-provide-elicit, and the five traps. Insomnia: at least 3 nights weekly for 3 months, multicomponent therapy over 4 to 8 sessions, leave bed after 15 to 20 minutes, restriction floor 5 hours, expand by 15 minutes at 85% efficiency, hygiene as adjunct only. Therapy explained as 5 to 20 sessions with homework designed jointly and confidentiality limits stated.
SKILL STATIONS	Consent: 5 disclosure elements, 2 legal standards, 8 documentation principles. Capacity: presumed, decision-specific, two stages, four abilities. Mental state: ownership and agency separate insertion from made thoughts and obsessions. Screens: 30 points, prorate omitted items, spelling scored by order, activation discount of 1 point on the movement scale, parkinsonism scale divided by 10 with a mean above 3, akathisia global 2 or more.
PROFESSIONALISM AND SAFEGUARDING	2023 regulations in abeyance from August 23, 2023; the 2002 code governs. No testimonials, no patient images, no bought visibility. Crossing versus violation turns on intent and exploitation. Confidentiality sits in Section 23 with its exceptions; the duty to protect needs an identifiable victim. Domestic violence: private enquiry, neutral words, five-item danger assessment, Sections 18 to 21 reliefs, breach punishable up to 1 year. Child abuse: report on knowledge, no private inquiry, Section 21(1) up to 6 months.

HOLD THIS

Every station is the same four moves: open and screen the agenda, gather with the cone, share in chunks with comprehension checked, and close with a negotiated plan and a named safety net. Content earns its marks only when it is spoken, checked and written into a plan the patient recognises.

PART VI

VI

Viva Tables I: Psychopathology & Investigations

The phenomenology table and the investigations table: definitions, discriminators, EEG, scans and reports.

01 Phenomenology

02 Named syndromes and culture

03 EEG

04 Neuroimaging

05 Laboratory monitoring and reports

06 Quick review

Viva Tables I: Psychopathology & Investigations

Five sub-areas · 27 topics · the two tables the examiner sits you down at

The viva tables reward a particular shape of answer: the phenomenon named in its own vocabulary, defined in one sentence, then separated from the thing it is most often confused with. **Definition, then discriminator, then the disorder it points to.**

This note is arranged as the two tables actually run. First the phenomenology table, where the examiner names a symptom and asks you to define it and tell it apart from its neighbour.

Then the investigations table, where an EEG, a scan or a laboratory report is put in front of you and the first sentence out of your mouth decides the rest of the conversation.

HOW TO USE THESE NOTES

Every topic runs in the same five beats. The **question band** is the examiner's question. **The shape of the answer** is the order to say it in, and its numbers are the order of speaking, not marks.

Then the body. Then **what earns the marks**. Then **see also**, which points to the other topics in this note that the same question opens onto.

Inside the body, a **Rule** holds across cases. A **Trap** is where marks are lost; some are traps because the sources are silent, and there the note says verify rather than handing you a figure. A **Hold this** block is the line to carry in, and the shaded cell in a grid is the discriminator the examiner is listening for.

Typed lines carry the lists: petrol for a concept or rule, marigold for a key number, coral for a trap. Numbers are carried only where a source carries them.

Phenomenology

The examiner works down a list: a disorder of perception, a disorder of thought, a disorder of possession, a motor sign, a disorder of memory or consciousness, a disorder of emotion or of the self, then insight. Each topic below is arranged so that a phenomenon turns into a definition, and a definition turns into a differential.

1 · Form, content, and what makes a symptom primary

PHENOMENOLOGY · TOPIC 01

What do you mean by the form of a symptom, and what makes a delusion primary?

PHENOMENOLOGY TABLE, OPENING QUESTION

THE SHAPE OF THE ANSWER

1	**Method**	Understanding grasps meaning, explaining establishes cause

2	**Form and content**	Form carries the syndrome, content carries the biography
3	**Primary experience**	Atmosphere, perception, idea, memory, awareness
4	**Defining a delusion**	Conviction, incorrigibility, impossibility, and the limits of those
5	**The neighbours**	Overvalued idea and obsession, separated on one grid

Answer the method question before the symptom question. Karl Jaspers published *Allgemeine Psychopathologie* in 1913 and established descriptive psychopathology as a discipline, warning against the somatic prejudice that reduces every mental phenomenon to a localised lesion.

Two methods cover conscious life.

- **Understanding (Verstehen).** The empathic grasp of meaningful connections from within, which divides into static understanding of the present state and genetic understanding of how one state emerges from another.
- **Explaining (Erklaren).** Causal mechanistic connection from without.

Depression after a severe loss is understood; delirium from anticholinergic intoxication is explained.

Form and content. The *form* is the operational structure, the how: auditory hallucination, delusional perception, overvalued idea, obsession. The *content* is the theme, the what: religious, persecutory, somatic, grandiose. Form carries the diagnostic specificity and defines the syndrome; content reflects history, culture and the technology of the day, so persecutory content shifts from sorcery to microchips while the form stays put.

- **RULE** In Jaspers, **primary means psychologically irreducible and non-understandable, not first in time.** A delusion arising after a delusional atmosphere is still primary, because its content cannot be derived from the mood.

The delusion. Classically a false, unshakeable belief out of keeping with the patient's educational, social and cultural background, its absurdity manifest to others of the same milieu (Sims).

Jaspers called it a pathologically falsified judgement with three external criteria: extraordinary conviction held with incomparable subjective certainty, incorrigibility to experience and counterargument, and impossibility or falsity of content, warning that these describe only the outside of it. Kraupl-Taylor added absolute, idiosyncratic, ego-involved, incorrigible and preoccupying.

DSM-5 dropped falsity, defining a delusion as a fixed belief not amenable to change in the light of conflicting evidence.

PRIMARY DELUSIONAL EXPERIENCE	WHAT THE PATIENT EXPERIENCES	THE DISCRIMINATOR
Delusional atmosphere (Wahnstimmung)	The environment has altered subtly and is laden with hidden, ominous, personal significance; profound perplexity and tension	**Muller-Suur's "certain uncertainty": certain that events carry meaning, uncertain what the meaning is. It ends when a delusion crystallises**
Delusional perception (Wahnwahrnehmung)	A real and normally perceived percept carries an immediate abnormal self-referential meaning	**The percept itself is intact. Schneider's two stages, normal perception then abnormal signification; Jaspers held the meaning is experienced immediately, not inferred**
Delusional idea (autochthonous delusion, Fish)	A delusion arrives suddenly, full-formed, out of the blue, like an inspiration	**No perceptual trigger, no antecedent affect, no preceding train of thought**
Delusional memory	Either a remembered event that clearly did not occur, or a genuine memory to which delusional significance attaches	**The Present State Examination form is the clearly recalled experience of a past event that did not happen**
Delusional awareness	Unshakeable intuitive knowledge of an altered reality	**No perceptual mediation at all**

Jaspers' four types of primary delusional experience, with delusional awareness as the fourth in the list he gives. Secondary delusions are the understandable ones: guilt in melancholia, grandeur in mania, persecution after persistent voices.

Delusion, overvalued idea, obsession. The concept of the overvalued idea came from Carl Wernicke, and David Veale's model is the one to quote: not a weak delusion but a belief linked to an *idealised value* that has taken overriding importance, fused with identity and held so rigidly that the consequences of acting on it are disregarded.

It is quantified on the Brown Assessment of Beliefs Scale, rating conviction, fixity, attempts to disprove and insight.

PARAMETER	DELUSION	OVERVALUED IDEA	OBSESSION
Domain	Thought content, a false belief	Thought content, an idealised value	Form and content, an intrusive thought, urge or image
Conviction	Absolute, beyond doubt	Strong and sustained, less than delusional	Recognised as senseless or excessive
Ego-syntonicity	**Ego-syntonic, experienced as personal reality**	**Ego-syntonic, identified with the self**	**Ego-dystonic, alien and unwelcome**
Inner resistance	**None; defended and acted on**	**None; the value is actively pursued**	**Typically present, provoking anxiety**
Insight	Absent	Partial or fluctuating	Preserved, except in poor-insight variants
Behaviour	Often inconsequential: intense belief, no logical action	Consequential, aligned with the idea	Neutralising or avoidance

Overvalued ideas dominate anorexia and bulimia nervosa, body dysmorphic disorder, hoarding and refractory obsessive-compulsive presentations, illness anxiety disorder, non-delusional morbid jealousy, querulant states, apotemnophilia and gender dysphoria, and pseudocyesis. A highly systematised, logically organised delusional system points to persistent delusional disorder rather than schizophrenia, where delusions are more often fragmented.

Delusional disorder, the numbers. One or more delusions for at least 1 month, criterion A for schizophrenia not met at any point, hallucinations not prominent and tied to the delusional theme, functioning relatively preserved outside the delusion's ramifications, and mood episodes brief relative to the delusional periods.

Estimated lifetime prevalence is about 0.2 per cent in population samples; mean age at presentation is about 55 years, with females about 56.5 per cent of cases; and about 65 per cent retain the diagnosis at four-year follow-up in a cohort of 71 patients, the remainder converting to schizophrenia. Persecutory content is the commonest subtype.

TRAP The truth fallacy. A delusion is not defined by being factually false. An unfaithful partner does not convert delusional jealousy into a normal belief if the conviction was reached by delusional intuition without valid evidence. Descriptive psychopathology asks about form and justification, not external verification.

ASK AND VERIFY Two standard follow-ups sit here and the library marks both unsourced: **folie a deux** as a definition with its transmission and its course on separation, and the **three stages of Cotard syndrome**. The folie a deux subtypes are given in topic 10; the Cotard staging is not printed anywhere in this note. Verify both from a textbook before the exam.

HOLD THIS

Form carries the diagnosis, content carries the biography. Primary means non-understandable, not first; and the three Jaspersian criteria are the outside of a delusion, not its nature.

WHAT EARNS THE MARKS

- **Both methods named**: understanding works from within, explaining from without
- **Primary means psychologically irreducible**: the examiner is listening for this, not for first in time
- **Ego-syntonicity as the dividing line**: it separates delusion and overvalued idea from the obsession
- **The truth fallacy refused**: factual falsity is not what makes a belief delusional

SEE ALSO 02 Hallucinations, illusions and pseudohallucinations 04 Possession, passivity and the first-rank list 08 Insight and the obsession boundary

2 · Hallucinations, illusions and pseudohallucinations

PHENOMENOLOGY · TOPIC 02

Define a hallucination, and separate it from a pseudohallucination and from an illusion.

PHENOMENOLOGY TABLE, DISORDERS OF PERCEPTION

THE SHAPE OF THE ANSWER

1 Definition	Esquirol's perception without an object, quoted first
2 Four characteristics	External space, sensory freshness, involuntary, full conviction
3 Illusions	Completion, affect and pareidolic, sorted by attention
4 The named types	Functional, reflex, extracampine, autoscopy, Charles Bonnet
5 By modality	Auditory commonest, visual prompting an organic search

Esquirol's line first, then the four Jaspersian characteristics. Esquirol in 1817 defined a hallucination as a perception without an object.

Jaspers established that hallucinations are not distortions of existing stimuli but false perceptions arising de novo alongside genuine ones, with four characteristics.

- **External space.** They appear in external objective space.
- **Sensory freshness.** They carry the substantiality and sensory freshness of a real percept.
- **Independent of the will.** They are not summoned and cannot be dismissed.
- **Full conviction.** They are accepted as concrete reality with full conviction of external existence.

Tinnitus and phantom limb are excluded, because there the subject knows the experience has no external source.

CHARACTERISTIC	NORMAL PERCEPTION	MENTAL IMAGERY	PSEUDOHALLUCINATION	TRUE HALLUCINATION
Spatial location	External objective space	Inner subjective space	**Inner subjective space, inside the head**	**External objective space**
Character	Concrete reality	Figurative, subjective	Figurative or sensory	Concrete, objective reality
Sensory elements	Full, vivid, fresh	Incomplete, poorly delineated	Vivid, relatively intact	Full, vivid, distinct
Constancy	Constant	Dissipates, must be recreated	Persistent, effortless	Constant
Voluntary control	Independent of will	**Dependent on will**	Independent of will	Independent of will
Insight	Perceived as real	Known to be self-generated	Often recognised as internal	Experienced as external reality

Jaspers' boundaries between percept, image and hallucination. Note the modern caveat: under Present State Examination conventions, voices heard inside the head with absolute conviction and distress carry the same diagnostic weight as voices from external space, so give the phenomenological distinction and then the diagnostic convention.

Illusions, after Sims. An illusion is a misperception of a real external stimulus, arising when sensory input combines with mental images or expectation, favoured by inattention, reduced cues, fatigue and heightened affect.

Three types.

- **Completion illusions.** Depending on inattention and familiarity, resolving the moment attention is directed.
- **Affect illusions.** Driven by fear, anxiety or guilt, transient and dissipating when arousal settles.
- **Pareidolic illusions.** Meaningful figures perceived in ambiguous unstructured stimuli such as clouds, embers or wallpaper.

RULE **Attention is the discriminator among illusions.** Completion and affect illusions disappear when attention is focused; pareidolia becomes more intricate and detailed. Pareidolia is a normal phenomenon in healthy adults and children.

Pareidolia can be formally tested with the Noise Pareidolia Test. In a study of 43 patients across dementia with Lewy bodies, Parkinson disease, typical Alzheimer disease and posterior cortical atrophy, pareidolic responses tracked visuoperceptual impairment rather than spontaneous hallucinations, and the posterior cortical atrophy group gave the most of them while reporting no spontaneous visual hallucinations.

PHENOMENON	DEFINITION	WORKED EXAMPLE	THE DISCRIMINATOR
Functional hallucination	A real external stimulus precipitates a hallucination in the *same* modality, both perceived simultaneously and distinctly	Derogatory voices only while the tap runs; the water and the voice are heard together and the voices cease when the tap closes	**Stimulus and hallucination coexist. In an illusion the real percept is lost and replaced**
Reflex hallucination	A real stimulus in one modality precipitates a hallucination in a *different* modality; a morbid form of synaesthesia	A vivid visual figure whenever a church bell tolls	**Cross-modal. True synaesthesia is a stable non-morbid trait in peripersonal space without persecutory distress**
Extracampine hallucination	Localised outside the boundaries of the sensory field	A figure standing behind the occiput, out of the visual field; voices from another city	**Carries no specific diagnostic indication: schizophrenia, organic states and affective disorders alike**
Autoscopy (doppelganger)	Seeing an image of oneself in external space while knowing the image is oneself (Sims)	The double appears in front of the patient, fleeting or prolonged	**Occurs in epilepsy, migraine, drug intoxication and organic psychoses**
Hypnagogic and hypnopompic	At sleep onset and on waking; intrusions of rapid eye movement physiology into wakefulness	Elementary shapes, animals, faces; bangs and footsteps; floating and falling	**Insight into unreality is usually retained, and the state is tied to the sleep-wake transition**
Complex nocturnal visual hallucinations	On sudden waking from non-rapid-eye-movement sleep, linked to neuro-ophthalmic pathology	Peduncular hallucinosis after pontine, midbrain or thalamic tegmental lesions: vivid coloured stereotyped scenes in the evening that vanish when the eyes open	**Not the benign sleep-onset phenomenon: look for the lesion or the eye disease**
Charles Bonnet syndrome	Complex visual hallucinations in older people with severe bilateral visual impairment	Macular degeneration or dense cataract, formed figures and scenes	**Insight preserved, sensorium clear. This is the sourced example of complex hallucinations with a clear sensorium**

Two epidemiological anchors. In a community survey of 49,772 individuals aged 15 to 100 years, 37 per cent reported hypnagogic and 12.5 per cent hypnopompic hallucinations.

In narcolepsy, hallucinations occur in 59 per cent of patients with cataplexy and 28 per cent without, and where treatment is indicated for severe episodes clomipramine 10 to 75 mg at bedtime is effective. Synaesthesia affects at least 1 per cent of the population, and visual hallucinations occur in about 25 per cent of patients with Parkinson disease.

By modality. Auditory hallucinations are the commonest in psychiatric disorders, from elementary noises to complex syntactical voices. Schneider named three as first-rank: thought echo (Gedankenlautwerden), running commentary, and third-person voices discussing the patient. Command hallucinations and second-person voices are separate types, belonging with the syntactical hallucinations, those carrying sentence structure. In severe affective disorder voices are mood-congruent.

Visual hallucinations are commonest in delirium, Lewy body and Parkinson disease dementia and substance states, and do occur in severe affective psychosis and schizophrenia, so they prompt an organic search rather than excluding a functional diagnosis. Somatic hallucinations are haptic, thermic, hygric or kinaesthetic, with formication in cocaine intoxication and withdrawal.

Gustatory and olfactory hallucinations are uncommon in non-organic psychosis and classical in temporal lobe epilepsy as uncinate fits.

ASK AND VERIFY Two examiner follow-ups here are unsourced in the library. The **formal definition of hallucinosis** as recurrent hallucinations in clear consciousness without the cognitive clouding of delirium is owed to a textbook, not to these sources; what is sourced is that alcoholic hallucinosis is predominantly auditory with derogatory or persecutory content, that delirium of any cause also produces auditory hallucinations, and that cerebral dysfunction can produce them with consciousness unimpaired. The second is the step after a **command hallucination** is elicited, namely asking about compliance and grading safety risk on the answer: take that from a dedicated risk reference and say so.

HOLD THIS

Functional is same-modality and simultaneous; reflex is cross-modal; extracampine is outside the sensory field and diagnostically neutral. A delusional perception is a disorder of thought, not of perception.

WHAT EARNS THE MARKS

- **Inner subjective space**: the phrase that places a pseudohallucination for the examiner
- **Attention as the discriminator**: pareidolia grows under attention, the other illusions dissolve
- **Same modality against cross-modal**: functional against reflex, settled in one sentence
- **Extracampine carries no diagnosis**: saying so protects you from over-reading it

SEE ALSO 01 Form, content, and what makes a symptom primary 04 Possession, passivity and the first-rank list 06 Consciousness, attention and memory

3 · Thought form, thought stream and the speech table

PHENOMENOLOGY · TOPIC 03

This patient's speech is hard to follow. Take me through the disorders of thought form.

PHENOMENOLOGY TABLE, THOUGHT AND SPEECH

THE SHAPE OF THE ANSWER

1 **Which domain**	Stream, form, content or possession, named before the sign
2 **The sign grid**	Definition, domain and discriminator for each term
3 **The theorists**	Bleuler, Cameron's four terms, Carl against Kurt Schneider
4 **Measurement**	Andreasen's scale, and the two factors that replicate
5 **Rule out**	Aphasia, dysarthria and the primary speech disorders

Say which domain you are in before you name the sign. Disorders of thought divide into stream (tempo and continuity), form (the logical, progressive, goal-directed organisation of thinking), content (delusions, overvalued ideas, depressive and suicidal ideation), and possession or alienation.

Formal thought disorder is failure to follow semantic and syntactic convention to a degree inconsistent with education, intelligence and culture, and is narrower than the Anglo-Saxon construct of thought disorder, which also takes in content. Thought is reached only through speech, writing and expressive behaviour, which is why speech and thought are examined as one table.

SIGN	DEFINITION	WHERE IT SITS	THE DISCRIMINATOR
Pressure of speech	Increased rate and quantity, speech without punctuation, hard to interrupt, sentences left unfinished in the rush to the next idea	Stream	**Rapid speech alone occurs in anxiety; rapid *and* pressured is cardinal in mania**
Poverty of speech (alogia)	Quantitative restriction of spontaneous output, brief concrete unelaborated replies, increased latency	Stream	**Reduced quantity. Negative schizophrenia and severe depression**
Poverty of content of speech	Adequate quantity conveying little information: vague, repetitive, stereotyped, over-abstract, empty philosophising	Form	**Adequate word count, negligible information. The interviewer notices length without an answer**
Circumstantiality	Long-winded, convoluted, excessive tedious and irrelevant detail, a circuitous route that arrives	Stream and continuity	**Reaches the goal idea. Obsessive traits, anxiety, epilepsy, cognitive impairment, some healthy people**
Tangentiality	Wanders off on tangents prompted by the patient's own words or the question; meaningful links to the immediately preceding remark	Stream and continuity	**Fails to reach the goal, drifts and does not return**

SIGN	DEFINITION	WHERE IT SITS	THE DISCRIMINATOR
Flight of ideas	Rapid switching with pressured speech; adjacent links understandable through rhyme, pun, clang or environmental distraction	Stream	**Understandable adjacent links plus marked distractibility. Mania, and other severely agitated psychotic states**
Loosening of associations, derailment	The train of thought slips into an obliquely related or unrelated channel; logical connection between consecutive ideas breaks down	Form	**Adjacent links illogical to the listener, at normal, slowed or variable tempo. Hecker described it in 1871**
Thought blocking	Abrupt interruption mid-sentence with failure to return; the patient reports the mind suddenly going blank	Stream	**Sudden arrest with subjective emptying. Depressive retardation is a generalised long latency, absence seizure has a lapse of consciousness and eyelid flutter confirmed electrographically, an aphasic pause preserves goal-directedness with circumlocution**
Incoherence, word salad (schizophasia)	Syntax and semantics collapse together, words strung into gibberish	Form, the extreme	**Breakdown at the level of the single word, not the phrase**
Illogicality	Conclusions that do not follow from the premises	Form	**Broken inferential logic, whereas a delusion keeps internal logic from an idiosyncratic premise**
Perseveration	Inappropriate continuation or recurrence of a thought, topic or verbal response after the eliciting stimulus has gone	Stream	**The repeated material is a previous answer that has outlived its stimulus. Of theme in depression; of associations, stock words plugged into unrelated topics, in coarse brain disease and schizophrenia**

SIGN	DEFINITION	WHERE IT SITS	THE DISCRIMINATOR
Verbigeration	Meaningless stereotyped repetition of the same word or short phrase, replacing understandable speech	Form and speech	**No triggering utterance and no communicative content, unlike echolalia and palilalia which repeat something just heard or just said**
Palilalia	Involuntary repetition of a word or phrase the patient has just spoken, typically terminal, with increasing speed and falling volume	Neurological counterpart of verbigeration	**Points to basal ganglia and extrapyramidal disease and chronic coarse brain disease**
Echolalia	Automatic echoing of words or phrases uttered by the examiner, often with mocking, mumbling or staccato intonation and without comprehension	Speech, an echo phenomenon	**Catatonia, autism, Gilles de la Tourette syndrome, transcortical aphasias**
Neologism	A newly coined word, or a conventional word used in a private sense inaccessible to the listener	Form	**An entirely new word or phonemic distortion. A word approximation instead uses existing vocabulary unconventionally, hand-shoe for glove, writer for pen**

Bleuler, Cameron and the two Schneiders. Bleuler in 1911 made loosening of associations the fundamental deficit and read it as the cognitive face of splitting (Spaltung), a collapse of hierarchical goal-directed intentionality rather than a mechanical failure of links.

Modern evidence refuses the specificity: loosening appears in a minority of people with schizophrenia and occurs frequently in mania (Solovay and colleagues, 1987).

Cameron, working from sorting tasks, named *overinclusion*, the failure to preserve conceptual boundaries so that irrelevant items enter a category; *asyndesis*, juxtaposition without causal or grammatical links; *metonymy*, approximate or idiosyncratic substitution; and *interpenetration of themes*, private preoccupations intruding into problem-solving. Carl Schneider classified form structurally into transitory, drivelling and desultory thinking.

Kurt Schneider was writing about possession and ego boundaries, not form.

TRAP Naming the wrong Schneider. **Carl** Schneider gave the structural subtypes of formal thought disorder; **Kurt** Schneider gave the first-rank symptoms and the disorders of thought possession. Form and possession are different domains.

Measurement. Nancy Andreasen's Scale for the Assessment of Thought, Language and Communication has 18 items including 1 global item, takes about 30 minutes, has weighted kappa interrater reliability from 0.35 to 0.80, and correlates with the Brief Psychiatric Rating Scale conceptual disorganisation item at r = 0.71.

It divides positive formal thought disorder (derailment, tangentiality, incoherence, illogicality, pressured speech, distractibility, clanging, neologisms, circumstantiality), which is commoner in affective psychoses and fluctuates with acute exacerbation, from negative formal thought disorder (poverty of speech and poverty of content), which is more closely tied to enduring schizophrenia spectrum deficits and predicts poor short-term outcome.

The Thought and Language Dysfunctions scale is a 30-item clinical rating instrument validated across 4 dimensions: positive objective, negative objective, positive subjective and negative subjective.

A systematic review of the factor structure of formal thought disorder describes three factors of which only the first two replicate consistently: a **disorganisation** factor (confused references, structural unclarities, loss of goal, derailment, tangentiality, incoherence) and a **negative or poverty** factor (poverty of speech, weakening of goal, missing information references, increased response latency, blocking).

The third factor is empirically present but its identity is unsettled, taken variously as verboseness (pressure of speech and clanging), linguistic control, incoherence or regulation of speech; the reviewers attribute the variability to methodological differences and found no specific trend. State the two replicated factors as settled and flag the third as disputed.

Proverbs. Structured interviews mask formal thought disorder, so use open conversation. Score two parameters separately: *literalness*, interpreting a metaphor concretely, and *bizarreness*, an idiosyncratic explanation the examiner cannot follow.

In a study of 94 patients, bizarre responses aggregated in schizophrenia spectrum disorders, 41 per cent against 13 per cent, p = 0.016, with specificity 86.89 per cent and sensitivity 40.85 per cent, while literal responses did not separate the groups at all, 38 against 39 per cent.

Bizarre responses correlated with self-disorder, with clinically rated formal thought disorder and with negative symptoms, but not with positive psychotic symptoms.

CLASSIC TRAP Answering that a **literal** proverb response confirms schizophrenia. It is the **bizarre** response that carries the specificity; concrete literalness is non-specific and appears at the same rate in other disorders.

The neurological bridge. Fluent nonsensical speech with neologisms occurs in both schizophasia and Wernicke aphasia, so test comprehension, repetition and awareness of deficit: Wernicke aphasia is a dominant temporoparietal lesion with severe comprehension failure, impaired repetition and anosognosia, while schizophasia preserves single-word comprehension and cognitive tracking without focal auditory agnosia.

In Kertesz's temporoparietal model, disconnecting auditory input from the semantic processor gives pure word deafness; disconnecting the auditory monitor gives semantic jargon with preserved repetition, as in transcortical sensory aphasia; the supramarginal gyrus and arcuate fasciculus give conduction aphasia with phonemic paraphasias and impaired repetition but preserved comprehension; the first two together give classical Wernicke aphasia and all three severe neologistic jargonaphasia.

Drivelling speech keeps syntax while the words make no sense, in chronic coarse brain disease and chronic schizophrenia; rambling speech is non-goal-directed and slurred, in acute intoxication.

Rule out the primary speech disorders before calling anything psychopathology.

- **Dysarthria.** A motor defect of articulation with intact linguistic formulation.
- **Dysphonia.** Impaired voicing from vocal cord or palatal weakness or spasticity.
- **Scanning speech.** Syllables stretched in a slow sliding cadence, typical of cerebellar lesions in multiple sclerosis.
- **Huntington chorea.** Mumbled, hesitant, dysrhythmic speech.
- **Staccato speech.** Psychomotor epilepsy.
- **Stuttering and cluttering.** Cluttering is dysrhythmic rapid jerky speech in erratic bursts.
- **Coprolalia.** Gilles de la Tourette syndrome.

Logorrhoea is cited alongside pressure of speech as a positive formal thought disorder denoting a larger amount of produced speech; a fuller bedside gloss is not sourced, so define it that way and stop.

HOLD THIS

Circumstantiality arrives, tangentiality does not; poverty of speech is quantity, poverty of content is information. Flight of ideas keeps understandable adjacent links, derailment loses them.

WHAT EARNS THE MARKS

- **Circumstantiality arrives**: the one-word test that separates it from tangentiality
- **Quantity against information**: poverty of speech against poverty of content of speech
- **Carl, not Kurt**: the structural subtypes of form belong to Carl Schneider
- **Bizarre, not literal**: bizarreness carries the specificity on proverb testing

SEE ALSO 01 Form, content, and what makes a symptom primary 04 Possession, passivity and the first-rank list 06 Consciousness, attention and memory

4 · Possession, passivity and the first-rank list

PHENOMENOLOGY · TOPIC 04

What is thought insertion, and how does it differ from an obsessional thought?

PHENOMENOLOGY TABLE, POSSESSION AND PASSIVITY

THE SHAPE OF THE ANSWER

1	**Two senses**	Ownership and agency, defined before any example
2	**Elicitation**	The non-leading questions for insertion, withdrawal and broadcast
3	**The phenomena**	Insertion, withdrawal, broadcast, made affect, impulse, act
4	**The list**	Eleven first-rank symptoms, seven of them self-disorders
5	**The frames**	Mechanism in three lines, then Bleuler and Crow

Ownership and agency are two different things, and the examiner wants both words. Mental activity normally carries a *sense of ownership*, the pre-reflective awareness that these thoughts or sensations occur within my own psychological and bodily space, and a *sense of agency*, the awareness that I initiate and author them.

In disorders of possession and passivity the boundary between self and non-self collapses: the patient remains conscious of the thought, sensation or movement but disowns its authorship or reception, attributing it to an external alien influence.

FEATURE	THOUGHT INSERTION	OBSESSIONAL THOUGHT	AUDITORY HALLUCINATION
Core nature	Delusional disorder of possession	Disorder of possession, involuntary recurrence	Sensory perceptual disorder
Source attribution	**External agency, alien origin**	**Internal mind, self-generated**	**External or internal space, heard as sound**
Sensory quality	Absent, pure thought	Absent, pure thought	Present, acoustic characteristics
Resistance and insight	Accepted with delusional conviction	Often resisted, insight typically preserved	Variable insight, perceived as true sound

Eliciting them without leading. Three sets of questions.

- **For insertion.** Do the thoughts in your mind feel like your own, or does it feel as though someone puts them there; has it felt as though thoughts are being sent into your head from outside; how can you tell these particular thoughts are not yours.
- **For withdrawal.** Do you find that while you are thinking, your thoughts are suddenly taken out of your head; when your mind suddenly goes quiet, what explanation do you have for it.
- **For broadcast.** Do you feel your thoughts escape from your head so that people around you know what you are thinking; do your thoughts spread out like a broadcast; have you tried anything, such as a special cap or covering, to stop thoughts escaping.

PHENOMENON	DEFINITION	THE DISCRIMINATOR
Thought insertion	Thoughts not the patient's own are placed into the mind by an outside agency, and are distinguished from native thoughts	**Loss of ownership. In obsession the patient says these are my thoughts but I cannot stop them**
Thought withdrawal	Thoughts are actively extracted or stolen out of the mind by an external agency, leaving it empty	**A delusional explanation is constructed. Simple thought block is a sudden blankness with no external agent invoked**
Thought broadcast	Thoughts leave the mental space and diffuse into the world, becoming accessible to others without speech. Variants: passive leakage, thinking in unison, telepathic knowing	**The vector runs outward. In a delusion of reference the world directs messages inward at the patient**
Made affect	Feelings such as sudden unprovoked weeping, anger or mirth are imposed into consciousness by an external controller	**The emotion is attributed to an outside agency. Incongruous affect is a mismatch with no such attribution**
Made impulse	An urge or drive to act is experienced as placed in the patient rather than arising from his own desire	**The drive itself is disowned, before any action**
Made volitional act	Limbs, body or voice carry out complex acts the patient experiences as operated from outside, like an automaton	**Motor execution preserved, authorship alienated. In abulia the patient acknowledges the wish but cannot self-initiate**
Somatic passivity	Bodily sensations imposed by an external force on a passive body: three features, passive reception of the sensation, external deliberate attribution, and loss of ego boundary	**Sensation *plus* deliberate external infliction. A somatic hallucination is a false bodily perception with no such requirement; depersonalisation keeps reality testing and the "as if"**

Typical verbatim reports for somatic passivity: my intestines are being pulled by someone from outside; there is a current being passed through my head that makes my thoughts heavy; they make me feel aroused against my will. Radio waves sent by the police are burning my liver is the worked example; I feel a stone rolling inside my bladder is a somatic hallucination.

The first-rank list. Schneider set out his criteria in *Clinical Psychopathology* to give operational guidance without inferred intrapsychic mechanisms. In the ICD-10 classification 11 symptoms are enumerated: audible thoughts, voices arguing or discussing, voices commenting on one's actions, somatic passivity, thought withdrawal, thought insertion, thought broadcasting, made feelings, made impulses, made volitional acts, and delusional perception.

The structural point worth a mark is that 7 of the 11 are ego-boundary disturbances, the self-disorders, which the German tradition keeps strictly separate from delusions: a self-disorder is an inner experience to which the patient has privileged access, a delusion concerns external states of affairs to which clinician and patient have equal access.

Ratings of bizarre delusions have low inter-rater reliability while passivity and self-disorders retain higher consistency, and Schneider raised delusional perception to first rank because the initial perception can be checked by the clinician.

If asked why the criteria were so restrictive: Schneider wrote in Germany at a time when the diagnosis exposed patients to forced sterilisation and state killing, and more than 140,000 psychiatric patients were killed.

DSM-IV accepted a single first-rank symptom for criterion A; DSM-5 removed the privilege and requires at least 2 criterion A symptoms, because reviews found poor sensitivity and limited specificity, with these symptoms occurring in affective and other non-schizophrenic psychoses.

ICD-11 restores no privilege, though at the developmental stage reviewed self-disorders were named among the mandatory symptoms and distinguished there from delusions.

RULE First-rank symptoms are not pathognomonic, and their bedside power now runs the other way: passivity and complex self-disorders are **rare in psychosis caused by medical or neurological disease**, so a newly presenting acute psychosis with none of them is a prompt to look for a somatic, toxic or neurological cause.

Mechanism, in three lines. A comparator deficit: an efference copy predicts the sensory consequences of a command or thought, and when prediction and feedback match the feedback is attenuated, which is what agency feels like, so abnormal prediction-error signalling makes self-generated processes feel externally imposed.

Aberrant salience: dysregulated dopaminergic transmission tags internal or irrelevant cues with exaggerated importance and the delusion is the explanation built for it. Body representation: abnormal activity and connectivity in parietal cortex, which attributes agency, and insula, which mediates interoception.

Bleuler and Crow, because they follow immediately. Bleuler used two axes. Fundamental symptoms must be present in any manifestation: loosening of associations, affective blunting or incongruity, autism, ambivalence. Accessory symptoms come and go: delusions, hallucinations, catatonic postures, mutism, somatic complaints.

Separately, primary symptoms follow directly from the assumed somatic disease process and secondary symptoms are psychological elaborations of the intact mind. Associative loosening is both fundamental and primary; delusions and hallucinations are accessory and secondary. The Four As, taught as his doctrine, is a later English-language mnemonic (McNally, 2009) that obscured Spaltung.

PARAMETER	CROW TYPE I	CROW TYPE II
Predominant symptoms	Positive: delusions, hallucinations, positive thought disorder	Negative: flattened affect, alogia, avolition
Onset	Acute or episodic	Insidious, gradual, chronic
Premorbid adjustment	Relatively intact	Frequently poor
Underlying pathology	**Neurochemical: hyperdopaminergic state with increased D2 receptors**	**Structural: tissue loss and cerebral ventricular enlargement**
Response to typical neuroleptics	Favourable	Poor or limited
Cognitive impairment	Largely absent or mild	Frequently present and persistent
Outcome	Potentially reversible, better prognosis	Enduring deficit state

Andreasen and Olsen added 3 subtypes, positive, negative and mixed, with negative schizophrenia defined across 5 domains: affective flattening, alogia, avolition, anhedonia and attentional impairment, and correlating with poor premorbid adjustment, impaired cognition and a raised ventricular-brain ratio.

The binary then gave way: analyses of 114 patients yielded 3 dimensions, reality distortion, disorganisation and psychomotor poverty, and factor analyses of the Positive and Negative Syndrome Scale identify 5, positive, negative, disorganised, excited and anxiety or depression. Andreasen's unifying proposal was cognitive dysmetria.

The lineage runs back to Reynolds in 1858 and to Hughlings Jackson in 1889, who held that negative symptoms follow dissolution of higher centres while positive symptoms are release phenomena from undamaged lower structures.

ASK AND VERIFY The paired follow-up to the first-rank list is **Schneider's second-rank symptoms** and their relative diagnostic weight. The library marks this unsourced and prints no list, so no enumeration appears here. Confirm it from a textbook of descriptive psychopathology before the exam.

HOLD THIS

Eleven first-rank symptoms, seven of them ego-boundary disorders. Fundamental is about diagnostic necessity, primary is about biological origin, and Bleuler put delusions and hallucinations in neither.

WHAT EARNS THE MARKS

- **Loss of ownership**: insertion disowns the thought, the obsession keeps it
- **Seven of the eleven are ego-boundary disorders**: the structural point that earns the extra mark
- **Not pathognomonic**: absent passivity in a new psychosis sends you hunting a cause
- **Sensation plus deliberate infliction**: the two-part test for somatic passivity

SEE ALSO 01 Form, content, and what makes a symptom primary 03 Thought form, thought stream and the speech table 07 Emotion and the disorders of the self 08 Insight and the obsession boundary

5 · Catatonia, motor phenomena and volition

PHENOMENOLOGY · TOPIC 05

Which signs would you examine for catatonia, and what are the thresholds?

SHORT CASE EXAMINATION, AND THE PHENOMENOLOGY TABLE

THE SHAPE OF THE ANSWER

1 **History**	Kahlbaum through to the current classifications, in one breath
2 **Thresholds**	Three signs of twelve, two of fourteen on screening
3 **The signs**	How each is elicited, catalepsy against posturing
4 **The challenge**	Lorazepam, then a fifty per cent fall in score
5 **Danger**	Malignant catatonia, and why antipsychotics are withheld

Give the history in one breath, then the thresholds. Kahlbaum described catatonia in 1874 as an independent cyclic disease entity of motor, behavioural and vocal abnormality, from 26 cases; Kraepelin folded it into dementia praecox in 1893, Bleuler kept it as a subtype of schizophrenia, and in 1976 Gelenberg drew attention back to its frequency in medical and neurological disease.

Contemporary nosology has decoupled it: it is identified in 0.5 per cent to 2.1 per cent of general psychiatric patients, about 10 per cent of inpatients, and 12 per cent to 17 per cent of young people with autism spectrum disorder.

It is commonest in mood disorders at 43 per cent of cases, then schizophrenia at about 33 per cent, then medical and neurological disorders in up to 25 per cent.

INSTRUMENT	ITEM THRESHOLD	TOTAL CRITERIA	THE DISCRIMINATOR
DSM-5	3 or more signs	12 signs	**Not attributable to delirium; specified under a primary psychiatric or a medical condition**
ICD-11	At least 3 signs	15 signs	**Requires co-occurrence of decreased, increased or abnormal psychomotor activity, and names staring, ambitendency, rigidity, verbigeration and combativeness**
Bush-Francis Catatonia Rating Scale	At least 2 signs	14 screening items of 23 total scale items	**Screening uses the first 14 items; research criteria add a duration of at least 1 hour**

The 12 DSM-5 signs: stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerisms, stereotypies, agitation not influenced by external stimuli, grimacing, echolalia, echopraxia.

SIGN	DEFINITION AND HOW IT IS ELICITED	THE DISCRIMINATOR
Catatonic stupor	Extreme hypoactivity, immobility and verbal or motor unresponsiveness to the environment, with sleep-wake cycles intact	**The patient is awake: blink and pupillary responses and active resistance to passive eyelid elevation are preserved, unlike true coma. Depressive retardation has slowing without catalepsy or waxy flexibility**
Mutism	Verbal unresponsiveness without aphasia or mechanical speech impairment; whispered, slowed or robotic speech in milder states	**Can occur in an ambulatory, posturing patient, so it is scored separately from stupor**
Negativism	Apparently motiveless resistance to instruction or to being moved. Passive: instructions are not executed. Active: the opposite is performed, eyes squeezed shut when asked to open them	**Motiveless and directed against the request itself**
Gegenhalten	Involuntary automatic resistance to passive movement that rises in proportion to the force the examiner applies	**Proportional and automatic, not voluntary. Its polar opposite is mitgehen**
Mitgehen	The limb moves in the direction of light fingertip pressure despite instruction to resist: the anglepoise lamp sign	**Pathological compliance to *light touch*. Automatic obedience is exaggerated compliance with verbal requests and literal repetition of actions requested once**
Catalepsy	Passive induction and maintenance of awkward anti-gravity postures placed by the examiner	**The examiner places the limb. Posturing is assumed spontaneously and maintained actively**
Waxy flexibility	On repositioning a limb, slight uniform initial resistance that releases smoothly like bending a warm candle, the limb then holding the new position	**A tactile sign. Rigidity is uniform resistance through the range without posturing, lacking the velocity-dependent clasp-knife catch of spasticity and the cogwheeling of Parkinson disease**
Echo phenomena	Echolalia, automatic non-purposeful repetition of the examiner's words; echopraxia, automatic imitation of the examiner's movements	**No conversational or imitative intent: a motor release phenomenon**

SIGN	DEFINITION AND HOW IT IS ELICITED	THE DISCRIMINATOR
Mannerism	Odd, stylised caricature of a normal motor act: tiptoe walking, hopping while walking, an exaggerated salute	**Purposeful in origin, abnormal in execution, variable and episodic**
Stereotypy	Repetitive, rhythmic, monotonous, non-goal-directed movement: finger-tapping, rocking, body patting	**Non-purposeful and rhythmic. Verbigeration is its vocal equivalent**
Ambitendency	The motor face of Bleulerian ambivalence: the patient is caught between opposing impulses. Hold out a hand while instructing the patient not to shake it; the hand extends halfway, pauses, withdraws, extends again	**Oscillating hesitancy, neither compliance nor refusal**
Tic	Rapid, sudden, recurrent, non-rhythmic stereotyped movement or vocalisation, suppressible briefly with effort	**A somatosensory premonitory urge, relief on execution, and rebound after suppression. Stereotypies and mannerisms have no premonitory urge**

The lorazepam challenge. Score the Bush-Francis scale, give lorazepam 1 to 2 mg intravenously, intramuscularly or orally (alternatives are diazepam 5 mg intravenously or zolpidem 10 mg orally), and re-examine at 10 minutes for the intravenous route, 15 minutes for intramuscular and 30 minutes for oral.

A positive challenge is a rapid reduction of at least 50 per cent in catatonic signs on a standardised scale.

Overall lorazepam response runs between 67 per cent and 86 per cent, reported as 60 per cent to 90 per cent in general reviews; catatonia arising from mood disorders responds at 92 per cent to 97 per cent against 82 per cent for medical causes and 59 per cent in schizophrenia.

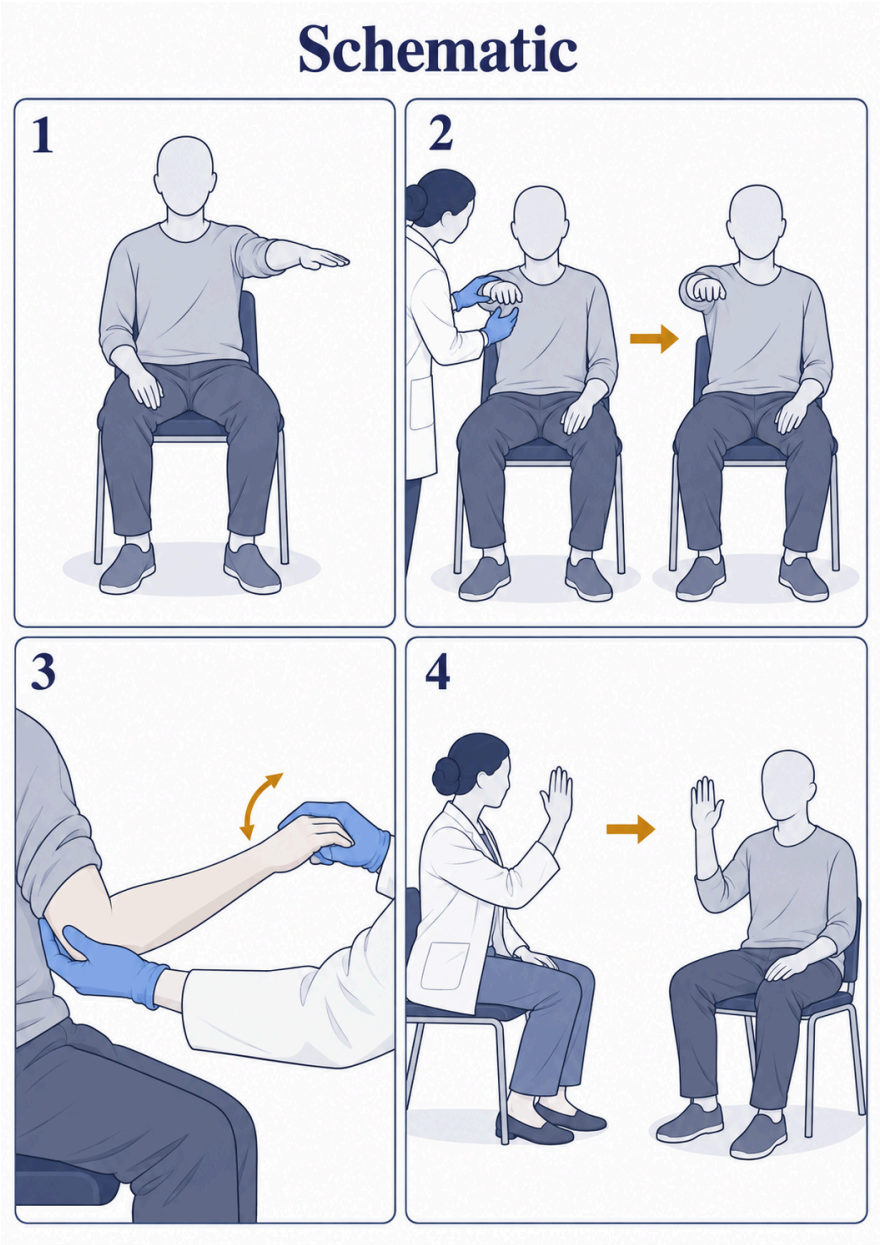
Titrate in daily increments to 8 to 24 mg per day, with selected refractory reports using up to 28 mg per day, and most responders remit within 3 to 7 days.

RULE Antipsychotics are **contraindicated in acute catatonia**, first-generation agents most of all, because they can worsen immobility and precipitate malignant catatonia or neuroleptic malignant syndrome. If residual psychosis needs treatment after catatonia resolves, prefer a second-generation agent with low D2 binding (quetiapine, clozapine) or a partial agonist (aripiprazole).

Malignant catatonia. Profound motor signs, severe stupor or non-purposeful excitement, with dysautonomia: hyperthermia, tachycardia, tachypnoea, labile blood pressure and diaphoresis. Untreated lethality is 10 per cent to 20 per cent.

Catatonia: who initiates the movement?

Companion to original page 368



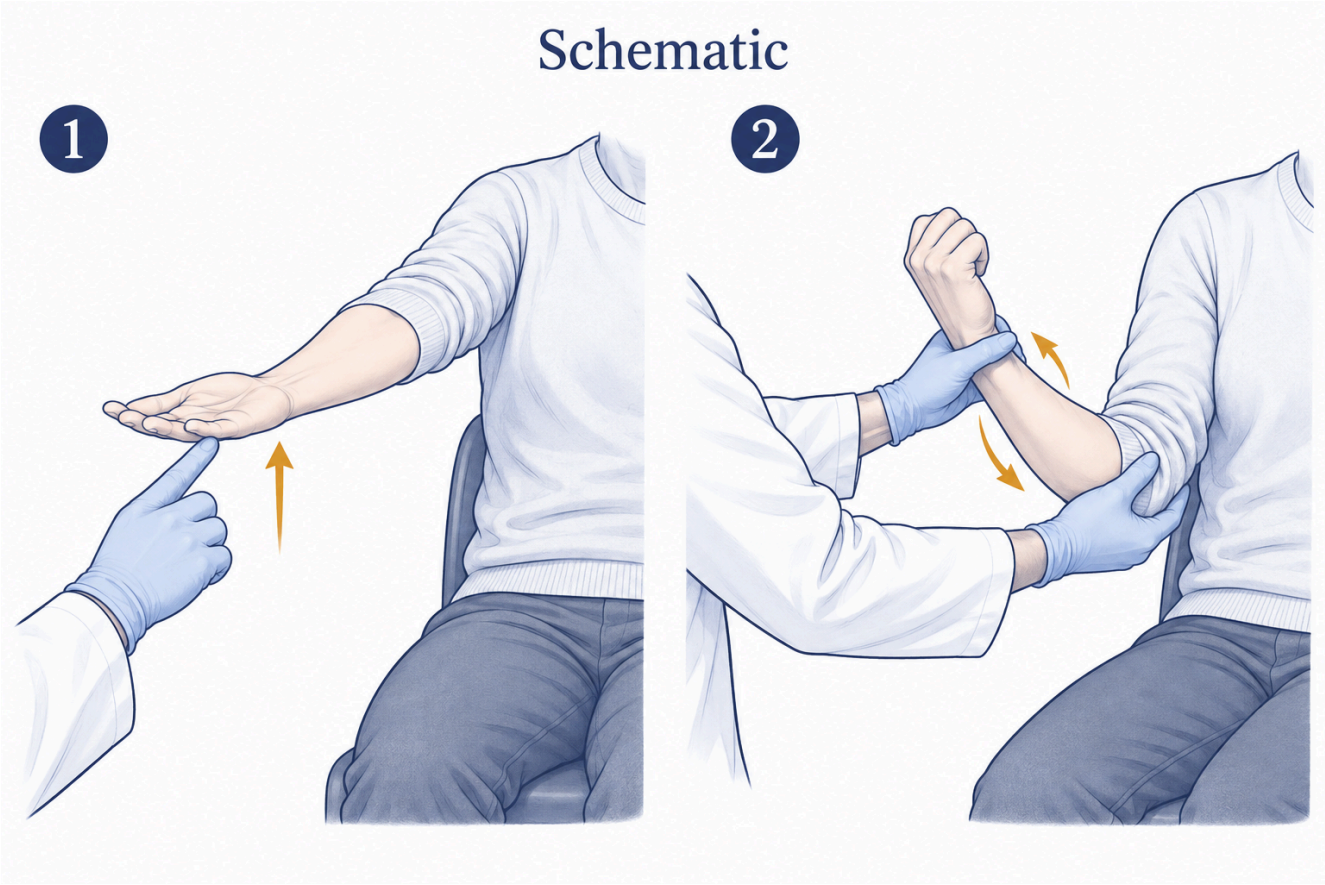
Schematic. 1: self-initiated posturing. 2: an examiner positions a limb and it remains held (catalepsy). 3: gentle passive repositioning to assess waxy flexibility. 4: imitation of an examiner gesture (echopraxia). Posturing is maintained spontaneously; catalepsy follows passive placement. Waxy flexibility is a resistance felt during examination, and echopraxia requires interpretation of imitation in context. The still cannot prove duration, resistance or involuntariness.

Distinguish spontaneous posture, maintained imposed posture, tactile resistance and imitation.

Schematic prepared by Luna for Weave Viva, based on P5A-08. AI-generated educational illustration.

Mitgehen and gegenhalten: different responses to touch

Companion to original page 368



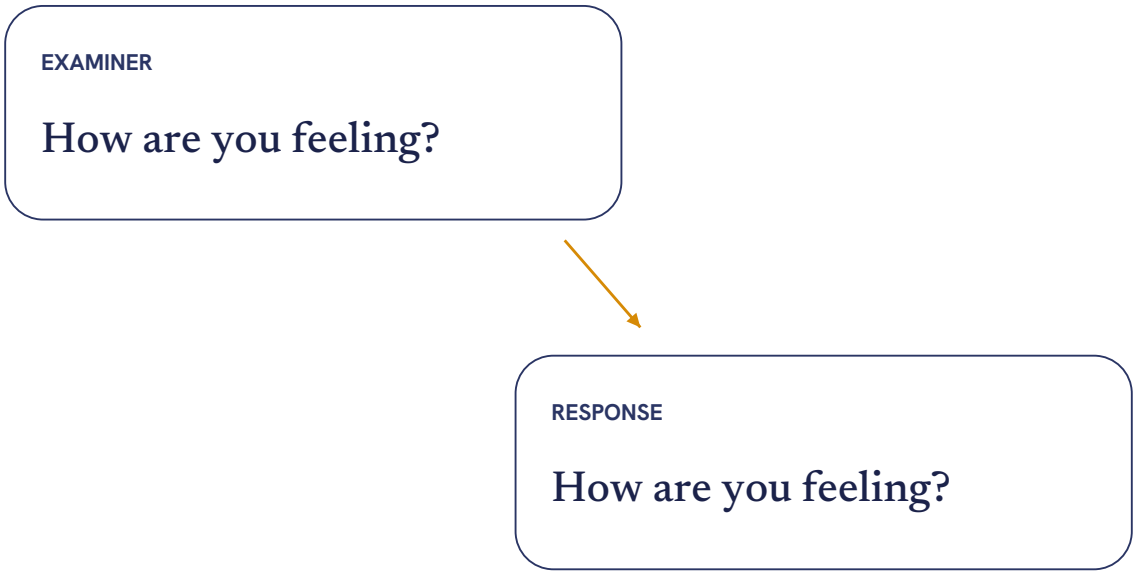
Schematic. Panel 1: mitgehen is illustrated by arm elevation with light fingertip contact beneath the palm, despite an instruction not to follow the movement. Panel 2: the examiner supports the elbow and wrist during passive movement; gegenhalten is resistance that increases with the applied force. The arrows indicate concepts, not measurements. Neither response can be diagnosed from a still image; distinguish these findings from sustained imposed posture and from behavioural opposition.

Compare the contact point, the instruction and the response before naming the sign.

Schematic prepared by Luna for Weave Viva. AI-generated anatomical illustration. Based on P5A-08; teaching references: Rogers et al., BAP catatonia guideline (2023), and University of Rochester BFCRS training manual. No source figure or scale reproduced.

Listen to what is repeated, and when.

An invented exchange to demonstrate echoing of speech.



The words return; the intended answer does not.

Schematic ▪ Original teaching diagram ▪ September 2026

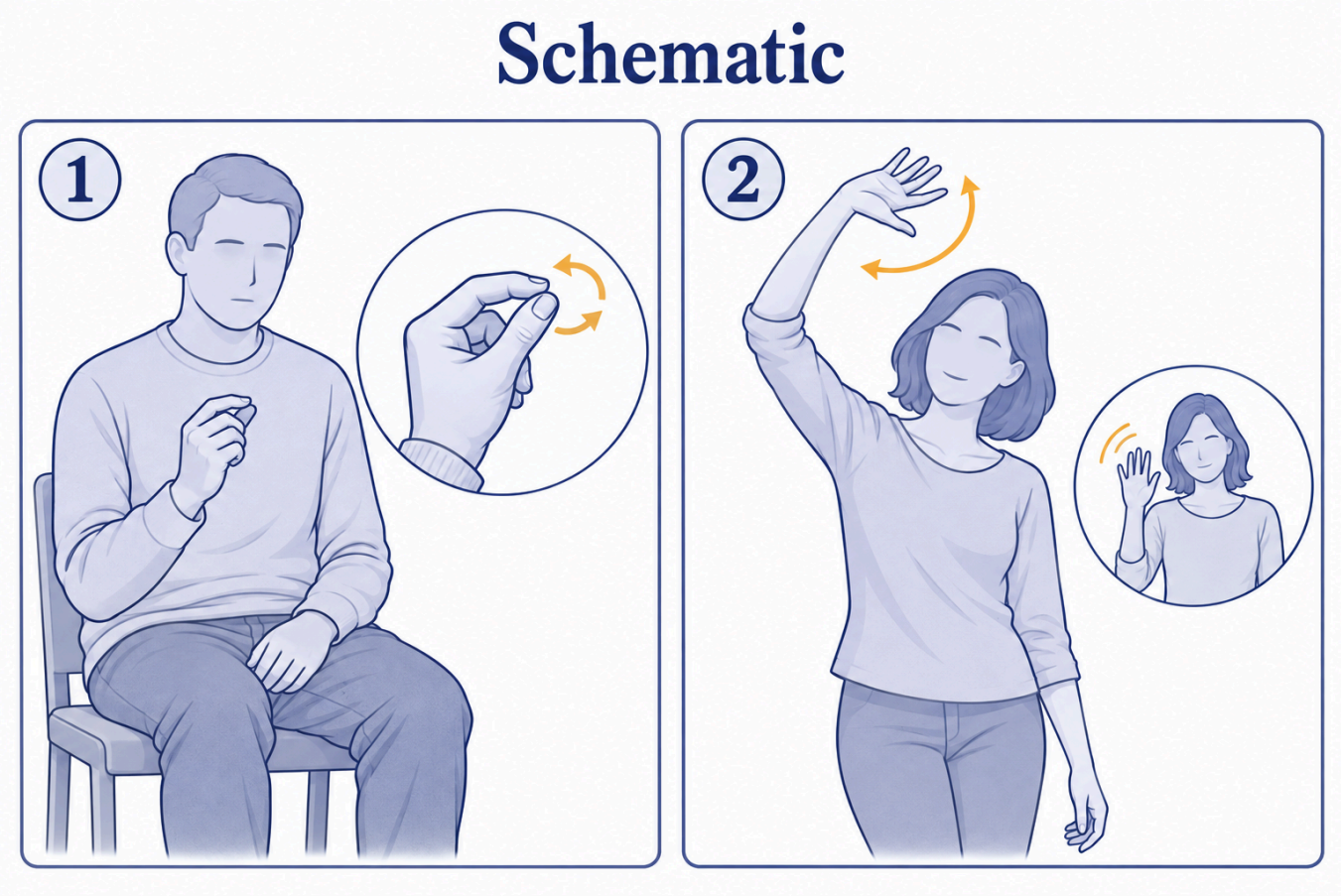
Schematic. An invented exchange illustrates repetition of another person's words rather than a direct answer. Echolalia must be interpreted in context: repetition may be requested, developmentally expected or associated with several conditions. This diagram is not a transcript, patient record or diagnostic test.

Distinguish echoing from answering, requested repetition and spontaneous repetitive speech.

Schematic prepared by Luna for Weave Viva, based on P5A-08. Original vector diagram.

Stereotypy and mannerism: pattern and context

Companion to original page 368



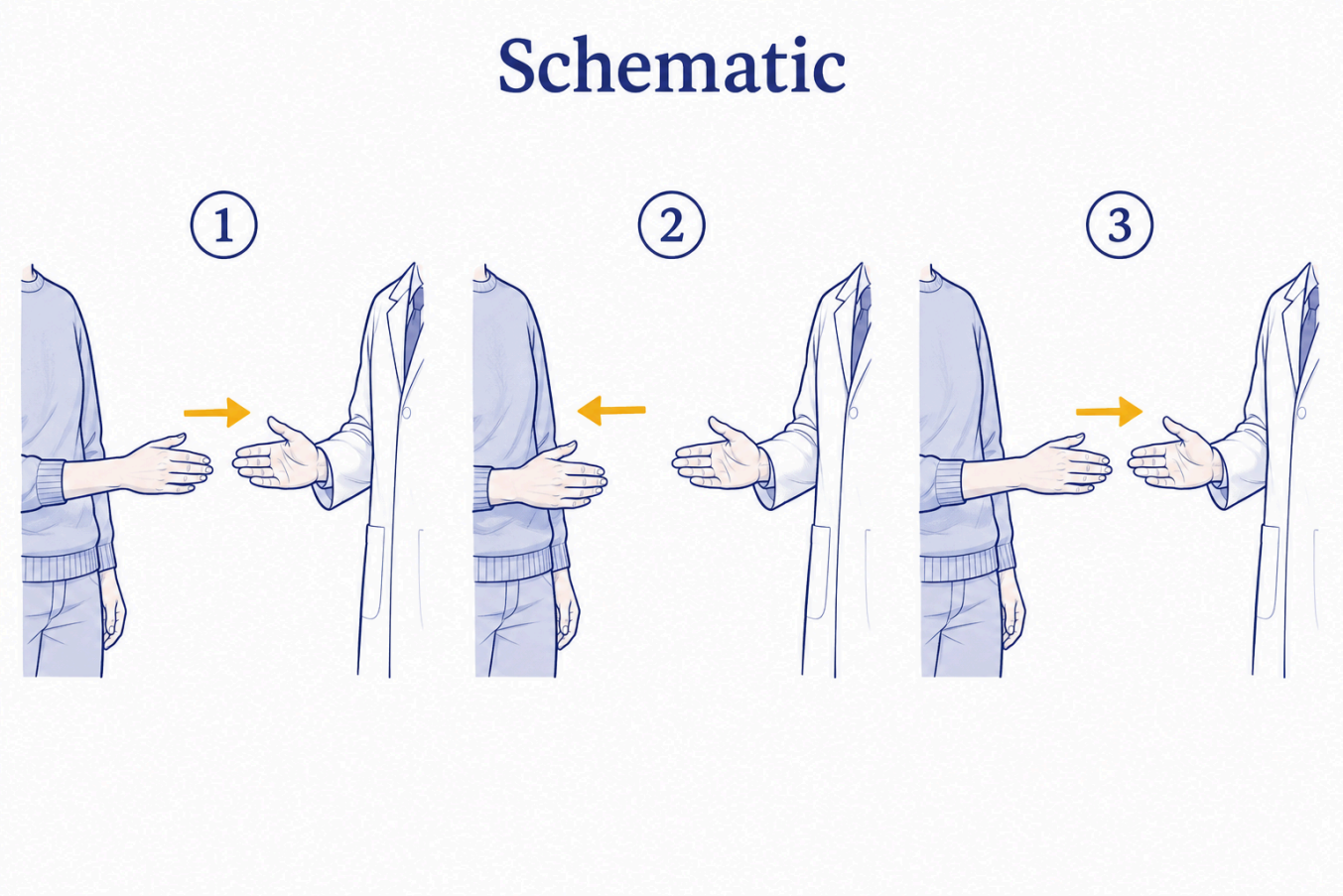
Schematic. Left: repeated finger rubbing illustrates a repetitive movement pattern. Right: an elaborate greeting is contrasted with an ordinary wave inset to illustrate an exaggerated or unusual form of a purposeful action. These are conceptual examples, not patient observations. Repetition or an unusual gesture alone does not establish catatonia; context, baseline, voluntariness and the complete examination matter.

Describe the movement before applying a phenomenological label.

Schematic prepared by Luna for Weave Viva, based on P5A-08. AI-generated educational illustration.

Ambitendency: approaching and withdrawing

Companion to original page 368



Schematic. A sequence of reaching towards an offered hand, withdrawing and approaching again illustrates motor indecision. It does not demonstrate a real patient's behaviour, and an isolated hesitation may reflect ordinary uncertainty, anxiety or misunderstanding. Assess the observed behaviour in context rather than diagnosing catatonia from a still sequence.

Distinguish observed motor indecision from an inferred feeling of ambivalence.

Schematic prepared by Luna for Weave Viva, based on P5A-08. AI-generated educational illustration.

Risks are dehydration, rhabdomyolysis, renal failure, deep vein thrombosis and fatal pulmonary embolism, so management is intensive or high-dependency care, aggressive intravenous rehydration, active temperature control, chemical thromboprophylaxis and immediate withdrawal of antidopaminergic agents.

Modified electroconvulsive therapy is the definitive treatment, with efficacy reported from 80 per cent upwards, and daily bitemporal treatment may be used initially in severe or febrile presentations. Modern delivery is under full anaesthesia and neuromuscular blockade with continuous oxygenation and cardiorespiratory and electroencephalographic monitoring, so the Indian practice standard is modified, not direct, treatment.

TRAP, AND WHAT THE SOURCES WILL NOT SETTLE

Malignant catatonia against **neuroleptic malignant syndrome**. Both give rigidity, hyperthermia and autonomic instability, so anchor on the drug history: a recent antipsychotic start, a dose increase or a high-potency first-generation agent shifts the balance toward the syndrome. Say plainly that the boundary is contested, with several authors treating the syndrome and toxic serotonin syndrome as medication-induced forms of catatonia. Creatine kinase belongs in the workup, but no cut-off, trend or onset-tempo criterion separating the two is sourced. The practical consequence: stop antidopaminergic agents, resuscitate, and treat along the shared pathway rather than waiting on the label. Separately, the statutory section governing modified electroconvulsive therapy in India and the restriction on its use in minors are unsourced in this chapter: carry them from the Act itself.

Volition, and the differential of not moving. Abulia is an abnormal lack of activity, or of the power to execute activity, despite the desire to do so, from the Greek for absence of will.

A Delphi consensus of British neurologists and psychiatrists confirmed it as a distinct entity: difficulty initiating and sustaining spontaneous movement, reduced spontaneous speech with long response latency, blunted emotional responsiveness, and loss of social initiative. It sits between apathy, abulia minor, and akinetic mutism, abulia major.

The circuit is the dopamine-dependent fronto-subcortical centro-medial core from medial prefrontal cortex to upper brainstem, with the anterior cingulate driving effort, the caudate as the striatal link, and the anterior and paramedian thalamus and capsular genu as disconnection points.

Causes fall into four groups.

- **Stroke.** Anterior cerebral, caudate, capsular genu and paramedian thalamic strokes.
- **Trauma.** Orbitofrontal and cingulate trauma.
- **Toxic and metabolic.** Hypoxia and carbon monoxide poisoning with pallidal necrosis, disulfiram neurotoxicity, and hepatic encephalopathy.

- **Degenerative.** The neurodegenerative disorders.

STATION: THE PATIENT WHO WILL NOT MOVE OR SPEAK

Synthetic case. A man in his forties is brought in immobile and mute after several weeks of low mood. The ladder the examiner walks you up:

1. Is he awake? Check eye opening, blink and pupillary responses, and resistance to passive eyelid elevation.
2. Score the catatonia scale: stupor, mutism, negativism, posturing, catalepsy, waxy flexibility, echo phenomena, ambitendency.
3. Test mitgehen with light fingertip pressure and gegenhalten with graded passive movement, and say which you found.
4. Is he febrile, tachycardic, diaphoretic or labile? If so, this is a medical emergency before it is a diagnosis.
5. What has he been given? Any antidopaminergic agent recently started or increased?
6. Address him indirectly, and if he is inert face to face, test the telephone effect, which points to abulia minor rather than catatonia.
7. Give the challenge dose and re-examine at the interval for the route you used, then say what a fifty per cent fall would mean.
8. What is the plan if he does not respond, and what do you stop before you start it?

Miller Fisher's telephone effect is the sign worth naming: in abulia minor the patient appears mute, unresponsive and inert face to face, yet converses fluently and promptly over a telephone, because auditory telephone stimulation acts as an external trigger that bypasses the disrupted internal initiation mechanism.

Its near neighbour is *pure psychic akinesia* or loss of psychic self-activation, described by Laplane after bilateral basal ganglia lesions such as globus pallidus damage from carbon monoxide or disulfiram poisoning, or bithalamic infarcts: a reversible mental void with no spontaneous thoughts, desires or actions, in which cognition and motor execution are remarkably preserved once the examiner stimulates or questions the patient.

Abulia keeps the internal desire; pure psychic akinesia loses the spontaneous ideation itself.

Catatonia examination: how each finding is obtained

Companion to original page 370

Schematic. Begin with unprompted observation, then assess interaction and responses to demonstrations or requests. Examine posture and tone gently, and integrate the findings with history and the physical assessment. The sequence separates observed behaviour from elicited responses; it is a teaching aid, not a diagnostic scale, score sheet or complete clinical protocol. An isolated illustrated sign does not establish catatonia.

Describe what you observed and how you elicited it; do not infer intention from non-response.

Schematic prepared by Luna for Weave Viva. Original vector teaching diagram. Based on P5A-08; teaching references: Rogers et al., BAP catatonia guideline (2023), and University of Rochester BFCRS training manual. No source figure or scale reproduced.

PHENOMENON	VOLITIONAL STATUS	PREMONITORY URGE	RESISTANCE	THE DISCRIMINATOR
Compulsive act	Ego-dystonic, felt as an internal compulsion against preference	Internal tension or obsessive dread	Subjectively present, with rising distress	**Preceded by anxiety and resisted; performed to avert harm**
Impulse phenomenon	Ego-syntonic at execution	Immediate drive or appetitive arousal	Absent or fleeting; regret comes afterwards	**Driven by temptation and anticipated relief, with remorse only after the act**
Mannerism	Ego-syntonic, without distress	Absent	Absent	**Purposeful in intent, unnatural in form**
Stereotypy	Involuntary or automatic	Absent	Absent	**A uniform non-purposeful motor loop**
Tic	Semi-voluntary, briefly suppressible	Characteristic somatosensory urge	Temporary, with rebound	**The urge and the relief are the diagnosis**

ASK AND VERIFY Two discriminators examiners reliably ask in this territory are unsourced in the library and are therefore not asserted here: **akinetic mutism against locked-in syndrome**, and **abulia against avolition** as a negative symptom of schizophrenia. Prepare both from a textbook. The distinction that *is* sourced is abulia against depression: the depressed patient has dysphoria, guilt and worthlessness, while the abulic patient has a pure deficit of initiation and blunted emotion without psychic anguish.

HOLD THIS

Catalepsy is placed by the examiner, posturing is assumed by the patient; mitgehen yields to light touch, gegenhalten resists in proportion. Three signs on the scale opens the diagnosis, and a fifty per cent fall on challenge confirms it.

WHAT EARNS THE MARKS

- **Placed against assumed**: catalepsy is placed by you, posturing is adopted by the patient
- **Mitgehen against gegenhalten**: light touch yields, proportional resistance opposes
- **Withholding the antipsychotic**: the safety sentence the examiner is waiting for
- **A fifty per cent fall confirms**: the challenge is diagnostic as well as therapeutic

SEE ALSO 04 Possession, passivity and the first-rank list 06 Consciousness, attention and memory 23 Reading the report they hand you

6 · Consciousness, attention and memory

PHENOMENOLOGY · TOPIC 06

Define consciousness, then tell me how you separate organic from dissociative amnesia.

PHENOMENOLOGY TABLE, COGNITION AND CONSCIOUSNESS

THE SHAPE OF THE ANSWER

1	**Two components**	Arousal from the brainstem, content from the cortex
2	**The quantitative ladder**	Clouding, drowsiness, stupor, coma, and stupor's two senses
3	**Attention**	Vigilance, concentration and distractibility, defined apart
4	**Memory**	Three phases, two kinds of failure, Ribot's law
5	**Syndromes**	Confabulation, Korsakoff, Ganser, and the qualitative states

Consciousness has two components, and naming them earns the first mark. Arousal or vigilance is wakefulness and responsiveness, driven by the ascending reticular activating system in upper brainstem and paramedian diencephalon, so bilateral lesions there depress arousal into obtundation, stupor or coma.

Content is cognitive processing, self-awareness, perception and memory, depending on widespread cortex with limbic and mesial frontal networks dominant, so toxic-metabolic or diffuse cortical pathology degrades content into confusion. A sudden extensive depression across both hemispheres can secondarily reduce arousal too.

STATE	DEFINITION	THE DISCRIMINATOR
Clouding of consciousness	Minimal to moderate reduction in clarity of awareness with impaired attention, slowed thinking and reduced perception of the environment	**The mildest end of the continuum, and the reason delirium is defined by attention and consciousness rather than by hallucination**
Acute confusional state	Used in the literature either as an isolated symptom or as a synonym for the delirium syndrome	**A taxonomic overlap worth naming rather than resolving**
Bonhoeffer's acute exogenous reaction type	An umbrella construct: diverse somatic, toxic and infectious insults produce a limited repertoire of stereotyped psychopathological reactions, of which delirium is the prototype	**Many causes, few pictures**
Wieck's transitional syndromes	Reversible organic states with cognitive, affective or behavioural change occurring in *clear* consciousness	**Organic without clouding, which is why they are separated from delirium**
Twilight state (Dammerzustand)	Narrowed field of awareness with complex, coordinated, apparently purposeful action while disconnected from surrounding reality	**Abrupt onset and termination with subsequent amnesia for the episode. Complex partial status, post-ictal states, acute dissociative states, severe closed head injury**
Oneiroid syndrome	Dream-like fantastical delusional experience with scenic hallucinations and pseudohallucinations and catatonic phenomena, named from oneiros, dream, and eidos, form; first delineated by Wilhelm Mayer-Gross in 1924	**Internal projection and *double orientation*: the patient knows he is on a ward while also participating in a cosmic storyline. In delirium the hallucinations sit in external space**

Stupor, and why the word means two things. In Anglo-American neurological usage stupor equals the continental sopor, a semi-comatose state of severely diminished arousal, arousable only by vigorous or noxious stimulation and lapsing back when it stops, with flaccid, decerebrate or decorticate posturing, from structural brainstem lesions, bilateral thalamic infarcts or toxic-metabolic encephalopathy.

In psychiatric usage it is a psychomotor syndrome with preserved wakefulness: the patient is fully awake, with absent voluntary movement and speech, mutism, akinesia, negativism, waxy flexibility or intense distress, in catatonic schizophrenia, severe depression or dissociative states.

The separation rests on motor reactions, eye opening and ocular and pupillary reflexes, and on deciding whether a response to command is reflexive posturing or voluntary compliance.

CONDITION	AROUSAL	AWARENESS	MOTOR RESPONSES
Coma	Absent, eyes closed	Absent	**Reflex posturing only, no command following**
Vegetative state (apallic syndrome, coma vigile)	Preserved, spontaneous eye opening and sleep-wake cycles	Absent	**Reflexive posturing only, no reproducible voluntary response: wakefulness without awareness**
Minimally conscious state	Preserved	Minimal but definite	**Inconsistent but reproducible purposeful responses, visual pursuit, simple command following**
Locked-in syndrome	Preserved, full vigilance	Preserved, cognitive content intact	**Quadriplegia and anarthria with voluntary vertical gaze and blinking preserved: not a disorder of consciousness at all**

Oneiroid, endogenous against exogenous. In the schizophrenic form self-consciousness is disturbed with disorientation for personal identity, catatonic features are frequent, the picture is stable, resolution is gradual over days to weeks and the course favourable.

In the exogenous organic form, after encephalitis, trauma, intoxication or corticosteroids, self-consciousness is preserved, catatonic features are absent, the state intensifies in the evening like delirium and resolves rapidly, often after deep sleep, with progression to an amential syndrome portending a poor outcome.

Stoianov in 1961 described 6 sequential stages in recurrent schizophrenia, from a general-somatic and vegetative disorder through delusional mood and affective, fantastic and illusional depersonalisation to the catatonic-oneiroid state.

In lethal or febrile catatonia, hyperthermia, cerebral oedema and cardiac failure supervene; antipsychotics are ineffective and dangerous, and the indicated measures are intensive care, benzodiazepines, dantrolene, bromocriptine, amantadine or ketamine.

ASK AND VERIFY The standard follow-up here is the **Glasgow Coma Scale**, its three components and its score range. The library marks all of that unsourced. Name the scale as the standard tool for grading depth of unconsciousness, keep it separate from the phenomenological assessment above, and quote component scores and the range only from a textbook you can name.

Attention terms. Attention span and persistence is the duration focus is held on one continuous task. Distractibility is loss of focus to extraneous stimuli or intrusive thoughts, in mania, delirium and attention deficit hyperactivity disorder. Selective attention is attending to the relevant while inhibiting background. Sustained attention or vigilance is alertness over prolonged intervals.

Shifting attention is disengaging one set and engaging another, tested by reversing the days of the week or the months.

Memory: the three phases and the two kinds of failure. Registration or immediate memory is encoding, tested by immediate digit span. Retention or recent memory is storage and consolidation over minutes to days, tested by delayed recall of a short list. Retrieval or remote memory is access to long-stored autobiographical and semantic knowledge.

Failures divide into quantitative defects, the amnesias, hypomnesias and hypermnesias, and qualitative distortions, the paramnesias, confabulations and delusional memories.

FEATURE	ORGANIC AMNESIA	DISSOCIATIVE AMNESIA
Predominant deficit	**Anterograde greater than retrograde**	**Retrograde for autobiographical detail, with anterograde encoding intact**
Personal identity	Preserved: name, age and family intact	Frequently lost, with profound loss of autobiographical identity
Temporal gradient	Present: older memories survive over recent ones	Absent or reversed: dense loss for identity with intact recent facts
Procedural memory	Intact	Intact
Trigger	Neurological insult: hypoxia, stroke, trauma, thiamine lack	Severe psychosocial trauma, conflict or stressor
Course	Gradual recovery or permanent deficit	Often sudden termination, sometimes on removal of the conflict
Forensic risk	Low rate of coerced confession	Vulnerable to suggestion; misused abreaction can yield false confessions

Ribot's law is the gradient itself: memory dissolution takes newly formed, fragile memories first while older well-consolidated childhood memories resist. Organic conditions obey it; dissociative amnesia characteristically violates it, so a patient may fail to recall his own name, spouse or childhood while retaining newly learned vocabulary, current general knowledge and the events of the preceding hours.

QUALITATIVE DISORDER	DEFINITION	THE DISCRIMINATOR
Confabulation	False memories produced without intent to deceive, in clear consciousness, typically with an organically induced amnesia; the patient believes them	**Needs a dual deficit: diencephalic or medial temporal amnesia *plus* frontal executive dysfunction, ventromedial prefrontal and orbitofrontal, disrupting reality filtering and source monitoring**
Provoked (momentary, embarrassed) confabulation	A fleeting plausible fabrication produced on direct questioning, filling an active gap	**Common in early amnesic syndromes, including Korsakoff**
Spontaneous (fantastic) confabulation	Elaborate, unprovoked, implausible or grandiose narrative recited without prompting	**Implies the frontal component, not amnesia alone**
Retrospective falsification	Unconscious recolouring of genuine memories to match the present affective state or delusional frame	**The depressed patient remembers childhood as unrelieved misery; the manic patient recalls failures as triumphs**
Retrospective delusion	A delusion projected backwards in time onto a past event that did not occur	**A delusion about the past, not a gap being filled**
Reduplicative paramnesia	Belief that a place, person or structure exists in duplicate or has been relocated to an identical site, with the patient stating he has been in one or more hospitals bearing the identical name	**Associated with confabulation, spatial disorientation and denial of illness; linked to right prefrontal and parietotemporal injury disrupting familiarity recognition**
Identifying paramnesia	A novel situation experienced as duplicating an earlier one in every detail	**This is deja vu, paramnesia tout court**

Deja vu and jamais vu. Deja vu is a fleeting feeling, seconds to minutes, that a novel event has been experienced before, frequent in young adults and triggered by fatigue, stress, inattention or a sensory cue.

Four mechanisms are offered: dyssynchrony between temporal neocortex as store and hippocampus as consolidator; a dual processing delay making new input feel retrieved; source-monitoring failure giving familiarity without retrieval; and focal temporal lobe epilepsy, where it is an aura.

Jamais vu is the converse, an intimately familiar person or place feeling entirely alien, a loss of the sense of familiarity with recognition preserved, in focal temporal lobe epilepsy, exhaustion and dissociative states.

RULE Normal deja vu is transient, sporadic and recognised by the patient as illogical, leaving no post-ictal state. **Epileptic deja vu is stereotypic, recurs without triggers, lasts longer, and comes with an epigastric rising sensation, olfactory hallucination or automatisms.**

Korsakoff syndrome is the organic amnesic disorder of chronic alcohol dependence: profound anterograde amnesia, variable retrograde amnesia following the temporal gradient, provoked confabulation especially early, intact immediate digit span and preserved procedural memory, profound lack of insight with emotional blunting or apathy, and a clear sensorium.

In an Indian ward the peripheral signs of chronic alcohol dependence, hepatomegaly, parotid swelling and spider naevi, sit alongside it.

ASK AND VERIFY The examiner usually opens the Korsakoff question with the **eponym: who described it and in what year**. That attribution is unsourced in the library, so it is not printed here. Carry it from a primary text. Note the near miss: Karl Bonhoeffer's 1901 monograph on the acute mental illnesses of habitual drinkers appears in the Fish bibliography and is a separate, later account of acute alcoholic psychoses, not the origin of the eponym.

Ganser syndrome. First described by Sigbert Ganser in 1898, from a lecture given in 1897, in 3 prison inmates: a rare condition following profound psychosocial or physical stress.

Its core is *Vorbeireden*, the approximate answer, where a simple question is answered incorrectly yet the answer shows the essence of the question was grasped, for instance three legs for a horse, or snow described as black; it contrasts with nonsensical, disorganised or perseverative answers.

Its relatives are *Vorbeirechnen*, approximate calculation, where simple arithmetic is slightly missed while harder items are occasionally solved, and *Vorbeihandeln*, approximate action, holding a pen upside down or signing in empty air.

The fourth element is the hysterical twilight state: transitory narrowing of the field of consciousness with disorientation in time and space while personal self-consciousness and vigilance remain. Enoch and Trethowan set out 4 symptoms: approximate answers, clouding of consciousness, somatic conversion symptoms with sensory change and fluctuating hypoalgesia to pinprick, and hallucinations both visual and auditory.

Ganser's own account also required a limited time course, sudden resolution, and residual amnesia for the episode.

The literature is thin and worth saying so: 79 papers over 118 years describing 117 case reports; in a review of 43 cases 74 per cent were male; average age 32 years, range 15 to 62.

Aetiological models run in four directions.

- **Dissociative.** Classified under dissociative disorder not otherwise specified in DSM-IV, and omitted as a distinct disorder in DSM-5-TR.
- **Catatonic negativism.** The view of Ganser's mentor Nissl, supported by a 1904 report finding approximate answers in as many as 21 per cent of catatonic patients observed.
- **An organic substrate.** Head trauma in 2 of Ganser's 3 original patients.
- **The factitious and malingering continuum.** Encountered in forensic settings.

FEATURE	GANSER SYNDROME	MALINGERING	DEMENTIA	DEPRESSIVE PSEUDODEMENTIA
Approximate answers	**Prominent, consistent, classic**	**Inconsistent, exaggerated under observation**	**Absent; wrong answers reflect genuine cognitive loss**	**Answers "I do not know" rather than approximating**
Consciousness	Twilight state, narrowed field	Clear	Clear until late	Clear
Dynamic	Dissociative defence against overwhelming stress	External tangible gain	Neurodegenerative	Mood-congruent psychomotor slowing
Response to interview style	Worsens with formal testing, improves in an informal register	Worsens when observed, disability held consistently	Fails consistently in any register	Improves with encouragement and gentle persistence
Clock drawing	Characteristically preserved	Inconsistently impaired	Impaired, with spatial and conceptual errors	Intact or slow without neglect
Later amnesia	Common for the symptomatic phase	Claimed selectively	Progressive and irreversible	Absent once the depression remits

The bedside technique matters: avoid confrontation and shift from an inquisitorial examination to an informal conversational register, which diminishes the approximate answering and unmasks intact capacity. Clock drawing is preserved; the Draw-a-Person test yields simplistic stick figures reflecting regression rather than the disintegration of organic dementia.

Management is supportive: reassurance, removal of secondary gain, safety monitoring, and allowing resolution, which typically occurs within days once the precipitant abates.

TRAP Treating the approximate answer as pathognomonic. It occurs in schizophrenia, catatonia, mania, intellectual disability and malingering. The diagnosis needs the twilight state, the somatic conversion signs, and abrupt termination with amnesia.

HOLD THIS

Organic amnesia keeps identity and loses new learning; dissociative amnesia loses identity and keeps new learning. Confabulation needs amnesia plus a frontal deficit, not amnesia alone.

WHAT EARNS THE MARKS

- **Arousal and content named separately**: this earns the first mark before any grading
- **Identity against new learning**: the organic and dissociative patterns run opposite ways
- **Confabulation needs a frontal deficit**: amnesia on its own does not produce it
- **Approximate answers show comprehension**: the question was understood, which is the whole point

SEE ALSO 05 Catatonia, motor phenomena and volition 07 Emotion and the disorders of the self 14 Encephalopathy, the named periodic patterns, and drugs 21 Spotters: blood, territories, hydrocephalus, infection

7 · Emotion and the disorders of the self

PHENOMENOLOGY · TOPIC 07

How would you describe this patient's affect, and what separates depersonalisation from a delusion of control?

PHENOMENOLOGY TABLE, AFFECT AND THE SELF

THE SHAPE OF THE ANSWER

1 **Four words**	Mood, affect, emotion and feeling, in that order
2 **Examining affect**	Range, reactivity, congruity, stability, appropriateness
3 **The negative states**	Anhedonia, apathy, blunting and flattening on one grid
4 **Self-disorders**	Depersonalisation, derealisation and the accompanying disturbances
5 **Cotard**	The nihilistic delusion, and what it is not

Four words, in order, before any adjective. A *feeling* is a positive or negative subjective reaction to an experience. An *emotion* is a stirred-up state from physiological change in response to a specific event, tending to maintain or abolish it. *Mood* is the pervasive and sustained feeling tone that colours the total experience and perception of the individual.

Affect is the outward, observable expression of immediate cross-sectional emotion. Mood is longitudinal and largely reported; affect is cross-sectional and observed.

TRAP Describing mood with cross-sectional words or affect with longitudinal ones. "Mood is blunted" and "affect has been depressed for three weeks" both lose the mark. **Mood is pervasive and sustained; affect is immediate and observed.**

Examining affect. Report it in a fixed order.

- **Subjective report.** In the patient's own words.
- **Objective presentation.** Facial expression, posture, motor behaviour and prosody.
- **Appropriateness.** The fit with the situation, whose failure is inappropriate affect.
- **Congruence.** The fit with the patient's own thought content, whose failure is incongruent affect.
- **Range.** Full or restricted.
- **Intensity and depth.** From normal through constricted and blunted to flat.
- **Mobility and lability.** How readily the affect shifts within the interview.
- **Reactivity.** The response to external cues.
- **Communicability.** Whether the affect evokes empathic resonance in the examiner.

STAGE	SEVERITY	PSYCHOMOTOR ACTIVITY	COGNITIVE FEATURE	SETTING
Euphoria	Mild	Mildly increased or normal	**Optimism and unwarranted self-satisfaction, not in keeping with events**	Hypomania
Elation	Moderate	**Distinctly increased**	Confidence and enjoyment	Mania
Exaltation	Severe	Substantially increased	**Delusions of grandeur**	Severe mania
Ecstasy	Very severe	Variable, may be stuporous or delirious	**Rapture, grace and bliss, overwhelming supreme well-being**	Delirious or stuporous mania, mystical religious states

The four stages of mood elevation. Expansive mood is uninhibited expression of feeling with overestimation of self-importance; grandiosity is the belief or feeling of elevated importance in identity, wealth or power.

Anhedonia. From the Greek prefix for without and hedone, pleasure: the inability to experience pleasure or the lack of enjoyment in normally pleasurable activity.

Under standard operational criteria a major depressive episode needs 5 or more of 9 symptoms for at least 2 weeks, with at least one of them either depressed mood or anhedonia, the latter defined as loss of interest or pleasure in all or nearly all activities, most of the day, nearly every day.

Two primary forms: social anhedonia, failure to derive pleasure from social contact and relationships, and physical anhedonia, loss of pleasure from physical, sensory or aesthetic stimuli including food, music, touch and sexual intimacy; sexual and musical anhedonia are named subtypes, musical anhedonia arising from altered connectivity between auditory and reward networks.

The neurobiology is dopaminergic disruption in the ventral striatum. Reversible medical contributors, vitamin D deficiency and hypothyroidism, are worth excluding at the outset.

FEATURE	ANHEDONIA	APATHY	BLUNTED AFFECT	FLAT AFFECT
Primary deficit	**Hedonic capacity**	**Motivation, concern and drive**	**Intensity of emotional expression**	**Outward expression of emotion**
Level of observation	Primarily subjective	Subjective and behavioural	Objective clinician rating	Objective clinician rating
Capacity to feel sadness	Often intact despite absent joy	Diminished, the patient is unconcerned	Present internally despite poor display	Present internally despite absent display
Characteristic setting	Depression, schizophrenia	Dementia, frontal lesions, depression	Schizophrenia, severe depression	Chronic schizophrenia

Note the source caveat the examiner may probe: the cited sources define *constricted* affect, a significant reduction in normal emotional responses, and *flat* affect, the absence of emotional expression, but do not describe blunted affect as a separate category. Use the terms in that order and say so if pressed.

Alexithymia was introduced by Sifneos and Nemiah in 1970 for the inability to identify or describe emotions in words, and is a multidimensional personality construct of 3 factors: difficulty identifying feelings and distinguishing emotional states from the bodily sensations of arousal; difficulty describing and communicating feelings to others; and an externally oriented thinking style with pragmatic thought, restricted imagination and scarce fantasy.

The Toronto Alexithymia Scale is a 20-item self-report instrument on a 5-point Likert scale with a total range of 20 to 100, and a validated total of 61 or higher indicates alexithymia.

In clinical depressive cohorts alexithymia is identified in 50 per cent of patients, and the total score accounted for an estimated 70 per cent of the variance in psychological symptom reporting.

Factor 1 correlates with somatisation through somatosensory amplification, factor 2 with depressive severity and suicidal ideation, and factor 3 with psychoticism, social withdrawal and impoverished ideation, mirroring negative symptoms.

PHENOMENON	DEFINITION	THE DISCRIMINATOR
Incongruous affect	Disharmony between outward expression and concurrent thought content or speech, classically giggling while recounting tragedy in disorganised schizophrenia	**Mismatch with *ideation*; inappropriate affect is mismatch with the external *situation***
Labile affect	Rapid, abrupt, transient shifts without provocation and unrelated to external stimuli	**Mania, delirium, pseudobulbar palsy**
Affective incontinence	Severe loss of voluntary control over expression, with outbursts disproportionate to the subjective feeling	**The outburst exceeds what is felt**
Made affect	The emotional display is experienced as created and controlled by an external agency while the patient feels something discordant inside	**External attribution. Incongruous affect attributes nothing to anyone**
La belle indifference	Inappropriate lack of concern about a severe, disabling physical symptom	**Classically described in conversion or dissociative neurological symptom disorder**

Anxiety phenomenology, separated by trigger, focus, duration and autonomic involvement. Anxiety is apprehension with somatic autonomic accompaniment, muscle tension, perspiration and tachycardia, caused by the anticipation of danger. Free-floating anxiety is diffuse and unattached to any conscious thought, object or situation. Fear is apprehension provoked by a realistic, consciously recognised external danger.

A panic attack is an acute, self-limiting episodic attack of intense anxiety with overwhelming dread, fear of dying or losing control, and severe autonomic discharge.

A phobia is an irrational, exaggerated fear producing persistent avoidance: agoraphobia, rigid avoidance of situations where escape might be difficult or help unavailable if incapacitating symptoms occur; social phobia, fear of scrutiny in social or performance settings; and the specific phobias including acrophobia, arachnophobia, claustrophobia, gamophobia, hemophobia and zoophobia.

Agitation is severe inner tension with purposeless motor restlessness, and dysphoria an unpleasant mixed state of sadness, irritability and apprehension.

Depersonalisation and derealisation. Depersonalisation is an enduring or recurring feeling of detachment, estrangement or unreality about one's own mental processes, emotional life or body: the patient reports being an outside observer of his own life, operating like an automaton, emotionally disconnected from his own actions.

Derealisation is the same unreality directed at the surroundings, which are dreamlike, artificial, flat, lifeless, or seen through a pane of glass or a fog.

The phenomenological discriminator is that reality testing remains intact, insight into the subjective nature of the disturbance is clear, and the patient speaks in "as if" language, which separates this from the fixed belief of psychosis.

DSM-5 places it among the dissociative disorders; the full criterion set adds clinically significant distress, and exclusion of a substance effect, another medical condition and another mental disorder.

Community prevalence is estimated at 1 per cent to 2 per cent, with secondary comorbid depersonalisation documented in 40 per cent to 80 per cent of psychiatric inpatients, a figure reflecting referral bias rather than population incidence.

There is minimal gender difference, onset is most often in adolescence or early adulthood, and the course is predominantly chronic and fluctuating; discrete episodes may last hours, days, weeks or months, and many people have unremitting symptoms over years.

In a clinical sample of 204 participants, 50 per cent had prior depression while overt traumatic triggers were identified in only 14 per cent.

In one sample of 49 individuals, 44 reported childhood emotional abuse, yet a separate clinical sample found childhood trauma in 43 per cent and a cohort of 223 patients documented low rates of overt trauma, so adversity is a strong predictor and not a prerequisite.

The accompanying disturbances, because they are asked: macropsia, micropsia, teleopsia, visual snow, metamorphopsia, loss of depth into a two-dimensional field; floating above the body, limbs distorted or detached, the head packed in cotton; and de-affectualisation, where the patient can laugh or cry intellectually without internal resonance.

Hunter and colleagues' cognitive-behavioural model is the maintenance account: a mild dissociative sensation triggered by anxiety, stress or a substance is appraised catastrophically as brain damage or impending insanity, which escalates arousal and drives hypervigilant self-monitoring, which intensifies the unreality. Treatment follows: cognitive behavioural therapy with psychoeducation, grounding, restructuring of catastrophic appraisals and attention training outward.

No agent holds regulatory approval; serotonin reuptake inhibitors treat comorbid depression and anxiety, lamotrigine and naltrexone have tentative support, short-term clonazepam may reduce acute anxiety, and antipsychotics show limited efficacy in the pure disorder and frequently worsen numbness.

PHENOMENON	WHERE THE OBSERVING SELF SITS	REALITY TESTING	THE DISCRIMINATOR
Depersonalisation	Inside the body, but detached from it	Intact, "as if" preserved	**Ownership affirmed: the thoughts and actions are still mine**
Passivity phenomenon	Inside the body, authorship removed	Lost, a genuine delusion	**An external force is held to control the patient**
Visual autoscopy	Anchored within the physical body, looking at the double	Variably preserved	**The double is seen in extrapersonal space from a normal bodily vantage point**
Out-of-body experience	Dislocated outside the body, looking down at it	Variably preserved	**The vantage point has moved; temporoparietal junction dysfunction, near-death events, severe dissociation**
Heautoscopy	Oscillating between, or simultaneously in, both the body and the apparition	Profound ontological doubt	**Severe distress and confusion about which form is the genuine self; temporal lobe epilepsy, parieto-occipital lesions**
Body dysmorphic disorder	Inside the body, which is owned but judged ugly	Variable, poor to absent in some	**Aesthetic shame focused on specific areas, with mirror checking and camouflage; the body feels ugly, not unreal**
Cotard syndrome	The self is held not to exist	Absent, an unshakeable delusion	**Nihilistic denial of existence or of organs, with emotional numbness taken as proof of being dead**

Cotard syndrome was delineated by Jules Cotard in 1880 as *le delire de negation* and affects less than 1 per cent of clinical psychiatric populations.

Core features: the delusion of non-existence, of being deceased or a walking corpse; denial of body function, that organs are absent, the heart stopped, the blood dried; severe emotional numbness read as proof of being dead; the paradoxical delusion of immortality; and severe self-neglect with refusal of food, fluid and hygiene because corpses need nothing.

It is not an independent category but a severe symptom cluster secondary to affective, psychotic or organic disease, commonest in psychotic depression, and also described in bipolar depression, schizophrenia, dementia, Parkinson disease, multiple sclerosis, stroke, subdural haematoma, trauma, temporal lobe epilepsy, encephalopathy, uraemia, electrolyte derangement and central nervous system infection.

Management is medical stabilisation first, with metabolic screening, toxicology and neuroimaging, then bilateral modified electroconvulsive therapy as the rapid definitive treatment in severe psychotic depression, with antipsychotic and antidepressant combination, mood stabilisers in bipolar depression, and family psychoeducation.

TRAP Three here. Do not read a strange bodily feeling as **somatic passivity** without the deliberate external attribution. Do not read Cotard as hypochondriasis: the hypochondriacal patient is anxiously preoccupied with having or acquiring a disease, while the Cotard patient holds the definitive conviction that the part or function no longer exists. And in late-onset nihilistic delusions look for structural or metabolic disease before attributing the state to functional depression. The **three classical phases of Cotard syndrome** are unsourced in the library and are not printed here.

HOLD THIS

Reality testing is the whole discriminator between dissociation and passivity. "As if I were a robot" is depersonalisation; "a machine is moving my arm" is a delusion of control.

WHAT EARNS THE MARKS

- **Sustained against cross-sectional**: mood is reported over time, affect is observed now
- **Reality testing is the discriminator**: the as-if quality against a delusion of control
- **Hedonic capacity against motivation**: anhedonia and apathy are different deficits
- **The euphoria ladder in order**: euphoria, elation, exaltation, ecstasy

SEE ALSO 01 Form, content, and what makes a symptom primary 04 Possession, passivity and the first-rank list 06 Consciousness, attention and memory 08 Insight and the obsession boundary

8 · Insight and the obsession boundary

PHENOMENOLOGY · TOPIC 08

How would you assess insight, and where does the boundary with an obsession fall?

LONG CASE MENTAL STATE, AND THE PHENOMENOLOGY TABLE

THE SHAPE OF THE ANSWER

1	**Not one thing**	Three dimensions: awareness, relabelling, treatment need
2	**Bedside grades**	Six grades, with intellectual and emotional at the top
3	**Instruments**	The scales, their items, and what each one adds
4	**Course**	Insight and symptoms move semi-independently over time
5	**The obsession**	Ego-dystonic and resisted, against loss of mineness

Insight is not one thing, and saying so is the answer. Aubrey Lewis defined it as a correct attitude to a morbid change in oneself with the recognition that the change is morbid and mental, stressing that it is not acquiescence to a label but an introspective recognition that anomalous experiences represent disorder rather than external reality.

Consensus now treats it as evolving, continuous and multidimensional with numerous intermediate states. Anthony David established 3 partially overlapping dimensions in 1990: awareness of illness; relabelling of unusual mental events as pathological; and recognition of the need for treatment with willingness to cooperate.

Amador and Strauss built a 2-dimension model, awareness of illness and the ability to attribute symptoms to it, which underlies the clinician-administered unawareness scale.

GRADE	DESIGNATION	CLINICAL FEATURE	REPRESENTATIVE STATEMENT
I	Complete denial	Denies any illness or difficulty; experiences are normal or external reality	**Nothing is wrong with me, my family brought me here for no reason**
II	Slight awareness with simultaneous denial	Admits something feels off, vaguely, and dismisses it as trivial	**I am just tired from work, it is nothing serious**
III	Awareness blamed on external factors	Recognises distress but attributes it to stressors, other people or physical illness	**I feel unwell because the neighbours are harassing me**
IV	Awareness without clarity	Knows the symptoms come from within but cannot articulate their nature; bewildered	**Something inside my mind is not right, but I do not understand what**
V	Intellectual insight	Names the symptoms and accepts a disorder, without applying it to behaviour or future decisions	**The doctor says I have psychosis and need medication, but I will not need follow-up after discharge**
VI	True emotional insight	Connects symptoms to emotional life, personality and behavioural change, and uses the awareness for lasting change	**My suspicious thoughts were part of the illness, and I understand why staying in therapy matters**

The 6-point bedside grading used in Indian practical examinations. Flag the caveat honestly: it comes from a secondary source that presents the grades as a linear hierarchy, whereas primary empirical work shows the dimensions do not progress as a single continuum, so a patient may be fully adherent while still misattributing his hallucinations.

Cognitive insight is Beck's addition, the metacognitive capacity to examine, question and distance oneself from abnormal beliefs. It is rated on the self-report Beck Cognitive Insight Scale across 2 dimensions: self-reflectiveness, covering introspection, openness to corrective feedback and willingness to consider alternative explanations, and self-certainty, covering inflexibility, dogmatism and overconfidence in one's own beliefs.

The composite is self-reflectiveness minus self-certainty. Concordance with clinical insight is moderate, and over a 1-year follow-up the two can dissociate, with treatment awareness improving while the cognitive composite declines.

Impairment of insight is prominent in non-affective psychosis, with 50 per cent to 80 per cent of people diagnosed with schizophrenia showing marked deficits in awareness of having a mental illness.

INSTRUMENT	TYPE AND ITEMS	DIMENSIONS	SCORING NOTE
SUMD	Clinician-rated interview, 20 items	3 global items, awareness of illness, need for treatment, social consequences, for current and past states, plus 4 symptom-specific scales across 17 symptoms	**Items on a 5-point scale; treated as the gold standard objective clinical measure**
ITAQ	Clinician-rated interview, 11 items	Recognition of disorder in the first 5 items, attitudes to medication, hospitalisation and follow-up in 6	**Scored 0 for no insight to 2 for good insight; useful for tracking treatment attitudes**
AMDP insight items	Clinician-rated, 3 items	Not feeling ill, lack of insight, uncooperativeness or refusal of treatment	**Extracted from a 100-item inventory, scored 0 absent to 3 severe; maps onto David's dimensions**
BIS	Self-report, 8 items	David's 3 dimensions	**Sensitive to change but yields lower insight scores than interview methods**
BCIS	Self-report	Cognitive insight: self-reflectiveness and self-certainty	**Produces subscale scores and the composite; correlates only weakly to moderately with clinical insight**
VAGUS	Clinician-rated and self-rated versions	Multidimensional insight into illness, symptoms and treatment	**Built to allow direct clinician-patient concordance comparison**

The longitudinal picture. In a prospective study of 75 inpatients, 49 per cent to 66 per cent showed moderate to severe unawareness at baseline, and at follow-up between 6 months and 2 years after stabilisation, 29 per cent to 49 per cent still showed fair to poor insight.

Awareness of past episodes improves over time even where awareness of present illness does not. Insight and psychopathology behave as semi-independent domains: change in symptom severity does not track change in insight, with correlations staying below $r = 0.48$, and insight emerges as a factor distinct from positive, negative and disorganisation symptoms.

The negative and disorganisation dimensions predict poorer treatment attitudes later; the psychotic dimension correlates with disturbed attribution at baseline but predicts no longitudinal change.

Across disorders. Schizophrenia shows significantly greater impairment of current awareness, symptom attribution and awareness of social consequences than affective psychosis. Insight in

schizoaffective disorder is comparable to affective psychosis and significantly better than in schizophrenia on feeling ill and on awareness of social consequences.

In bipolar disorder insight fluctuates with polarity, characteristically impaired in acute mania and increasing in depressive phases. In obsessive-compulsive disorder insight is characteristically preserved, with recognition that obsessions are the patient's own and irrational.

The insight paradox. Improving insight promotes adherence, reduces relapse and improves psychosocial functioning, and at the same time raises the risk of negative affective outcomes. In a cohort of 443 patients with first-episode psychosis, higher baseline insight was cross-sectionally linked with more severe depressive symptoms, lower subjective well-being and heightened suicidality.

Over a 4-week early-intervention interval, however, improvement in insight was associated with symptom remission and better functional recovery without an increase in depressive symptoms, which suggests that interventions aimed at insight are reasonably safe over that short interval when paired with supportive and pharmacological care.

RULE Present insight in this order: the verbatim screening questions, then the patient's verbatim answer, then the grade, then David's dimensions, and say explicitly whether adherence is preserved despite impaired relabelling. Distinguish rapport, an established alliance, from amenability, cooperativeness with the interview without one.

TRAP Four ways to lose this mark. Reading adherence as insight: swallowing tablets because staff insist is not grade VI. Reading recited terminology as insight: naming the diagnosis without translating it into relapse planning is grade V. Expecting insight to recover as symptoms do: the domains are semi-independent. And presenting rising insight as unmixed good news without screening for emergent depression and suicidal ideation. Separately, **equating poor insight with legal incapacity is wrong**, and the statutory capacity and admission sections that govern this are unsourced in the library, so argue the principle and cite the Act from the forensic note, not from here.

The obsession, strictly defined. Jaspers, building on Westphal, defined a true obsession as an intrusive idea against which the person wages an active inner struggle, because the content is immediately experienced as nonsensical, absurd and alien to the personality.

Kurt Schneider put it as contents of consciousness that cannot be repressed although assessed as nonsensical or as dominating without reason, and a compulsion as an action arising from a true obsession, carried out under subjective compulsion with immediate awareness of its senselessness.

The operational systems simplified: DSM-5 requires recurrent, persistent thoughts, urges or images experienced at some time as intrusive and unwanted, which the individual attempts to ignore, suppress or neutralise, and compulsions as repetitive behaviours or mental acts driven by an urge or rigid rules, not realistically connected or clearly excessive.

ICD-11 is almost identical, dropping active resistance and permitting absent insight.

FEATURE	CLASSICAL CONCEPT (JASPERS, SCHNEIDER)	ICD-10	DSM-5 AND ICD-11
Definitional core	Intrusive thought with immediate awareness of absurdity and active inner struggle	Recurrent obsessional thoughts or compulsive acts, acknowledged as the patient's own	Recurrent persistent thoughts or images experienced as intrusive and unwanted at some time
Inner resistance	**Mandatory; its absence points to the schizophrenia spectrum**	**At least one thought or act must be resisted unsuccessfully**	**Not required; neutralising through a compulsion suffices, and the term is omitted from the criteria**
Insight	Preserved and simultaneous with the experience	Recognised as senseless or irrational, and distinguished from thought insertion	A dimensional specifier allowing good or fair, poor, or absent insight with delusional beliefs
Link to compulsion	The compulsion must stem from a true obsession	The compulsion can be unrelated to an obsession	Compulsions can occur without an articulated link to a discrete obsession

The resistance criterion has been progressively demoted: ICD-8 in 1974 made resistance to the feeling of compulsion defining; DSM-III in 1980 required ego-dystonicity and active attempts to ignore or suppress; DSM-IV in 1994 dropped that if the person neutralises by a compulsion instead; DSM-5 in 2013 and ICD-11 in 2019 omit the word. On the Yale-Brown scale resistance survives as a dimensional rating of how often the person yields against attempts to resist, not as a categorical gate.

Ego-dystonic against loss of mineness. Being alien to the personality and lacking the first-person perspective are different things.

In a true obsession the content is repugnant and contrary to the person's values while the structure of consciousness stays saturated with for-me-ness or ipseity: Schneider noted that obsessions retain the I-character of experience, and the patient acknowledges that it is his own mind producing the abhorrent idea.

In thought insertion the self-coincidence of consciousness is disrupted, the thought lacks mineness, and it is experienced as placed into the mind by an external agency.

Insight, in real time against in retrospect. In true obsessions insight is simultaneous with the intrusion: even at peak anxiety the patient recognises concurrently that the doubt is irrational. Operational instruments rate what the patient articulates in a calm interview, and many people with severe obsessions lose insight transiently during the paroxysm and regain it afterwards.

So elicit whether he recognised the absurdity at the moment the compulsion was triggered, not whether he can say so now.

SYMPTOM	AGENCY AND MINENESS	TRIGGER AND CONTEXT	EMOTIONAL TONE
Obsession	**Intact first-person perspective**	Often elicited by external or associational triggers	**Marked distress with active struggle**
Thought interference	**Diminished mineness, experienced as autonomous**	Sudden irruption without context, derailing the flow	**Emotionally neutral, evokes perplexity, cannot be suppressed**
Silent thought echo	**Attenuated mineness, acoustic-like doubling**	Autonomous repeating or doubling of thoughts	**Experienced passively; precedes psychotic transition**
Thought insertion	**Severely compromised mineness, external attribution**	Direct implantation into consciousness	**The thoughts belong to an outside agency**

Pseudo-obsessions and the spectrum markers. The term is Rado's and Vanggaard's, from the pseudoneurotic schizophrenia of Hoch and Polatin: repetitive thoughts, images or ruminations lacking true inner resistance and insight, frequently violent, macabre, sexual or dysmorphophobic, with chaotic affect or striking shallowness.

ICD-10 retains obsessive ruminations without inner resistance as a criterion for schizotypal disorder, and Bleuler classified obsessive-compulsive phenomena in schizophrenia as automatisms, "it thinks in me".

The numbers behind the concern.

- **Symptoms inside schizophrenia.** About 30 per cent of patients with schizophrenia show obsessive-compulsive symptoms.
- **Re-examination in obsessive-compulsive clinics.** This has uncovered undetected spectrum disorders in 8 per cent to 29 per cent, with schizotypal personality disorder in 33 per cent.
- **Comorbid schizotypal personality disorder.** Runs 10 per cent to 25 per cent in clinical samples, is strongly associated with poor insight and with spectrum disorders in first-degree relatives, and predicts poorer response to serotonin reuptake inhibitors and exposure therapy.

The phenomenological markers that should make you re-open the diagnosis are drawn from the Examination of Anomalous Self-Experience and from classical phenomenology.

- **Spatialisation of thought.** Intrusive thoughts or images with an objective spatial location inside the head, repeating at the back or located at the front.
- **Cinematographic imagery.** Prolonged autonomous highly detailed visual scenes lasting seconds or minutes that zoom in on details independently of will, against the fleeting obsessional image.
- **Interrogative attitude.** Minkowski's term for endless questioning of trivial everyday phenomena, reflecting lost pragmatic attunement to the world.
- **Morbid geometrism.** Minkowski's term for compulsive static preoccupation with arrangement, symmetry and bilateral balance, being compelled to touch an object with the left hand because the right hand touched it, without any harm-avoidance premise.
- **Compensatory compulsions.** Performed to compensate for bodily depersonalisation, lost common sense or profound ambivalence rather than to neutralise a discrete harm obsession.

Content carries less weight than candidates expect. DSM-5 names cleaning with contamination fears and washing; symmetry with ordering, arranging, repeating and counting; forbidden or

taboo thoughts, aggressive, sexual or religious, with mental neutralising and reassurance seeking; and fears of harm with checking.

Hoarding is no longer an obsessive-compulsive content dimension but an independent category whose behaviour must not be explained by delusions in schizophrenia. And symmetry, aggressive, violent and sexual contents are also described in obsessive phenomena in schizophrenia, so content supports the diagnosis without settling it. Form, that is resistance, insight and mineness, is the decisive level.

TRAP Calling depressive rumination or generalised worry an obsession because it is unpleasant. Ruminations and worries are **ego-syntonic in form**: the patient regards them as relevant, realistic concerns. An obsession is ego-dystonic because its premise is immediately identified as senseless and alien. And absent-insight obsessive-compulsive disorder is not a primary delusion: the belief grows out of repetitive doubt and magical neutralising, whereas a primary delusion arrives as a sudden unmediated revelation on a background of delusional mood.

HOLD THIS

An obsession keeps mineness and fights back; thought insertion loses mineness and does not. Resistance is no longer in the criteria, but its persistent absence still points away from primary obsessive-compulsive disorder.

WHAT EARNS THE MARKS

- **Adherence is not insight**: swallowing tablets on instruction does not reach the top grade
- **Three dimensions named first**: the structure of the answer comes before any grade
- **Mineness kept and fought**: the obsession against thought insertion
- **Screening as insight rises**: improving insight calls for depression and suicide screening

SEE ALSO 01 Form, content, and what makes a symptom primary 04 Possession, passivity and the first-rank list 07 Emotion and the disorders of the self 11 Suicide and self-harm: the Indian numbers

Named syndromes and culture

The eponym question is a memory test with a clinical tail: the examiner wants the name, the one-line definition, the neighbour it is confused with, and then what you would do about it.

The culture question is different in kind, because in Indian practice these presentations are common rather than exotic, and the mark is for treating them as idioms of distress that mask treatable psychiatric morbidity.

9 · Culture-bound syndromes and the cultural formulation

NAMED SYNDROMES AND CULTURE · TOPIC 09

What is a culture-bound syndrome, and how would you work up a man presenting with Dhat?

NAMED SYNDROMES TABLE, LONG CASE FORMULATION

THE SHAPE OF THE ANSWER

1 The category	Idiom of distress, defined before the case is opened
2 Dhat	Semen-loss anxiety, its clinic share and its comorbidity
3 Koro	Genital retraction, with the exclusion step taken first
4 The regional list	Possession, ascetic and gilhari states, and the rest
5 The interview	The Cultural Formulation Interview, and what it adds

Define the category before the case. A culture-bound syndrome is a clinical entity that is not a mere variant of a universally recognised psychiatric disorder, has a geographically defined distribution, and is largely shaped in its symptomatology and expression by native beliefs, assumptions and idioms of distress.

Western classifications once treated these as exotic folk illnesses confined to non-Western countries; cross-cultural psychiatry has since established that culture shapes the perception, interpretation, somatic amplification and help-seeking pathway of all psychiatric morbidity.

DSM-IV added the Outline for Cultural Formulation as a narrative guide; DSM-5 replaced the appendix of culture-bound syndromes with the operationalised Cultural Formulation Interview and divided the phenomena into cultural syndromes, cultural idioms of distress and cultural explanations; and ICD-11 moved away from categorising them as exotic deviations, coding them as QE82, other specified cultural syndromes.

SYNDROME	SHARE OF ONE CLINIC SAMPLE	CHARACTERISTIC FOCUS	PRINCIPAL COMORBIDITY
Dhat syndrome	80 per cent	Semen loss and somatic exhaustion	**Depressive disorder**
Possession syndrome	14 per cent	Replacement of personal identity by an external entity	**Depressive disorder and stressors**
Ascetic syndrome	2 per cent	Severe self-denial and social withdrawal	**Depressive disorder**
Gilhari syndrome	2 per cent	Sensation of a moving subcutaneous swelling	**Anxiety and depression**
Koro	2 per cent	Anxiety about penile retraction into the abdomen	**Generalised anxiety and depression**

Proportions from a cross-sectional study of 50 consecutive patients presenting with culture-bound syndromes to a tertiary psychiatric outpatient department in Delhi. In Indian community surveys the prevalence of Dhat syndrome has been documented at 12.5 per cent. Across Indian studies, depressive disorder is the single commonest associated comorbidity.

Dhat syndrome. Severe anxiety, somatic distress and hypochondriacal preoccupation attributed to perceived loss of semen.

The belief system sits in traditional Ayurvedic physiology, in which semen (*shukra*) is the final and most refined product of bodily transformation and the reservoir of physical stamina, mental vigour and longevity, so its loss is believed to drain vitality, produce impotence and shorten life; folk belief holds that many drops of blood produce one drop of semen.

The distortions are sustained by sexual taboo, absent formal sex education, and advertising by roadside practitioners. Patients are typically young, single or newly married men from rural or semi-urban backgrounds, though educated urban men present too.

They report a whitish discharge on micturition, post-micturition dribbling, nocturnal emissions, involuntary leakage on straining at stool, or preoccupation after masturbation; pervasive fatigue, weakness, unrefreshing sleep, aches in the thighs and back, dizziness, poor concentration and loss of appetite; erectile dysfunction, premature ejaculation or perceived loss of genital size and tone; and marked guilt, dread of sexual inadequacy, demoralisation, withdrawal and irritability.

The comorbidity to screen for is depressive disorder, generalised anxiety and panic, somatic symptom disorder, and psychosexual dysfunction.

INDIAN PRACTICE NOTE

Management is integrated and non-confrontational. Routine urine examination demonstrates the absence of infection, pus cells or pathological spermatorrhoea and serves as an objective reassurance tool, with semen analysis if the complaint persists. Psychoeducation explains male reproductive anatomy and that discharge on straining at stool is physiological prostatic or seminal vesicular secretion rather than loss of vital essence. Culturally adapted cognitive behavioural therapy addresses the catastrophic assumptions, re-attributes somatic fatigue to emotional distress, and restructures sexual guilt. Counsel the family to stop discussing the complaint with neighbours, which prevents ridicule and isolation.

Pharmacotherapy is directed at the comorbid disorder. A reported Indian inpatient course used vilazodone 40 mg daily with clonazepam 0.5 mg daily alongside psychoeducation, with clinical recovery within 2 weeks.

Koro. The name comes from the Malay for "to shrink", or from *kura*, tortoise, for the retraction of the head into the shell. It is an acute, severe anxiety state centred on the catastrophic belief that the external genitalia are shrinking and retracting into the abdomen and that complete disappearance will cause death.

Patients grip the penis manually or apply strings, clamps or weights, occasionally producing trauma or ischaemia, and family members often assist in holding the organ. Yap described those affected as emotionally dependent, immature, lacking confidence in their virility and in persistent psychosexual conflict.

In India it occurs sporadically in young adult men, sometimes with Dhat syndrome, and as explosive epidemic outbreaks documented in North-Eastern states and in Assam and West Bengal. Management is urgent reassurance, immediate psychoeducation about the anatomical impossibility of abdominal retraction, acute anxiolysis, and treatment of the underlying affective or anxiety disorder.

ASK AND VERIFY Indian examiners reliably probe the exclusion step before the culture-bound label is applied: a **secondary or organic cause of the retraction experience**, for example a metabolic disturbance, substance intoxication or withdrawal, or a central nervous system lesion. The specific list of organic mimics and the work-up for them are unsourced in the library, so raise the exclusion unprompted and take the list from a standard textbook. Do not attach a chapter number to code QE82 either; the code and its title are sourced and its placement is not.

FEATURE	DISSOCIATIVE POSSESSION TRANCE	SCHIZOPHRENIA SPECTRUM	POSSESSION OF THOUGHT ON THE MENTAL STATE EXAMINATION
Cultural status	**Culturally sanctioned, coherent with community religious folklore**	**Deviant and bizarre, outside cultural norms**	**A psychopathological symptom recognised across settings**
Course	Episodic, self-limiting, precipitated by interpersonal stress	Continuous, chronic, with insidious functional decline	Chronic, episodic or persistent
Phenomenology	Total identity replacement by an external agent, with vocal and motor display	Delusions of control, voices discussing the patient	Thought insertion, withdrawal or broadcast, that is thought alienation
Between episodes	Personality and functioning preserved	Blunted affect, avolition, cognitive decline	Impaired insight with cognitive and functional deficits

Separate the transcultural possession state from Schneiderian thought possession explicitly. The first is a dissociative trance in which identity is temporarily supplanted in a sanctioned context; the second is an individual psychotic symptom of thought alienation.

The rest of the regional list. *Amok*, traditionally described in Southeast Asia, is an initial period of brooding, withdrawal and depression followed by a sudden unprovoked outburst of indiscriminate aggressive, violent or homicidal behaviour against people and objects, terminating in exhaustion, sleep and amnesia for the event, sometimes ending in suicide or homicide.

Latah, also described in Southeast Asia, is severe hypersuggestibility with automatic obedience, echolalia and echopraxia precipitated by a sudden startle, most often in middle-aged or elderly women.

Ascetic syndrome, described by Neki in North India in 1972, is severe self-denial, withdrawal, an ascetic lifestyle, neglect of grooming and study avoidance in adolescents and young adults, viewed as a pseudo-spiritual path but often reflecting adolescent depression, obsessive pathology or a psychotic prodrome.

Gilhari syndrome is the sensation of a moving squirrel-like swelling under the skin travelling upward toward the throat, a somatic manifestation of panic or illness anxiety whose nosological status is unsettled.

Suudu syndrome, from Tamil Nadu, is painful urination and lower abdominal burning attributed to accumulated inner heat from dehydration, rooted in Siddha and Ayurvedic hot-cold humoral balance and managed indigenously with sesame or castor oil at the navel and soaked fenugreek seeds.

Mass hysteria appears as community outbreaks of acute psychogenic somatic symptoms or shared perceptual phenomena, spreading by suggestion, social reinforcement and shared belief.

The Cultural Formulation Interview. The DSM-5 Cross-Cultural Issues Subgroup operationalised the narrative outline into an instrument with 3 components.

- **The core interview.** A semistructured questionnaire administered to the patient.
- **The informant version.** A parallel interview for family, carers or community members.
- **The supplementary modules.** These expand specific domains such as explanatory models, religion and spirituality and cultural identity, or address specific populations such as immigrants, refugees, children and older adults.

The core interview assesses four domains.

- **Cultural definition of the problem.** In the patient's own idiom.
- **Cultural perceptions of cause, context and support.** Covering explanatory models, perceived causes, psychosocial stressors and the role of the community.
- **Cultural factors affecting self-coping and past help-seeking.** Including engagement with traditional and faith healers.
- **Cultural factors affecting current help-seeking.** Covering expectations of care, barriers and perceived clinician-patient cultural divergence.

It was validated in an international field trial of 321 patients across 11 clinical settings in 6 countries, which confirmed feasibility, acceptability and high clinical utility in building rapport and tailoring the formulation.

TRAP Treating Dhat syndrome as a urological problem. Prescribing placebos, aphrodisiacs or symptomatic tonics reinforces the belief that semen leakage is a physical disease. And do not assume these presentations belong to illiterate or rural patients only: educated urban men, including graduates, develop full presentations under stigma, internet misinformation and unaddressed sexual taboo. Distinguish sporadic cases, rooted in individual psychosexual guilt and isolation, from epidemic outbreaks driven by community panic, mass suggestion and rumour.

HOLD THIS

A culture-bound syndrome is an idiom of distress, and the commonest thing it is an idiom for is depression. Screen for the mood disorder in every one of these presentations.

WHAT EARNS THE MARKS

- **Idiom, not entity**: naming the category keeps you from exoticising the patient
- **Screening for the mood disorder**: depression is the commonest comorbidity in these presentations
- **Organic cause cleared first**: the retraction experience has secondary causes to exclude
- **The literacy assumption refused**: these presentations are not confined to one social group

SEE ALSO 01 Form, content, and what makes a symptom primary 07 Emotion and the disorders of the self

10 · The eponym table: misidentification and the rest

NAMED SYNDROMES AND CULTURE · TOPIC 10

Tell me the delusional misidentification syndromes, starting with Capgras.

NAMED SYNDROMES TABLE, EPONYMS

THE SHAPE OF THE ANSWER

1 **Group first**	Hypoidentification against hyperidentification, before any list
2 **The grid**	Attribution, a one-line definition, the discriminator
3 **Folie a deux**	The shared psychosis, with its subtypes
4 **The models**	The two-factor account, and what each factor supplies
5 **The lesions**	Right hemisphere, right frontal, with the figures attached

Group the misidentifications before you list them. The delusional misidentification syndromes are fixed false beliefs about the identity of familiar persons, places, objects or the self; the umbrella term was systematised by Christodoulou in 1986, and the unifying theme is the concept of the double (*sosie*).

Christodoulou grouped them along an axis of familiarity: *hypofamiliarity* or under-identification, and *hyperfamiliarity* or over-identification. Capgras is hypofamiliarity for a person, Cotard and mirrored-self misidentification are hypofamiliarity for the self; Fregoli and intermetamorphosis are hyperfamiliarity for a person, reduplicative paramnesia is hyperfamiliarity for a place, and the syndrome of subjective doubles is hyperfamiliarity for the self.

SYNDROME	ATTRIBUTION AND DATE	DEFINITION IN ONE LINE	THE DISCRIMINATOR
Capgras	Capgras and Reboul-Lachaux, 1923	A close relative or spouse has been replaced by an identical imposter	**Appearance identical, identity believed replaced. Hypofamiliarity**
Fregoli	Courbon and Fail, 1927, after the quick-change actor Leopoldo Fregoli	A familiar persecutor takes on the appearance of various strangers	**Appearance varied, identity believed the same. Hyperfamiliarity**
Intermetamorphosis	Courbon and Tusques, 1932	People transform into others, sharing both physical appearance and psychological identity	**Both form and identity change, unlike Fregoli where the form is unchanged**
Syndrome of subjective doubles	Christodoulou, 1978	A physical doppelganger of the patient exists and acts independently	**A delusion about a duplicate elsewhere, not a visual hallucination of one's own body**

SYNDROME	ATTRIBUTION AND DATE	DEFINITION IN ONE LINE	THE DISCRIMINATOR
Reduplicative paramnesia	Coined by Arnold Pick, 1903	A place has been duplicated, relocated, or exists in two geographic sites at once	**Hyperfamiliarity for place; right prefrontal and parietotemporal injury**
Mirrored-self misidentification	Described within the group	One's own reflection is another person	**Hypofamiliarity directed at the self through the mirror**
Cotard	Jules Cotard, 1880, as le delire de negation	Nihilistic delusion of being dead, non-existent, putrefying, or without organs, blood or brain, sometimes with delusions of immortality	**Hypofamiliarity for the self. A classic series of 100 cases by Berrios and Luque in 1995**
Charles Bonnet	Lilliputian hallucinations introduced by Leroy in 1909	Formed, complex, vivid visual hallucinations in severe pathological vision loss, without psychiatric illness, cognitive decline or delirium	**Insight retained, and it is visual only. Symptoms are temporary and usually resolve within 12 to 18 months**
de Clerambault (erotomania)	Also psychose passionnelle	Fixed belief that another person, typically of higher social or celebrity standing, is secretly in love with the patient, with innocuous cues read as coded affirmation	**Classically three stages: hope, resentment, then grudge and persecution. Forensic risk of stalking, harassment and threat**
Othello (morbid jealousy)	Delusional jealousy	Fixed conviction of partner infidelity substantiated by trivial proofs, creases on bedsheets, minor travel delays	**High risk of intimate partner violence, coercive surveillance and homicide; seen in delusional disorder, paranoid schizophrenia, alcohol use disorder, dementia and right frontal lesions**
Ekbom (delusional parasitosis)	Delusional infestation	Fixed monothematic delusion of infestation of skin, tissues or home by living organisms, with formication	**The matchbox sign: skin flakes, scabs, hair or lint brought in a container as proof. Excoriations and chemical burns from picking and antiseptics**

SYNDROME	ATTRIBUTION AND DATE	DEFINITION IN ONE LINE	THE DISCRIMINATOR
Ganser	Syndrome of approximate answers	Approximate answers, clouding of consciousness, conversion features, and hallucinations	**The answer shows the question was understood. In dementia the errors are genuine agnosia, anomia or memory failure without thematic proximity**
Munchausen	Factitious disorder imposed on self	Intentional fabrication, induction or simulation of signs with no external incentive; the drive is the sick role	**The triad: peregrination, pseudologia fantastica, and multiple surgical scars. Malingering instead has an overt external incentive**
Munchausen by proxy	Factitious disorder imposed on another	Fabrication or induction of illness in a dependent, usually a child, by a carer	**Recognised as severe child abuse requiring statutory child protection notification**
Diogenes	Senile squalor	Severe domestic and personal squalor, extreme self-neglect, hoarding of refuse or useless items (syllogomania), withdrawal, and indifference with little insight or shame	**Primary or secondary to frontal dementia, chronic schizophrenia, severe depression or substance misuse**

Folie a deux is a shared delusional disorder in which delusional ideation is transmitted from a primary individual with established psychosis to one or more secondary individuals.

Four subtypes.

- **Folie imposee.** Transferred to a passive suggestible recipient, and remitting on separation.
- **Folie simultanee.** Delusions emerging at the same time in genetically predisposed, closely knit individuals.
- **Folie communiquee.** The secondary person develops the delusion after prolonged resistance, and keeps it after separation.
- **Folie induite.** New delusions are added to an already psychotic individual under a primary patient's influence.

The essential first intervention is physical separation of the two; the primary patient needs intensive antipsychotic treatment, and the secondary person's beliefs frequently attenuate or resolve after separation without long-term pharmacotherapy.

The cognitive models, for the two-hit question. Coltheart's two-factor account: factor 1 is an anomalous perceptual or cognitive experience, such as loss of affective familiarity for a face, and factor 2 is impaired belief evaluation and hypothesis testing localising to right frontal networks, which prevents rejection of the implausible explanation and crystallises the delusion.

Ellis and Young's dual visual route model gives the substrate: a ventral occipitotemporal visual-limbic stream mediates conscious overt structural face recognition, and a dorsal temporoparietal stream to amygdala and cingulate mediates affective covert familiarity and autonomic reactivity measurable as skin conductance. In prosopagnosia the ventral pathway is damaged, so conscious recognition fails while autonomic reactivity persists.

In Capgras the dorsal pathway is disrupted while the ventral is intact, so the patient recognises the features and feels nothing: she looks identical to my mother, therefore she must be an imposter. An autobiographical memory account runs alongside: hypofamiliarity when external percepts fail to trigger autobiographical memory, hyperfamiliarity when internal autobiographical memories are unconstrained by conflicting perception.

The lesion evidence, worth quoting precisely. A systematic review of 61 patients with lesion-confirmed acute neurological injury and misidentification found this distribution.

- **Direction of the error.** 28 hypofamiliar, 27 hyperfamiliar and 6 with both.
- **Demographics.** Mean age 52 years, median 56, men 67 per cent, that is 41 of 61.
- **Aetiology.** Ischaemic stroke 39 per cent, traumatic brain injury 28 per cent, haemorrhagic stroke 20 per cent and other causes 13 per cent, with premorbid psychiatric illness in 7 per cent.

Right hemisphere involvement was present in 92 per cent and right frontal involvement in 63 per cent, both at p less than 0.001, with left hemisphere involvement in 8 per cent and bilateral lesions in 20 per cent; left hemispheric damage was commoner in hypofamiliar than hyperfamiliar cases.

Of 56 patients evaluated neuropsychologically, the deficits ranked in this order.

- **The commonest.** Memory impairment at 73 per cent, then executive dysfunction 52 per cent, visuospatial impairment 38 per cent and attentional neglect 32 per cent.
- **The rest.** Facial recognition deficits 16 per cent, confabulatory memory disorder 13 per cent and language deficits 5 per cent.
- **The contrast that carries a mark.** Executive deficits were commoner in hyperfamiliar than hypofamiliar delusions, 67 per cent against 33 per cent, $p = 0.01$.

Targets were place in 58 per cent, person in 50 per cent, self in 14 per cent, event in 9 per cent and object in 3 per cent, with multiple concurrent syndromes in 29 per cent. Median latency to onset was 60 days and median resolution 42 days, with bilateral lesions and confabulation each predicting longer duration.

About 25 per cent of Capgras cases across the literature show an underlying neurological contribution, rising with age.

RULE A delusional misidentification syndrome in a patient with a new neurological event is **a right-hemisphere sign until proved otherwise**, and right frontal in particular, which is exactly what the two-factor model predicts.

ASK AND VERIFY Management of **de Clerambault syndrome**, including protection of the target, is unsourced in the library and is not stated here. The examiner expects treatment of the underlying delusional or affective illness, risk management for the target, and admission where forensic risk is high; verify that against a primary text. In Othello syndrome the same applies to the statutory route for urgent admission and to the legal insanity provisions: name the Act and confirm the section numbers from the Bare Acts rather than reciting them from a clinical review. For Diogenes syndrome, note that the Maintenance and Welfare of Parents and Senior Citizens Act 2007 is a maintenance and welfare statute with a tribunal that can order maintenance, not a hygiene enforcement or adult protective services mechanism, so squalor and self-neglect in India are managed through community outreach, guardianship and supported decision-making provisions, municipal and social welfare services, and family involvement.

HOLD THIS

Capgras is the same face with a changed identity; Fregoli is a changed face with the same identity. Prosopagnosia loses recognition and keeps the autonomic response; Capgras keeps recognition and loses the response.

WHAT EARNS THE MARKS

- **Same face, changed identity**: the Capgras line, said before anything else
- **Grouping before listing**: hypo and hyper identification organises the whole table
- **Recognition kept, response lost**: Capgras against prosopagnosia
- **A right-hemisphere sign**: a new misidentification alongside a neurological event

SEE ALSO 01 Form, content, and what makes a symptom primary 07 Emotion and the disorders of the self 09 Culture-bound syndromes and the cultural formulation 19 Imaging in the primary psychiatric disorders

11 · Suicide and self-harm: the Indian numbers

NAMED SYNDROMES AND CULTURE · TOPIC 11

What are the Indian suicide figures, and what does the current law say?

NAMED SYNDROMES TABLE, AND THE LAW OR ETHICS QUESTION

THE SHAPE OF THE ANSWER

1 Provenance	How a death becomes a statistic in the national record
2 The numbers	Rate, total deaths, method shares, age and income
3 Methods	Hanging and poisoning, and the means-restriction argument
4 Causes and gaps	Reported causes, and why the count is an undercount
5 Theory and law	Durkheim on two axes, then the statutory position

Start with how a death becomes a statistic. The Government of India classifies a death as suicide if three criteria are satisfied: death has occurred; the intent to die originated within the deceased; and a reason exists for the person to end their life, which may or may not be recorded in a note.

If any one is absent the death is counted under another heading such as illness or murder. National statistics are collated by the National Crime Records Bureau under the Ministry of Home Affairs and published in the annual Accidental Deaths and Suicides in India report, first published in 1967.

Primary data come from police first information reports and inquest records, compiled by District Crime Records Bureaus, forwarded to State Crime Records Bureaus and submitted onward, with cities of 10 lakh population or more recorded separately.

PARAMETER	FIGURE	WHAT THE EXAMINER WANTS SAID ABOUT IT
Total reported suicides, 2023	171,418	**A rise of 0.29 per cent over the 170,924 recorded in 2022**
National rate, 2023	12.3 per 100,000 population	**A slight decline from the peak of 12.4 in 2022, having risen from 10.2 in 2018**
Peak vulnerable age	18 to 30 years 33 per cent and 30 to 45 years 33 per cent	**Two thirds of all suicides fall in the 18 to 45 bracket, which inverts the Western age pattern**
Minors and elderly	Under 18 years 6 per cent; over 60 years 9 per cent	**Among minors female suicides outnumber male, an inversion of the adult ratio**
Income	Annual income below 1 lakh rupees: 66 per cent	**Up from 64 per cent in 2022; the gradient runs inversely with income**
Largest occupational group	Daily wage earners, 47,170 deaths, 28 per cent	**Then housewives at 24,048, self-employed 20,148, salaried 16,517, unemployed 14,234, students 13,892 and agricultural workers 10,786**
Students	13,892 deaths, 8.1 per cent of the national total	**Up from 7.6 per cent in 2022**
Highest rate, union territory or small state	Andaman and Nicobar Islands 49.6; Sikkim 40.2	**Rates, not volumes: quote the denominator or the number means nothing**
Highest rate, major state	Kerala 30.6, Telangana 27.7	**Against the lowest, Bihar at 0.7, which is a reporting artefact as much as an epidemiological fact**
Highest volume	Maharashtra 22,687 (14.16 per cent), Tamil Nadu 19,483, Madhya Pradesh 15,662	**The top three states contribute more than a third of the national total**
Urban excess	Mega cities averaged 16.2 per 100,000 in 2023	**Against the national average of 12.3**

Methods, and the means-restriction argument. Hanging is the leading method nationwide across both sexes and is increasing, accounting for 60.9 per cent in 2023 against 58.2 per cent in 2022. Poisoning is second at 25.0 per cent, with agricultural pesticide ingestion a major share, and drowning accounts for 4.1 per cent.

Fire and self-immolation, jumping and firearms follow in lower proportions. The policy point to make: after a national ban on endosulfan over the period 2011 to 2014, pesticide-related suicide rates fell measurably, which is the cleanest Indian demonstration that restricting access to lethal means works.

Reported causes. The Bureau assigns a single primary cause to each death, which oversimplifies a multifactorial event, and you should say so before quoting the table.

Family problems lead nationally with 54,653 deaths, then illness physical and mental with 32,503, marriage and relationship issues with 17,057, alcohol and substance misuse with 12,019, and economic strain with 10,903; in the 2019 percentages, family problems were 32.4 per cent, illness 17.1 per cent, economic causes 7.0 per cent combined, love affairs 4.5 per cent and examination failure 2.0 per cent.

The psychiatric layer belongs alongside these, not instead of them: affective disorders, substance use disorders and psychoses are significant risk factors, and the National Mental Health Survey found that nearly 80 per cent of individuals with mental morbidity had received no formal care for over a year.

Domestic violence is a prominent independent risk factor for suicidal behaviour among married women, and infertility and dowry harassment are categories in which female deaths exceed male.

Under-reporting. Administrative police data underestimate true mortality because of stigma, fear of legal consequences and incomplete rural registration.

Comparing the Global Burden of Disease estimates with the Bureau's reports across a decade gives the size of the gap.

- **Additional deaths.** An additional 802,684 suicide deaths, 333,558 male and 469,126 female.
- **Annual average under-reporting.** 27 per cent for males, range 21 per cent to 31 per cent, and 50 per cent for females, range 47 per cent to 54 per cent.
- **Where it is worst.** Among youth, older adults and low Socio-Demographic Index states.

A landmark study estimated 187,000 suicides in India for a year in which 134,600 were officially reported.

THE LAW, AND THE STRATEGY

Section 309 of the Indian Penal Code historically criminalised attempted suicide with simple imprisonment for up to 1 year, a fine, or both. The Mental Healthcare Act 2017, passed in April 2017 and commenced in July 2018, overrides it: Section 115 establishes a statutory presumption that a person who attempts suicide shall be presumed, unless proved otherwise, to have been under severe stress, and shall not be tried or punished. The National Suicide Prevention Strategy, released in November 2022, sets a target of reducing suicide mortality by 10 per cent by 2030, aligned with the multisectoral framework and integrating health, education, social justice and agriculture ministries with state administrations and grassroots health workers, prioritising restriction of access to lethal means, gatekeeper training in educational institutions and communities, responsible media reporting, and strengthened surveillance.

TRAP Saying Section 309 was deleted or repealed. It remains in the Code; what changed is that Section 115 created a rebuttable presumption of severe stress that bars trial and punishment. The further claim that the statute places a duty on the state to treat and rehabilitate is unsourced here, as are the **Supreme Court rulings on Section 309 and Article 21** that examiners pair with this question. Read the judgments before quoting them.

Durkheim, in the two-axis form. Suicide rates depend on social integration, the strength of social ties, and social regulation, the degree of normative control over desires, which yields four types. *Egoistic* suicide follows deficient integration: detachment, alienation, severed social connection. *Altruistic* suicide follows excessive integration: life sacrificed for a collective good or group code.

Anomic suicide follows deficient regulation: sudden economic, agrarian or social upheaval leaving expectations unmatched by reality. *Fatalistic* suicide follows excessive regulation: oppressive rules, extreme discipline and powerlessness, with suicide perceived as the only escape.

MODEL	CORE PROPOSITION	THE DISCRIMINATOR
Shneidman, psychache	Suicide is a fatal response to unendurable psychological pain arising from thwarted vital needs such as autonomy, love, affiliation and positive self-regard	**The act is an escape from intolerable affect when cognitive constriction removes alternatives**
Joiner, interpersonal theory	Suicidal desire requires perceived burdensomeness plus thwarted belongingness	**Lethal behaviour needs a third element, acquired capability, through habituation to pain and reduced fear of death**
O'Connor, integrated motivational-volitional model	Pre-motivational background vulnerability, then a motivational phase of defeat and humiliation with entrapment generating ideation, then a volitional phase	**The volitional moderators, access to means, acquired capability, planning and impulsivity, govern the move from ideation to act**
Beck, cognitive model	The negative cognitive triad of self, world and future producing hopelessness, helplessness and worthlessness	**Hopelessness is the primary cognitive mediator of risk**

RULE Do not import the Western epidemiological profile. In India two thirds of suicides fall in the young adult bracket, suicide is the leading cause of death in the 15 to 39 age group, and among minors female deaths exceed male. **Anomic is deficient regulation, egoistic is deficient integration**, and the agrarian crisis case is anomic, not egoistic.

HOLD THIS

Rate 12.3 per 100,000 and 171,418 deaths; hanging 60.9 per cent, poisoning 25.0 per cent; two thirds aged 18 to 45 and two thirds earning under a lakh. Section 115 presumes severe stress and bars trial.

WHAT EARNS THE MARKS

- **Rate with its denominator**: the figure and the population it belongs to, together
- **The Indian age profile**: two thirds in young adulthood, not the Western pattern
- **Section 309 remains in the Code**: what changed is the presumption of severe stress
- **The undercount stated**: naming the limitation is part of the answer

SEE ALSO 08 Insight and the obsession boundary 09 Culture-bound syndromes and the cultural formulation 23 Reading the report they hand you

EEG

The examiner puts a tracing in front of you. The mark is not in the diagnosis; it is in the order of description, because a candidate who describes the record systematically arrives at the right label and a candidate who guesses the label first cannot defend it.

Say the metadata, the technical parameters, the background, the state changes, the response to activation, then the transients, then the impression.

12 · Describing the record: the normal EEG

EEG · TOPIC 12

Describe this record for me.

INVESTIGATIONS TABLE, EEG STATION

THE SHAPE OF THE ANSWER

1 **The sequence**	Background, then organisation, then abnormality, in order
2 **Electrodes**	The ten-twenty system, bipolar against referential montages
3 **The rhythms**	Posterior dominant alpha, then beta, theta, delta, mu
4 **Activation**	Hyperventilation, photic stimulation, sleep deprivation
5 **Variants and artifacts**	The benign variants, and the misreads to refuse

The description sequence, in the order to say it.

- **First, the metadata.** Age, level of alertness, behavioural state, relevant history, and current medication, specifically benzodiazepines, barbiturates or antiseizure drugs.
- **Then the technical parameters.** Calibration, display speed, conventionally 30 mm per second, amplifier sensitivity, conventionally 7 microvolts per millimetre, bandpass filter settings, conventionally 1 to 70 Hz with a 50 Hz notch filter in Indian and British practice and 60 Hz in North America, the montage in use, and electrode impedances, which should sit between 100 and 5000 ohms.
- **Then the waking background.** Presence, frequency, amplitude, distribution, symmetry and reactivity of the posterior dominant rhythm, and the frontocentral resting frequencies.
- **Then the state transitions and sleep elements.** And after them the response to hyperventilation and photic stimulation.
- **Then the transients.** Artifacts, benign variants, focal or generalised slowing, and interictal epileptiform discharges.

Then a one-line impression classifying the record as normal, abnormal non-epileptiform, whether diffuse encephalopathy or focal disturbance, or abnormal epileptiform.

RULE The polarity convention, which is worth a mark on its own: the amplifier measures input 1 minus input 2, so when **input 1 is more negative than input 2 the deflection is upward**, and when input 1 is more positive the deflection is downward.

Electrodes and montages. The 10-20 system divides the distances between nasion, inion and preauricular points into intervals of 10 per cent and 20 per cent. Letters give the region, odd numbers are left, even right, subscript z midline, and a clinical study needs a minimum of 21 electrodes, with true anterior temporal leads supplementary.

In a *bipolar* montage each channel records the difference between adjacent electrodes, and a focal abnormality is localised by a **phase reversal**, deflections in two adjacent channels sharing an electrode pointing toward each other. In a *referential* montage each channel pairs an active electrode with a neutral reference, and localisation is by **maximal amplitude**.

BAND	FREQUENCY	NORMAL OCCURRENCE	ASSOCIATION
Delta	Under 4 Hz	Deep sleep, infancy	**Generalised in encephalopathy, focal over a structural lesion**
Theta	4 to 8 Hz	Drowsiness, children, the elderly	**Cognitive effort, limbic activity, focal pathology**
Alpha	8 to 13 Hz	Relaxed wakefulness with eyes closed	**The posterior dominant rhythm, attenuated by visual alerting**
Beta	Over 13 to 30 Hz	Alert wakefulness, frontocentral	**Benzodiazepines, barbiturates, breach rhythm**
Gamma	Over 30 Hz	Not reliably isolated on scalp recording	**Cortical processing, and muscle contamination**

The posterior dominant rhythm is the landmark of the waking record: sinusoidal, bilateral and symmetric, maximal over parietal and occipital derivations, stable between 8 and 13 Hz in healthy adults, attenuating promptly on eye opening or mental concentration and returning on eye closure.

Interhemispheric frequency asymmetry should stay below 1 Hz; amplitude is often slightly higher over the non-dominant hemisphere, and a persistent voltage asymmetry exceeding 50 per cent is abnormal. The *alpha squeak* is a transient acceleration immediately after eye closure. Harmonically related slow and fast alpha variants share the occipital distribution and reactivity.

In about 25 per cent of normal individuals the rhythm is poorly developed, and fewer than 10 per cent show resting voltages below 15 microvolts. **Bancaud phenomenon**, unilateral failure of the alpha rhythm to attenuate on eye opening, indicates an ipsilateral subcortical or cortical lesion.

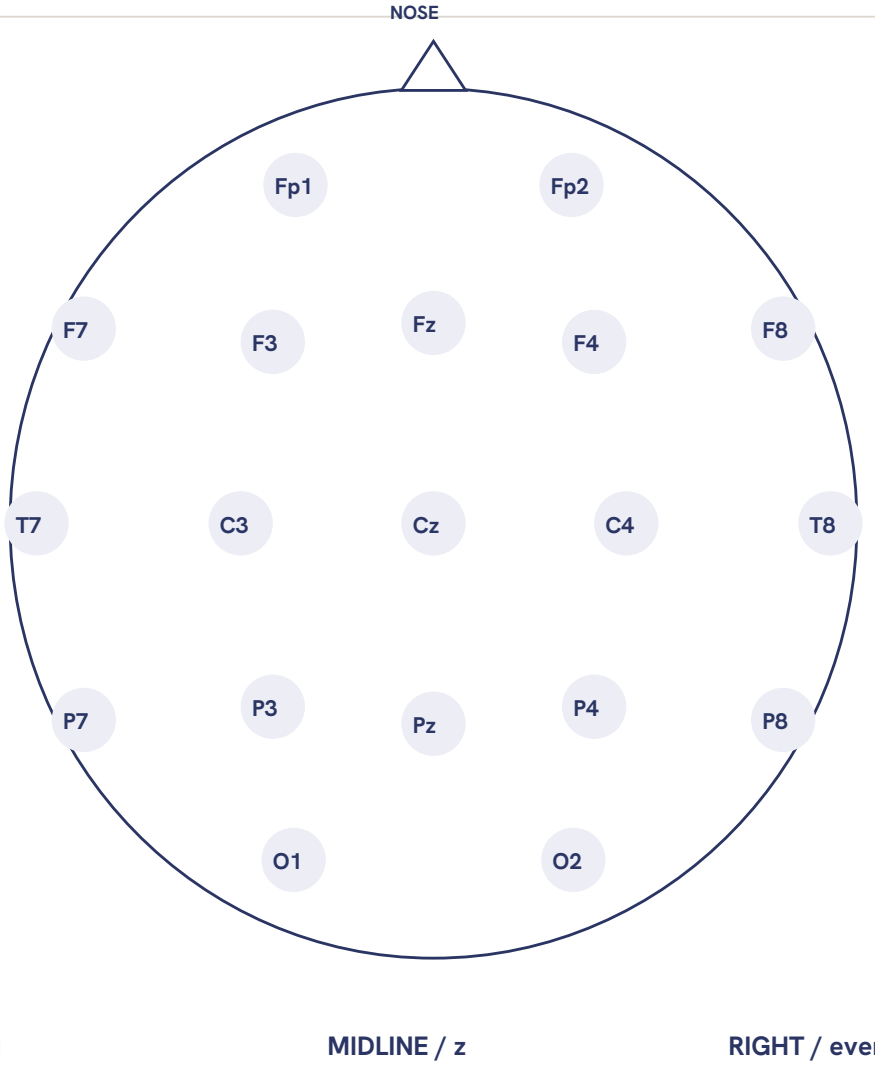
The other waking rhythms. The *mu* rhythm is an arciform 8 to 10 Hz central rhythm of motor cortex at rest: it does not attenuate on eye opening but blocks with contralateral movement, and is frequently asymmetric without pathology.

Normal *beta* is frontocentral at 18 to 25 Hz under 20 microvolts, and widespread 14 to 16 Hz beta above 50 microvolts for more than half the record is characteristically drug-induced. A *breach* rhythm over a craniotomy defect raises amplitude more than threefold with a spiky contour that is not epileptiform without true slowing.

Lambda waves are positive occipital sharp transients of 160 to 250 milliseconds during visual scanning, disappearing on eye closure.

Read the electrode name before the trace.

Conceptual 10–20 scalp layout, viewed from above; nose at top.



Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Schematic 10–20 scalp positions: odd numbers are on the left, even numbers on the right and z denotes the midline. Frontal, central, parietal, occipital and temporal labels organise the recording locations. T7/T8 and P7/P8 correspond to older T3/T4 and T5/T6 labels. This top view is not a scaled placement template; clinical placement requires measurements.

Do not confuse EEG recording electrodes with ECT stimulus electrodes.

Schematic prepared by Luna for Weave Viva, based on P5B-01. Original vector diagram.

STAGE	DEFINING FEATURES ON THE ROUTINE RECORD
N1, drowsiness	**The posterior rhythm fragments and attenuates; slow rolling lateral eye movements appear; frontocentral beta becomes relatively prominent; vertex sharp transients (V-waves), diphasic and about 200 milliseconds, appear near the end of the stage; positive occipital sharp transients of sleep emerge singly or in runs**
N2, light sleep	**Sleep spindles, sinusoidal 12 to 14 Hz bursts (10 to 12 Hz frontally) with a waxing and waning envelope, maximal centrally, and K-complexes, high-voltage diphasic complexes over 200 milliseconds with an initial sharp negative deflection then a slow positive wave, maximal frontocentrally and often triggered by sudden sound. Spindle voltage asymmetry above 50 per cent is abnormal**
N3, slow wave sleep	**High-amplitude delta above 75 microvolts at 1 to 2 Hz occupying more than 20 per cent of the epoch; spindles and positive occipital transients may persist early**
REM	**Low-voltage mixed-frequency background with centrally predominant saw-toothed waves, rapid eye movements and profound muscle atonia. Sleep-onset REM on a daytime routine record more often reflects severe sleep deprivation than narcolepsy**

Activation. Hyperventilation for 2 to 3 minutes produces hypocarbia, cerebral vasoconstriction and alkalosis, and the normal response is symmetrical frontally dominant rhythmic theta and delta buildup resolving within 1 to 2 minutes of stopping.

It triggers 3 Hz generalised spike-and-wave in up to 80 per cent of untreated absence epilepsy and activates discharges in under 10 per cent of focal epilepsies. Contraindications: acute or recent stroke, severe carotid or intracranial stenosis, cardiopulmonary compromise, sickle cell disease or trait, and advanced pregnancy.

Photic stimulation uses a strobe under 30 cm at 1 to 30 Hz in trains of about 10 seconds, and the normal response is photic driving time-locked to the stimulus or its harmonics.

BENIGN VARIANT	WHAT IT LOOKS LIKE	THE DISCRIMINATOR AGAINST AN EPILEPTIFORM DISCHARGE
Rhythmic temporal theta bursts of drowsiness (the old psychomotor variant)	Runs of 5 to 7 Hz rhythmic, notched or flat-topped theta maximal mid-temporally in drowsiness, in 0.5 per cent to 2.0 per cent of normal adults	**No evolution in frequency or field, and no correlation with epilepsy**
Midline vertex theta (Ciganek rhythm)	4 to 7 Hz smooth or arciform theta maximal at the vertex in wakefulness or drowsiness	**Does not block with limb movement, and is unrelated to epilepsy**
6 Hz spike-and-wave (phantom spike-wave)	Bursts of 5 to 7 Hz, characteristically 6 Hz, lasting 1 to 2 seconds	**Two forms: female, occipital, low amplitude, drowsy is benign; wakefulness, high amplitude, anterior, male carries a greater association with seizures**
14 and 6 Hz positive bursts (ctenoids)	Comb-shaped positive transients at 14 Hz or 6 to 7 Hz lasting 0.5 to 1.0 second posterior temporally in light sleep, prominent in adolescents	**Positive polarity, best resolved on referential montages with long interelectrode distances**
Benign epileptiform transients of sleep (small sharp spikes)	Under 50 microvolts and under 50 milliseconds, steep on both slopes, anterior to mid-temporal in N1 and N2	**No background disruption, no significant aftercoming slow wave, an oblique transverse dipole, and they vanish in N3. A true temporal spike exceeds 50 milliseconds, carries a slow wave, and persists or activates in N3**
Wicket spikes	Single waveforms or trains of 6 to 11 Hz arciform waves up to 200 microvolts over temporal regions in adults older than 30 years during drowsiness	**Same frequency as the background rhythm, no focal slow wave, background architecture intact**
Subclinical rhythmic electrographic discharge in adults	Runs of rhythmic, sharply contoured 5 to 7 Hz theta maximal temporoparietally, or progressive vertex sharp sequences, lasting 40 to 80 seconds in people older than 50 years	**It looks like a seizure and is not one: no evolution in frequency or topography, no symptoms, and no post-ictal slowing**

Artifacts, briefly, because a misread artifact is a wrong diagnosis. The eyeball is a dipole, cornea positive to retina: a vertical blink rotates the globes upward and brings the positive cornea near the frontopolar electrodes, and lateral gaze produces out-of-phase anterior temporal deflections. The tongue is also a dipole, so swallowing gives broad delta-like frontotemporal potentials.

Muscle artifact lasts under 20 milliseconds against cerebral spikes above 20, lacks aftercoming slow waves and stops on jaw relaxation. Pulse artifact is time-locked one-to-one to the

electrocardiogram. Impedances above 5000 ohms admit line interference, and an electrode pop is a steep deflection confined to one derivation with no physiological field.

TRAP Three classic misreads. **Lambda waves** are positive, 160 to 250 milliseconds, appear only during saccadic exploration of a textured pattern and vanish on eye closure, whereas occipital spikes are surface-negative, disrupt the background and persist. The **photomyogenic response** is frontalis muscle activity 50 to 60 milliseconds after each flash that stops with the train, whereas a photoparoxysmal response is cortical, generalised and frequently outlasts the stimulus. And the **mu rhythm** is not central epileptiform activity: it fails to attenuate with eye opening and attenuates with contralateral hand movement.

HOLD THIS

Bipolar montages localise by phase reversal, referential montages by maximal amplitude. Input 1 more negative gives an upward deflection, and a spike without an aftercoming slow wave and without background disruption is probably a benign variant.

WHAT EARNS THE MARKS

- **A description before a diagnosis**: the examiner grades the order of your sentences
- **Phase reversal against maximal amplitude**: how each montage localises a discharge
- **Reactivity of the alpha rhythm**: eye opening attenuates it, so say that you checked
- **Background disruption as the test**: a spike without it is probably a benign variant

SEE ALSO 13 The epileptiform record 14 Encephalopathy, the named periodic patterns, and drugs 15 Sleep staging, polysomnography and depression 16 Evoked potentials and the gating measures

13 · The epileptiform record

EEG · TOPIC 13

Is this discharge epileptiform, and what does it point to?

INVESTIGATIONS TABLE, EEG STATION

THE SHAPE OF THE ANSWER

1 **Duration**	Spike under seventy milliseconds, sharp wave seventy to two hundred
2 **The company it keeps**	Aftercoming slow wave, background disruption, field
3 **The absence pattern**	Three per second against slow spike-and-wave
4 **Focal records**	Temporal lobe epilepsy, and secondary bilateral synchrony
5 **Status**	What makes a run a seizure, with the two time points

Duration defines the transient; the company it keeps defines its meaning. Spikes and sharp waves carry identical clinical significance as markers of cortical hyperexcitability, and are separated only by duration: a **spike** lasts under 70 milliseconds, a **sharp wave** between 70 and 200 milliseconds.

Three criteria separate a true epileptiform transient from a benign variant, a sleep transient or an artifact.

- **Amplitude.** High amplitude standing out from the background.

- **Background disruption.** Disruption of the background with an aftergoing slow wave.
- **Field.** A physiological potential field across adjacent electrodes with clear voltage gradients or phase reversals, which a single-electrode mechanical artifact cannot produce.

In genetic or idiopathic generalised epilepsies spikes are generalised with maximal frontal voltage, most often phase-reversing at F3 and F4.

FEATURE	CLASSICAL 3 HZ SPIKE-AND-WAVE	SLOW SPIKE-AND-WAVE
Frequency	**2.5 to 4 Hz, classically a regular 3 Hz**	**Slower than 2.5 Hz**
Morphology	Monomorphic and highly regular	Irregular and polymorphic
Bursts	Discrete, well demarcated, abrupt onset and sudden offset	Less discrete, prolonged, ill defined
Interictal background	**Completely normal between bursts, with no post-ictal slowing**	**Abnormal, with diffuse slowing and multifocal spikes**
Syndrome	Idiopathic or genetic generalised epilepsy, childhood absence epilepsy	Lennox-Gastaut syndrome, typically presenting between 1 and 8 years with intellectual disability and multiple seizure types
Seizure correlate	Typical absence	Atypical absence, tonic, atonic, drop attacks

A run of 3 seconds or more is required before a record is described as showing spike-wave complexes rather than repetitive spikes. At onset the discharge is often faster, 4 to 5 Hz, then stabilises at 3 Hz and may decelerate to 2 Hz or 1.5 Hz before abrupt termination; bursts are often under 10 seconds. In absence beginning in adolescence the discharges are faster and irregular, 3 to 5.5 Hz spike-wave and polyspike-wave.

PATTERN	DEFINITION	CLINICAL CORRELATE
Polyspikes	Multiple repetitive spikes grouped together, discharging at about 20 Hz with an aftergoing slow wave	**Juvenile myoclonic epilepsy; the electrographic hallmark of myoclonic seizures, where a single jerk time-locked to a brief discharge counts as an electroclinical seizure with consciousness preserved**
Hypsarrhythmia	Four features together: continuous in wakefulness, amplitude above 200 microvolts, generalised polymorphic slowing, and complete absence of organised background with chaotic multifocal spikes and sharp waves	**West syndrome, or infantile epileptic spasms syndrome: the triad of spasms, hypsarrhythmia and developmental delay or regression, with peak onset between 3 and 7 months**
Generalised paroxysmal fast activity	Paroxysmal bursts of generalised fast rhythms above 10 Hz, most prominent in non-REM sleep	**Tonic seizures in Lennox-Gastaut syndrome; meets criteria for brief ictal rhythmic discharges under the 2020 standardised terminology**
Electrodecrement	Abrupt marked attenuation or flattening of voltage across all channels, often preceded by a high-amplitude sharp transient or slow wave	**Infantile spasms and epileptic drop attacks, although it can occur as an asymptomatic interictal discharge**
Lateralised periodic discharges (the old PLEDs)	Focal periodic patterns lateralised to one hemisphere, often spikes or sharp waves recurring every 1 to 2 seconds	**Acute destructive cortical processes: stroke, viral encephalitis, hypoxic injury. They sit close to the ictal end of the continuum and are strongly associated with seizures, frequently focal motor**

Temporal lobe epilepsy. The temporal lobe is the most epileptogenic region, and 90 per cent of individuals with temporal interictal epileptiform abnormalities have a seizure history.

Interictally: anterior temporal sharp waves and spikes with maximal phase reversals at F7 or T3 and F8 or T4, activating in sleep, and temporal intermittent rhythmic delta activity around 1 Hz as an interictal marker of epileptogenicity.

Ictally: localised rhythmic delta or theta over the anterior temporal region rising in amplitude, reorganising into rhythmic sharp activity at 2.5 to 3 Hz, spreading regionally, then slowing before offset. Mesial onset accounts for almost 80 per cent of temporal seizures and may show only subtle herald slowing on the scalp, which is why video monitoring is needed.

Hippocampal sclerosis is the substrate in about two thirds of operated cases, with cell loss in CA1, CA3 and the dentate hilus and sparing of CA2; drug resistance is defined after 2 failed adequate trials, and lobectomy in selected refractory mesial cases gives seizure freedom in 70 to 80 per cent.

Secondary bilateral synchrony is the trap built into the 3 Hz pattern: a focal cortical discharge propagates rapidly to engage bilateral cortico-thalamic circuits, producing generalised synchronous spike-wave that mimics primary generalised epilepsy. The tell is a focal herald spike or asymmetric onset in the earliest fractions of the burst, or persistent focal interictal spikes.

It matters because the misreading leads to the wrong drug, and resecting the focal lesion abolishes both the focal spikes and the secondarily generalised discharges.

SEIZURE TYPE	TIME T1, BEGIN TREATMENT	TIME T2, INJURY BEGINS
Bilateral convulsive (tonic-clonic)	5 minutes	30 minutes
Focal with impaired consciousness	10 minutes	Greater than 60 minutes
Typical absence	10 to 15 minutes	Not established in the sources

Status epilepticus occurs when endogenous termination mechanisms fail. Time t1 is the point beyond which spontaneous termination is unlikely and emergent therapy must begin; time t2 is the point beyond which neuronal injury, altered receptor networks and long-term deficits occur. For tonic, myoclonic and spasm types the sources establish neither threshold.

What makes a run a seizure. Electrographically a seizure must evolve in frequency, accelerating or decelerating, in morphology, or in spatial distribution; fluctuation in amplitude alone is not evolution, which is why frequent interictal spikes and runs of rhythmic slowing are not seizures.

Discharges must persist for more than 10 seconds unless there is a definite time-locked clinical correlate such as a brief absence or a myoclonic jerk, and periodic patterns exceeding 2.5 Hz that continue for 10 seconds or more are also classified as seizures.

Non-convulsive status is defined on continuous recording as electrographic seizure activity continuing for 10 or more continuous minutes, or occupying at least 20 per cent of any 60-minute epoch. Post-anoxic myoclonic status is commonly diagnosed when repetitive jerks with time-locked epileptiform activity persist beyond 30 minutes on a severely suppressed background.

TRAP Reading eye flutter as frontal spike-and-wave. Rapid rhythmic bifrontal oscillations from flutter stay **strictly confined to the frontopolar leads** and lack an aftergoing slow wave and any field extension into the parasagittal chains. The same discipline applies to vertex sharp waves, positive occipital transients and rhythmic head-scratching artifact.

HOLD THIS

Spike under 70 milliseconds, sharp wave 70 to 200; regular 3 Hz on a normal background is typical absence, under 2.5 Hz on an abnormal background is not. Evolution, not fluctuation, makes a seizure.

WHAT EARNS THE MARKS

- **Duration measured first**: the measurement defines the transient before you interpret it
- **Evolution, not fluctuation**: the test that turns a run into a seizure
- **Background as well as frequency**: three per second means little without the background
- **Eye flutter refused**: its distribution and its missing slow wave give it away

SEE ALSO 12 Describing the record: the normal EEG 14 Encephalopathy, the named periodic patterns, and drugs 15 Sleep staging, polysomnography and depression

14 · Encephalopathy, the named periodic patterns, and drugs

EEG · TOPIC 14

This record shows generalised slowing. How would you grade it, and what would you look for?

INVESTIGATIONS TABLE, EEG STATION

THE SHAPE OF THE ANSWER

1 Grading	Grade the slowing before you name any cause
2 The periodic patterns	Definitions, then the periodicity that separates them
3 Prion against herpes	Where each discharge sits, with the figures
4 Coma patterns	Frequency, voltage, reactivity, and the cause each suggests
5 Drugs	What the common psychotropics do to the record

Diffuse means diffuse: grade the slowing before you name a cause. Focal slowing indicates localised pathology, and slowly progressive dementias such as Alzheimer disease keep a preserved record until late. Grade in three steps.

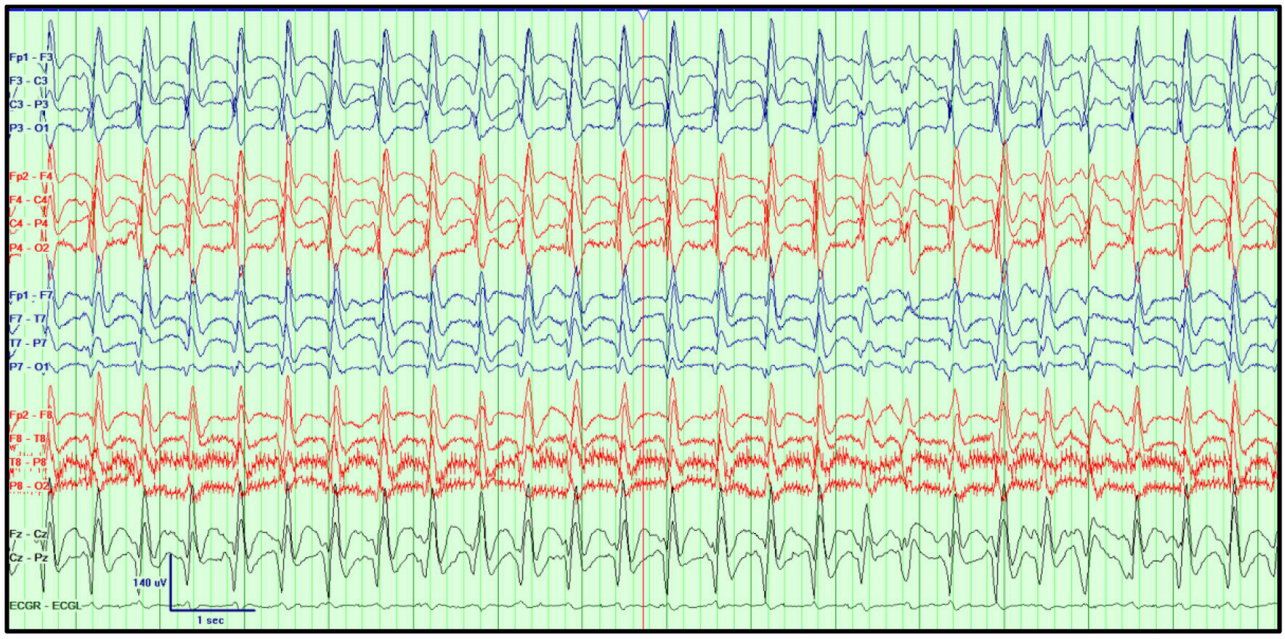
Background slowing, mildest, keeps a reactive posterior rhythm at a frequency subnormal for age, the lower limit following the 5-6-7-8 rule, 5 Hz at age 1, 6 at 3, 7 at 5 and 8 from age 8, so a 7 Hz rhythm in an awake adult is the mildest stage of diffuse encephalopathy.

Intermittent slowing, moderate, gives generalised bursts of polymorphic delta, rhythmic frontally as frontal intermittent rhythmic delta activity, with reactivity and state transitions retained. *Continuous slowing*, severe, is polymorphic delta over more than 80 per cent of the record, usually unreactive with the background absent, most often toxic, metabolic or systemic.

PATTERN	DEFINITION	THE DISCRIMINATOR
Triphasic waves	Frontally dominant positive sharp transients above 70 microvolts, a prominent positive phase flanked by smaller negative deflections, the first larger than the last, bilateral, synchronous, repeating rhythmically or pseudo-rhythmically at 1 to 3 Hz	**An anterior-to-posterior time lag on longitudinal bipolar montages, usually absent on ear-referential montages, and typically unreactive. Classically hepatic, but etiologically non-specific: uraemic and other toxic-metabolic states and non-convulsive status all produce them**
Periodic sharp wave complexes of Creutzfeldt-Jakob disease	Bilateral synchronous periodic complexes recurring at a periodicity of about 1 to 2 seconds	**The periodicity is the answer: about 1 to 2 seconds here against about 4 to 10 seconds in subacute sclerosing panencephalitis. No anterior-to-posterior lag, unlike triphasic waves**
Extreme delta brush	Continuous or semi-rhythmic high-amplitude delta at 1 to 3 Hz with superimposed rhythmic beta bursts at about 20 to 30 Hz riding synchronously on the delta	**Specific for anti-NMDA receptor encephalitis and a marker of severity, but absent in early illness, so its absence does not exclude the diagnosis**
Burst-suppression	Periodic bursts of high-voltage mixed sharp and slow waves lasting 1 to 3 seconds separated by generalised suppression below 10 microvolts lasting about 5 to 10 seconds	**Identical morphology in two settings: reversible with drug elimination after propofol, barbiturate or benzodiazepine loading, irreversible after prolonged cerebral anoxia**
Electrocerebral inactivity	No cerebral electrical activity exceeding 2 microvolts, recorded at a sensitivity of 2 microvolts per millimetre with verification of system integrity and absent reactivity	**Supportive of brain death, which remains a clinical diagnosis: the record is confirmatory and not mandatory**

Triphasic morphology with a slow background

Companion to original page 414



EEG in a bipolar montage showing generalised periodic discharges with triphasic morphology on diffuse irregular delta–theta activity. The source toxic-metabolic case had kidney disease, sepsis and cefepime exposure. Triphasic morphology is not specific to hepatic encephalopathy or any single metabolic cause. Interpret the periodic pattern with the background, medication history and clinical state.

Distinguish periodic complexes from the slow background and avoid treating triphasic morphology as a cause-specific diagnosis.

Sonali Sharma, Michelle Nunes and Ayham Alkhachroum. Frontiers 951286 Figure 6. CC BY 4.0. https://www.frontiersin.org/journals/neurology/articles/10.3389/fneur.2022.951286/full#F6 Licence: https://creativecommons.org/licenses/by/4.0/ Unmodified.

COMA PATTERN	FREQUENCY	VOLTAGE AND REACTIVITY	CAUSE
Alpha coma	8 to 12 Hz	Continuous, unreactive, diffuse or anteriorly dominant rather than posterior	**Severe anoxic-ischaemic encephalopathy after cardiorespiratory arrest; poor prognosis when unreactive**
Beta coma	15 to 22 Hz	Above 30 microvolts, continuous, unreactive	**Acute sedative-hypnotic intoxication**
Spindle coma	13 to 16 Hz	Monotonous, widespread, unreactive, resembling sleep spindles	**High brainstem or diencephalic lesion with relative cortical sparing**

Creutzfeldt-Jakob disease, the numbers. Periodic sharp wave complexes occur in about two thirds of sporadic cases with a positive predictive value of 95 per cent, and in a series of 27 autopsy-confirmed cases 94 per cent showed periodic patterns on serial recordings.

The evolution: early non-specific diffuse slowing, frontal intermittent rhythmic delta or focal slowing with lateralised periodic discharges in half of cases; then bilateral synchronous periodic complexes; terminally areactive coma patterns, burst-suppression or alpha coma. They attenuate in sleep, can be masked by sedatives, and may disappear terminally, and seizures are uncommon, in under 15 per cent.

They are prominent in the classic MM1 and MV1 subtypes, 60 per cent to 70 per cent of sporadic disease with a mean duration of about 4 to 6 months, rare in valine homozygosity, present in about 10 per cent of genetic disease and absent in the variant form.

Herpes simplex encephalitis against prion disease. Herpes encephalitis produces focal, unilateral or bilateral independent periodic lateralised discharges over temporal and frontotemporal regions, emerging acutely and resolving within days to weeks.

In an emergent series of 1948 records such discharges appeared in 79 patients, 4 per cent, with central nervous system infection and inflammation 35.4 per cent, stroke 32.9 per cent and metabolic encephalopathy 11.4 per cent.

The separation from prion disease rests on fluid and imaging, not the tracing: herpes gives lymphocytic pleocytosis with raised protein and asymmetric temporal oedema, while routine fluid in prion disease is normal and the scan shows cortical ribboning with basal ganglia diffusion restriction. In prion disease lateralised discharges persist and convert into generalised periodic complexes rather than resolving.

RULE An unresponsive or severely agitated patient whose record shows **generalised polymorphic delta slowing or periodic discharges has an organic encephalopathy, non-convulsive status or drug toxicity until proved otherwise**. Uncomplicated psychiatric catatonia or acute functional psychosis keeps a preserved, reactive posterior alpha rhythm with intact sleep architecture.

TRAP Triphasic waves against non-convulsive status. Triphasic morphology is necessary and not sufficient. Look for spatial, morphological and frequency **evolution**, which status shows and metabolic triphasic waves do not; for the response to an intravenous short-acting benzodiazepine, which resolves electrographic seizure activity and may clear the sensorium in status while merely suppressing voltage or deepening slowing in metabolic encephalopathy; and for the clinical fragments, rhythmic eyelid myoclonus or nystagmoid jerks favouring status, asterixis favouring hepatic or uraemic failure.

Drug effects. Sedatives and hypnotics enhance diffuse beta at 15 to 22 Hz at therapeutic doses and produce beta coma above 30 microvolts, then burst-suppression, at toxic doses.

Clozapine causes dose-dependent background slowing with theta and delta and can induce paroxysmal sharp waves, spike-wave complexes and polyspikes, correlating with rapid titration and higher plasma levels, and dose reduction with an antiepileptic is the standard response to myoclonus or seizures.

Lithium slows the background, and in toxicity gives severe slowing, frontal intermittent rhythmic delta and triphasic-like sharp waves mimicking metabolic encephalopathy or prion disease. First-generation antipsychotics and tricyclics slow the alpha background and lower the seizure threshold. The clozapine and lithium specifics are carried as unverified notebook material, so give the direction and not a number.

HOLD THIS

Periodicity separates the periodic patterns: about 1 to 2 seconds in prion disease, about 4 to 10 seconds in subacute sclerosing panencephalitis. A 7 Hz posterior rhythm in an adult is mild encephalopathy, not a normal variant.

WHAT EARNS THE MARKS

- **Grading before naming**: the examiner is testing sequence, not vocabulary
- **Periodicity as the discriminator**: one to two seconds against four to ten
- **Triphasic morphology is not sufficient**: look for evolution and for reactivity
- **A slow posterior rhythm in an adult**: mild encephalopathy, not a normal variant

SEE ALSO 06 Consciousness, attention and memory 12 Describing the record: the normal EEG 13 The epileptiform record 24 Cerebrospinal fluid, neuroinfection and neurosyphilis

15 · Sleep staging, polysomnography and depression

EEG · TOPIC 15

Take me through polysomnography, and tell me what it shows in depression.

INVESTIGATIONS TABLE, EEG STATION

THE SHAPE OF THE ANSWER

1 **The signals**	The three that define a study, named before anything else
2 **Staging**	The older system against the one in current use
3 **Normative numbers**	Latency, cycle length, stage proportions
4 **Depression**	Continuity, architecture, and the two REM measures
5 **Respiratory**	The indices, and where they mislead you

Name the three signals, then the staging system, then the numbers. Diagnostic polysomnography integrates electroencephalography, electro-oculography and surface electromyography.

Electrodes follow the 10-20 system, with recommended derivations from frontal, central and occipital sites referenced to the contralateral mastoid: frontal leads register slow-wave delta, central leads capture vertex sharp waves and spindles, and occipital leads identify the posterior dominant alpha rhythm.

Three submental chin electrodes are placed, 2 active and 1 backup, and chin tone falls progressively across deeper non-REM sleep to its nadir in REM.

Eye movement is detected through the permanent corneoretinal dipole, cornea electropositive to retina, with one electrode 1 cm below the left outer canthus and a second 1 cm above the right outer canthus, so conjugate movements produce out-of-phase deflections.

PARAMETER	RECHTSCHAFFEN AND KALES, 1968	AMERICAN ACADEMY OF SLEEP MEDICINE, 2007 ONWARD
Stage count	**5 stages: 1, 2, 3, 4 and REM**	**4 stages: N1, N2, N3 and stage R**
Slow-wave sleep	Split: stage 3 with 20 to 50 per cent delta, stage 4 with more than 50 per cent	Combined into N3 when slow waves occupy at least 20 per cent of the epoch
Spindle frequency	Classically 12 to 14 Hz	Broadened to 11 to 16 Hz bursts
Epoch duration	30 seconds	30 seconds

The reason for the merge, which is the follow-up question: stages 3 and 4 showed identical restorative physiology, homeostatic regulation and clinical implications, so no clinical, metabolic or staging distinction justified keeping them apart.

Scoring, in one paragraph. Each 30-second epoch takes the stage occupying more than half of it.

Stage W needs alpha at 8 to 12 Hz over more than half the epoch posteriorly with eyes closed, or beta with eyes open, plus blinks at 0.5 to 2 Hz and high chin tone.

Up to 20 per cent of healthy people generate no occipital alpha. In them wakefulness is scored on irregular conjugate eye movements, blinks and maintained tone, and N1 on vertex waves, slow eye movements or background slowing of at least 1 Hz from wakefulness.

N2 needs at least one K-complex unassociated with arousal or one train of spindles in the first half of the epoch, spindles being 11 to 16 Hz bursts of at least 0.5 seconds maximal centrally and K-complexes diphasic waveforms of at least 0.5 seconds maximal frontally.

N3 needs slow waves of 0.5 to 2 Hz at 75 microvolts or more over at least 20 per cent of the epoch. Stage R needs three things together: a low-amplitude mixed-frequency background, the lowest chin tone of the night, and rapid eye movements under 500 milliseconds, with sawtooth waves as support.

PARAMETER	NORMATIVE RANGE IN HEALTHY ADULTS
Sleep efficiency	**Total sleep time over time in bed, normally 80 per cent or more, typically 80 to 86 per cent**
Sleep onset latency	**Normally under 30 minutes from lights out to the first sleep epoch**
REM latency	**Sleep onset to the first epoch of stage R, normally 70 to 90 minutes, quoted also as 60 to 120 minutes**
Cycle duration and number	**About 90 minutes, the first cycle shortest at 70 to 100 minutes and later cycles 90 to 120, across 4 to 6 cycles a night**
Stage proportions	**N1 2 to 5 per cent, N2 45 to 55 per cent, N3 15 to 20 per cent, stage R 20 to 25 per cent of total sleep time**
Arousal index	**An arousal is a shift toward alpha, theta or faster than 16 Hz activity, excluding spindles, lasting at least 3 seconds and preceded by at least 10 seconds of stable sleep; a normal index in young adults is under about 10 to 25 events per hour**
Periodic limb movement index	**Normally under 15 events per hour in adults; 15 or more is abnormal**

Architecture across the night: N3 predominates in the first third, coinciding with maximal pulsatile growth hormone secretion, while REM episodes are brief early, often under 5 to 10 minutes, and lengthen to 20 to 60 minutes in the final third. Scoring an arousal in stage R additionally requires a concurrent rise in chin tone lasting at least 1 second.

FINDING IN MAJOR DEPRESSIVE DISORDER	WHAT IT CONSISTS OF	STATE OR TRAIT
Impaired continuity	Prolonged sleep onset latency, frequent awakenings, raised wake after sleep onset, reduced efficiency	**State: sleep disturbance is reported in about 80 to 85 per cent of patients**
Slow-wave sleep deficit	Reduced N3 percentage and duration with irregular temporal distribution of slow-wave activity and reduced delta power	**State, and the reason the first cycle looks wrong**
Shortened REM latency	Interval from sleep onset to the first REM period significantly reduced, frequently under 60 minutes against the normal 70 to 90	**Trait: it persists in clinical remission and is documented in unaffected first-degree relatives, making it a candidate endophenotype**
Altered REM distribution and density	REM redistributed into the first third of the night with an elongated first REM period, and increased REM density, more eye movements per unit of REM sleep	**Density is a state marker: it normalises with remission, which is what separates it from latency**

RULE Shortened REM latency is shared, so use the route into sleep. **Depression enters sleep through non-REM** with a prolonged onset latency and then an early REM period. **Narcolepsy enters sleep through REM**, with a sleep-onset REM period conventionally defined as REM within 15 minutes of sleep onset, and the diagnostic multiple sleep latency test uses 4 to 5 nap opportunities at 2-hour intervals showing a mean sleep latency of 8 minutes or less with 2 or more sleep-onset REM periods, one of which may come from the preceding overnight study.

RESPIRATORY MEASURE	DEFINITION AND THRESHOLD
Apnoea	**Complete cessation of airflow for at least 10 seconds**
Hypopnoea	**Airflow reduction of at least 30 per cent from baseline for at least 10 seconds with either a 3 per cent desaturation or a cortical arousal, or alternatively a 4 per cent desaturation under the stricter rule**
Respiratory effort-related arousal	**Progressive flattening of inspiratory nasal pressure with rising effort ending in an arousal, without meeting apnoea or hypopnoea criteria and without requiring desaturation**
Apnoea-hypopnoea index	**Apnoeas plus hypopnoeas per hour of sleep: normal under 5, mild 5 to 15, moderate 15 to 30, severe above 30**
Respiratory disturbance index	**Apnoeas plus hypopnoeas plus effort-related arousals per hour, which is what detects arousal-driven fragmentation in a thin young patient with unrefreshing sleep and no substantial desaturation**

TRAP Two scoring errors and one clinical one. A **K-complex followed immediately by an arousal** cannot be used to continue scoring N2. Stage 4 **does not exist** in current guidelines. And relying on the apnoea-hypopnoea index alone in a young patient with chronic fatigue misses effort-related arousal, which needs the full disturbance index from an in-laboratory study.

HOLD THIS

REM density is the state marker, REM latency the trait marker. Depression comes to REM late through non-REM; narcolepsy arrives at REM within fifteen minutes.

WHAT EARNS THE MARKS

- **Three signals named**: electroencephalography, eye movement, chin muscle activity
- **State against trait**: REM density against REM latency
- **The route into sleep**: depression enters through non-REM, narcolepsy through REM
- **Stage four is gone**: the current guidelines keep four stages, not five

SEE ALSO 12 Describing the record: the normal EEG 14 Encephalopathy, the named periodic patterns, and drugs 16 Evoked potentials and the gating measures 25 Neuroendocrine tests and the status of biomarkers

16 · Evoked potentials and the gating measures

EEG · TOPIC 16

What is the P300, and what does it tell you in schizophrenia?

INVESTIGATIONS TABLE, EEG STATION

THE SHAPE OF THE ANSWER

1 **Definition**	A time-locked averaged response, with latency and amplitude defined
2 **The P300**	Where it is generated, and what each measure indexes
3 **Endophenotype**	Why the amplitude reduction qualifies as one
4 **Mismatch negativity**	Pre-attentive, and what it tracks over time
5 **Gating**	P50 suppression against prepulse inhibition

Define the potential, then say what the amplitude and the latency each mean. An event-related potential is a voltage change time-locked to a sensory, motor or cognitive event, extracted from the ongoing record by signal averaging, which cancels random background activity; waveforms are named by polarity, P for positive and N for negative, and by nominal post-stimulus latency in milliseconds.

Exogenous or sensory potentials occur early, typically under 100 milliseconds, and reflect obligate sensory pathway registration and thalamocortical transmission, for instance the auditory brainstem responses and the P50 wave. Endogenous or cognitive potentials occur later, typically beyond 100 milliseconds, and reflect cognitive operations, attentional allocation, stimulus evaluation and response preparation, for instance N100, mismatch negativity and the P300.

The P300 is a positive deflection over central and parietal sites occurring about 300 milliseconds after stimulus onset, analysed in a window of about 280 to 400 milliseconds, elicited by an auditory oddball paradigm in which infrequent target tones are interspersed among frequent standard tones and the subject discriminates them by pressing a button or counting mentally.

Its amplitude, measured in microvolts, reflects allocation of focal attentional resources and working memory context updating when a task-relevant event occurs. Its latency indexes cognitive processing speed, the time to evaluate and classify the stimulus, independently of motor reaction time.

MEASURE	SCHIZOPHRENIA	BIPOLAR AFFECTIVE DISORDER
P300 latency	Significantly prolonged, pooled effect size 0.57	**Also significantly prolonged over central and temporal leads**
P300 amplitude	**Markedly reduced at midline and left temporal leads, pooled effect size 0.85**	**Preserved, not significantly reduced against controls**
Verbal fluency	Significantly impaired, and the impairment correlates with the prolonged latency	**Superior to schizophrenia and comparable to controls**

From a meta-analysis of 46 studies covering 1,443 patients with schizophrenia and 1,251 healthy controls. This is the cleanest electrophysiological dissociation between the two disorders in the viva: both slow the latency, only schizophrenia flattens the amplitude.

Why the amplitude reduction counts as an endophenotype. In Scottish family pedigrees a balanced chromosomal translocation involving chromosomes 1 and 11 segregates with a psychiatric spectrum phenotype spanning schizophrenia, bipolar disorder and recurrent major depression, and its breakpoint disrupts two brain-expressed genes, DISC1 and DISC2.

The translocation yields a logarithm-of-odds score of 3.6 for schizophrenia alone, 4.5 for affective disorders and 7.1 when all diagnoses in the pedigree are combined. Both symptomatic and asymptomatic carriers show P300 amplitude reduction and latency prolongation against non-carriers, so the abnormality cosegregates with genetic vulnerability independently of clinical expression, which is the definition of a trait marker.

FEATURE	P50 SUPPRESSION	PREPULSE INHIBITION
What is measured	**Sensory gating**	**Sensorimotor gating**
Recording	Scalp electroencephalography at the vertex	Facial electromyography over the orbicularis oculi
Stimulus	Paired identical clicks at 89 dB sound pressure level, 1 millisecond, 500 milliseconds apart, against continuous 60 dB background noise, 120 pairs over a 26-minute session	A startling burst of white noise at 115 dB lasting 40 ms, preceded 60 to 100 ms earlier by a weak non-startling prepulse at about 75 dB
Calculation	One minus the ratio of the second to the first response amplitude, times one hundred	One minus the ratio of the prepulse-trial magnitude to the pulse-alone magnitude, times one hundred
Healthy value	Mean suppression 67.8 per cent, standard deviation 27.5, with reference cohorts at 63.6 to 70.1 per cent	Inhibition of about 54.9 per cent, standard deviation 29.1, up to about 65 per cent
Circuit	Thalamic reticular nucleus and primary auditory cortex, modulated by hippocampus and orbitofrontal cortex	Pontine reticular formation modulated by a forebrain cortico-striato-pallido-thalamic loop: medial prefrontal cortex, hippocampus, nucleus accumbens, ventral pallidum, pedunculopontine tegmental nucleus
Pharmacology	Mediated by alpha-7 nicotinic cholinergic receptors and noradrenergic tone; impaired by yohimbine without altering baseline startle or inhibition	Modulated by dopamine, whose agonists disrupt it, by GABA, glutamate with NMDA antagonists disrupting it, acetylcholine, noradrenaline and serotonin

In schizophrenia the second click is not suppressed, giving raised ratios, with a pooled effect size of 1.56 across 20 studies covering 421 patients and 401 controls, while P50 peak latency does not differ from controls.

Prepulse inhibition is likewise diminished, habituation of the startle response over repeated trials is blunted, and the magnitude of the loss correlates with the severity of formal thought disorder.

Crucially, in simultaneous recording the two measures do not correlate significantly across blocks or session averages, although during the early trial block, while active habituation is taking place, a positive correlation appears. They are complementary, not interchangeable.

Mismatch negativity is an early negative deflection to an unannounced deviant stimulus within a train of repetitive standards, representing a pre-attentive, automatic sensory memory comparison that occurs even when the subject is not attending to the auditory stream.

Hierarchical factor analysis of auditory potentials separates the deficits in schizophrenia into two independent dimensions.

- **The early sensory factor.** Comprising N100 and mismatch negativity, indexing primary auditory cortical pathology and correlating clinically with negative symptoms, specifically alogia and formal thought disorder.
- **The late cognitive factor.** Comprising P3a and P3b, indexing higher-order deficits of context evaluation and resource allocation, and correlating with avolition, attentional impairment and delusions.

Beyond schizophrenia. Patients with obsessive-compulsive disorder show attenuated prepulse inhibition and blunted P50 gating, and in discrimination paradigms show preserved perceptual sensitivity with prominent psychomotor slowing, reaction times prolonged by about 20 per cent, read as failure of cognitive gating in hyperactive orbitofrontal-thalamic and orbitofrontal-amygdala-basal ganglia loops that lets intrusive thoughts breach awareness.

In acute mania and severe depression P50 suppression is diminished and the P300 altered, but there the gating deficit behaves as a state marker that normalises with recovery, whereas in schizophrenia it is a stable trait. Attenuated prepulse inhibition is also documented in Huntington disease, in schizotypal personality disorder and in unaffected first-degree relatives of people with schizophrenia.

On treatment, first-generation dopamine antagonists typically fail to reverse P50 gating deficits despite relieving positive symptoms, while clozapine has been shown to normalise P50 suppression and, in preclinical startle paradigms, to reverse gating disruption induced by non-competitive NMDA antagonists.

Given the dependence of gating on alpha-7 nicotinic receptors, heavy smoking in schizophrenia has been read as an attempt at self-medication of the sensory gating defect.

TRAP Four conflations to avoid. P50 suppression is **not** prepulse inhibition: one is an electroencephalographic measure of sensory gating from paired identical clicks, the other an electromyographic measure of sensorimotor gating during acoustic startle, and they do not correlate across a session. P300 amplitude is **not** equally reduced in bipolar disorder. P50 gating failure is **not** pathognomonic, occurring in schizotypal personality disorder, relatives, obsessive-compulsive disorder and acute mood episodes, with temporal stability the discriminator. And antipsychotics do **not** normalise all of these waveforms uniformly.

HOLD THIS

Both disorders prolong the P300 latency; only schizophrenia reduces the amplitude. Early sensory potentials map to alogia and thought disorder, late cognitive potentials to delusions and avolition.

WHAT EARNS THE MARKS

- **Latency against amplitude**: speed of processing against resource allocation
- **Shared latency, specific amplitude**: both disorders slow it, one of them flattens it
- **Two gating measures, two methods**: scalp electroencephalography against facial electromyography
- **The conflation refused**: the two measures do not correlate, so keep them apart

SEE ALSO 12 Describing the record: the normal EEG 15 Sleep staging, polysomnography and depression 19 Imaging in the primary psychiatric disorders

Neuroimaging

Films are handed across in a fixed order: name the modality, name the plane, give your proof, then describe systematically, then commit to a finding. The vocabulary is part of the mark, because attenuation belongs to one modality and signal intensity to the other, and mixing them tells the examiner you are guessing.

17 · Naming the modality: CT, MRI, windows and sequences

NEUROIMAGING · TOPIC 17

What is this scan, and how do you know?

INVESTIGATIONS TABLE, IMAGING SPOTTER

THE SHAPE OF THE ANSWER

1	First sentence	Modality, plane, and the proof for each of them
2	The comparison	Physics, resolution, contrast, safety, on one grid
3	Windows	Width and level, and what each window is for
4	Sequences	T1, T2, FLAIR, diffusion, and the fluid test
5	Ordering	Which to request in practice, and the contraindications

First sentence: modality, plane, proof. Computed tomography is recognised by dense bright cortical bone forming the calvarium on standard brain and bone windows, with air in the paranasal sinuses and mastoid air cells completely black; lesions are described as hypodense, isodense or hyperdense, that is by **attenuation** or density.

Magnetic resonance imaging shows the cortical bone of the vault as a dark signal void while the diploic space, containing marrow fat, is bright on sequences without fat suppression; lesions are described as hypointense, isointense or hyperintense, that is by **signal intensity**, and stated relative to a named reference tissue.

Axial slices run parallel to the orbitomeatal line and show the hemispheres symmetrically; coronal slices are viewed from the front and show vertex to skull base with the lateral temporal lobes; sagittal slices show the midline structures, corpus callosum, brainstem, cerebellar vermis and fourth ventricle. Volumetric acquisition allows reformatting in all three planes.

PARAMETER	COMPUTED TOMOGRAPHY	MAGNETIC RESONANCE IMAGING
Energy source	Ionising radiation, X-rays	A strong static magnetic field, typically 1.5 or 3 Tesla, with radiofrequency pulses
Mechanism	A rotating tube and detector array measure differential tissue X-ray attenuation	Protons align in the field, radiofrequency excitation tips the net magnetisation, and relaxation releases signals detected by receiver coils and spatially encoded by gradients
Tissue property measured	Electron density and physical radiodensity	Proton density and the longitudinal and transverse relaxation times of hydrogen protons
Spatial resolution	**High: excellent bony margins and anatomical edges**	**Moderate against thin-slice bone imaging**
Contrast resolution	**Lower soft tissue contrast**	**Superior: separates grey matter, white matter and infiltrative pathology**
Acquisition time	1 minute or less	30 minutes to 1 hour
Motion sensitivity	Low, because speed minimises blur	High: movement causes severe distortion and phase artifact

Reading the plain head scan. Work from the scout view down.

- **Scout radiograph.** Skull dysmorphism, gross fracture, radiopaque foreign bodies, previous clips and burr holes.
- **Brain parenchyma, from the vertex downward.** Symmetry, gyral architecture, focal hypoattenuation of infarction or oedema and hyperattenuation of acute haematoma or calcification.
- **Ventricles, then the basal cisterns.** Size, configuration and symmetry appropriate to age, then the perimesencephalic, suprasellar, quadrigeminal and prepontine cisterns, whose effacement indicates raised pressure or impending herniation.
- **The dependent recesses.** Checking the interpeduncular fossa and the occipital horns for small layering hyperdense subarachnoid blood, and the major vessels for the hyperdense middle cerebral artery sign or basilar tip hyperdensity.
- **Grey-white differentiation on a narrow window.** Verifying the cortex against subcortical white matter, and the demarcation of caudate, lentiform nuclei and internal capsule.
- **Early insular ribbon loss.** Loss of the insular ribbon and lentiform obscuration indicate acute cytotoxic oedema in middle cerebral artery territory.
- **The extra-axial spaces on a dedicated window.** Inspecting the inner table, the falx and the tentorium.
- **The extracranial soft tissues, then bone.** Facial planes, globes, retrobulbar fat and scalp.

WINDOW	WIDTH AND LEVEL	WHAT IT IS FOR
Standard brain	Width 80, level 40	Parenchyma, ventricles, sulcal spaces, mass lesions
Stroke or grey-white	Width 8, level 32, or width 40 and level 40	**Insular ribbon loss, basal ganglia blurring, early ischaemic oedema**
Subdural or blood	Width 130 to 300, level 50 to 100	**Separating a hyperdense subdural collection from dense calvarial bone**
Soft tissue	Width 350 to 400, level 20 to 60	Orbits, globes, scalp haematoma, pharynx, paranasal soft tissue
Bone	Width 2800, level 600	Vault fractures, skull base erosion, temporomandibular alignment

Window width sets the range of attenuation values displayed and window level the central midpoint luminance, which is why a window is quoted as a pair.

ASK AND VERIFY The examiner's next question is usually the **Hounsfield anchor values** for air, water, dense cortical bone and acute intracranial blood. Those numbers are unsourced in the library, so they are not printed here. Say that the scale is an attenuation scale anchored to fixed reference materials, which is why a window is quoted as a width and a level, and quote the anchors only from a textbook you can name.

SEQUENCE	CEREBROSPINAL FLUID	WHITE AGAINST GREY MATTER	WHAT IT IS FOR
T1-weighted	Low, dark	**White matter brighter than grey**	The anatomical sequence; subacute haemorrhage; post-contrast enhancement
T2-weighted	High, bright	**White matter darker than grey**	Vasogenic oedema, gliosis, demyelination, fluid collections
FLAIR	Suppressed, black	**White matter darker than grey, because it is an inversion-recovery T2 sequence**	Periventricular and cortical oedema, subcortical ischaemic lesions, demyelinating plaques
Diffusion-weighted	Low	Low to intermediate	Acute ischaemic stroke, hypercellular tumour, abscess cavity
Apparent diffusion coefficient map	High, bright	Brighter than grey	**The objective check: true restricted diffusion is dark here**
Susceptibility-weighted	Variable	Intermediate	Microhaemorrhage, cavernoma, calcification, petechial shearing injury, through blooming from paramagnetic blood products or diamagnetic calcium

Intravenous gadolinium, typically 5 to 15 mL, shortens T1 so that vascularised tumour and areas of broken blood-brain barrier become brightly hyperintense. Spectroscopy detects N-acetylaspartate at 2.0 ppm, choline at 3.2, creatine at 3.0 and lactate and lipids at 1.3. Functional imaging detects blood-oxygen-level-dependent change, and diffusion tensor imaging maps white matter tract trajectories.

- **RULE** Two two-second tests. To separate T1 from T2, look at the ventricular fluid: dark on T1, bright on T2. To separate FLAIR from T1, where the fluid is dark on both, look at the **white matter**: brighter than grey on T1, darker than grey on FLAIR.

Which to order, in psychiatric practice. Plain computed tomography carries five indications.

- **The acute neurosurgical emergency.** Suspected intracranial haemorrhage, extradural or subdural collection after head injury.
- **Thunderclap headache.** To exclude subarachnoid haemorrhage.
- **The severely agitated or delirious patient.** Who cannot stay motionless for a long acquisition, since the scan takes a minute or less.
- **Calvarial or facial trauma, and foreign bodies.** The bone window answers the question directly.
- **Where availability and cost decide.** It is widely available across Indian district and taluk hospitals.

Magnetic resonance imaging carries six.

- **First-episode psychosis in an older adult, or an atypical presentation.** To exclude a space-occupying lesion, a low-grade frontal or temporal glioma or a meningioma.

- **The dementia work-up.** Where regional atrophy, hippocampal volume loss and subcortical leukoaraiosis all matter.
- **Suspected acute ischaemic stroke within the first few hours.** Where plain computed tomography is frequently normal.
- **Posterior fossa and brainstem lesions.** Where dense petrous bone causes beam-hardening artifact.
- **Suspected neuroinflammatory and autoimmune disease.** Presenting with affective or psychotic symptoms.
- **The problem-solving scan.** After an equivocal screening study.

Contraindications. Answer in two groups and declare the provenance honestly: each individual hazard below is sourced, while the sorting into absolute and relative is the conventional teaching framework rather than a cited classification, so defend each placement on its mechanism.

Treat two groups as absolute.

- **Ferromagnetic foreign bodies and implants.** The static field exerts translational and rotational force, so sheet-metal fragments in welders and metal workers, shrapnel or bullet fragments near vital structures risk tissue damage or vascular laceration, a projectile and torque hazard no amount of cooperation removes.
- **Implanted electronic devices that are not labelled conditional.** Including pacemakers, implantable defibrillators, deep brain stimulators and older aneurysm clips, where the mechanism is displacement, electrical interference or radiofrequency lead heating.

Treat as relative: cochlear implants and other devices carrying a conditional label, where the decision turns on the device documentation and the stated field strength; severe claustrophobia and tremor, where mild sedation is commonly used, which is what makes it relative; and large fluid collections or ascites, which distort the field and degrade the image rather than endangering the patient.

Pregnancy is handled as a caution rather than a prohibition, and the trimester rules and the gadolinium position in pregnancy are unsourced, so do not quote a rule you cannot cite. Gadolinium carries a risk of nephrogenic systemic fibrosis in severe renal impairment, which restricts the contrast agent and not the scan.

On the other modality, ionising radiation means protocols follow the principle of keeping dose as low as reasonably achievable, repeated scans in children and young patients are avoided where a non-ionising option exists, abdominal shielding is used when a head scan is necessary in pregnancy, and iodinated contrast carries risks of contrast-induced kidney injury, anaphylactoid reaction and extravasation.

TRAP Three that lose marks. Calling an MRI lesion "hypodense": density and attenuation belong to computed tomography only. Reading every bright diffusion lesion as acute infarction: the sequence retains residual T2 weighting, so chronic fluid-rich lesions **shine through**, and true restriction must be confirmed as dark on the coefficient map. And reviewing a head scan only on standard brain settings, where acute blood against the inner table blends with bone, so a thin crescentic or isodense subdural collection is missed; switch to the subdural window and verify that the sulci reach the inner table.

HOLD THIS

Attenuation for one modality, signal intensity for the other; fluid dark on T1, bright on T2, suppressed on FLAIR. Do not call restricted diffusion without checking the coefficient map.

WHAT EARNS THE MARKS

- **The proof named, not assumed**: point to the feature on the image that fixes the modality
- **Attenuation against signal intensity**: the vocabulary has to match the modality
- **The two-second fluid test**: ventricular fluid separates T1 from T2 at a glance
- **Coefficient map before restricted diffusion**: bright on diffusion alone is not enough

SEE ALSO 18 Dementia imaging and the visual scales 19 Imaging in the primary psychiatric disorders 20 Functional imaging: PET, SPECT, DaTscan, fMRI 21 Spotters: blood, territories, hydrocephalus, infection

18 · Dementia imaging and the visual scales

NEUROIMAGING · TOPIC 18

Grade the medial temporal atrophy on this scan.

INVESTIGATIONS TABLE, IMAGING SPOTTER

THE SHAPE OF THE ANSWER

1 Plane	Coronal sections at the anterior pons, stated first
2 The scale	Choroid fissure, temporal horn, hippocampal height
3 Age thresholds	The cut-offs, and where the score stops working
4 Vascular contribution	Small vessel disease and its share of the age effect
5 Other scales	The posterior signature and the brainstem spotters

Say the plane before the score. Structural magnetic resonance imaging is the reference modality for neurodegenerative dementia, with volumetric T1 and axial T2 fluid-attenuated inversion recovery sequences on 1.5 or 3 Tesla scanners.

Hippocampal atrophy is assessed on high-resolution coronal T1 sections reconstructed perpendicular to the long axis of the temporal lobe, that is parallel to the brainstem axis, through the body of the hippocampus at the level of the anterior pons. Both hemispheres are scored independently, and raters may use the higher hemisphere score or the average of the two.

Visual rating is reproducible: the intraclass correlation coefficient was 0.86 in a sample of 553 participants. Visual scoring of medial temporal atrophy is validated on computed tomography as well as on magnetic resonance imaging, which matters wherever plain computed tomography is the accessible first-line study.

GRADE	CHOROID FISSURE	TEMPORAL HORN	HIPPOCAMPAL HEIGHT
0	Normal	Normal	**Normal, with no fluid visible around the hippocampus**
1	Slightly widened	Normal	**Normal, with mild fluid visibility in the fissure**
2	Moderately widened	Mildly enlarged	**Mild loss of height**
3	Markedly widened	Moderately enlarged	**Moderate loss of height, a definite volumetric reduction**
4	Markedly widened	Markedly enlarged	**Severe volume loss with internal architecture lost**

The three morphological boundaries the scale grades, at the level of the anterior pons. Sensitivity is about 75 per cent and specificity about 85 per cent for separating Alzheimer disease from cognitively unimpaired individuals. A known limitation: it does not assess the entorhinal cortex, which degenerates early, and a complementary entorhinal scale addresses that region.

THRESHOLD SET	ABNORMAL AT
Conventional clinical	**Under 75 years, a score of 2 or more; aged 75 years or older, a score of 3 or more**
Original threshold	Average of 1 or more below age 75, and 1.5 or more above 75
Memory clinic criteria, average score	Under 65 years 1.0 or more; 65 to 74 years 1.5 or more; 75 to 84 years 2.0 or more; 85 years or older 2.0 or more
Decade-specific set	45 to 64 years 1.5 or more; 65 to 74 years 1.5; 75 to 84 years 2.0; 85 to 94 years 2.5
Simplified age-independent cut-off	**Above 1, validated against biomarker-confirmed cohorts, derived from a healthy control group of 195 participants with a mean of 0.2 and standard deviation 0.4; accuracy 0.73, sensitivity 0.88, specificity 0.57, against accuracy 0.62, sensitivity 0.99 and specificity 0.24 for the standard age-adjusted criteria**

Why the score decays as a test in the very old. Diagnostic utility declines above age 85, because medial temporal volume reduction in nonagenarians is driven by competing co-pathologies.

In community cohorts at death with a mean age of 89.7 years, limbic-predominant age-related TDP-43 encephalopathy is present in 17.3 per cent of amnestic dementias, Alzheimer pathology in 39.4 per cent and cerebrovascular disease in 24.8 per cent.

And in mild cognitive impairment, automated atlas-based and manual volumetry both predict conversion to Alzheimer dementia at 2 years better, with areas under the curve of 0.71 each, than the visual rating at 0.65 or lateral ventricular volume at 0.60.

The vascular contribution, which is the part candidates omit. White matter hyperintensities represent cerebral small vessel disease, with population prevalence rising from 10 to 20 per cent around age 60 to near-ubiquity in extreme old age.

Mediation analyses show that they account for 30 per cent to 42 per cent of the association between chronological age and the medial temporal atrophy score, whole-cohort estimates 40 to 42 per cent and cognitively impaired subgroups 30 to 32 per cent.

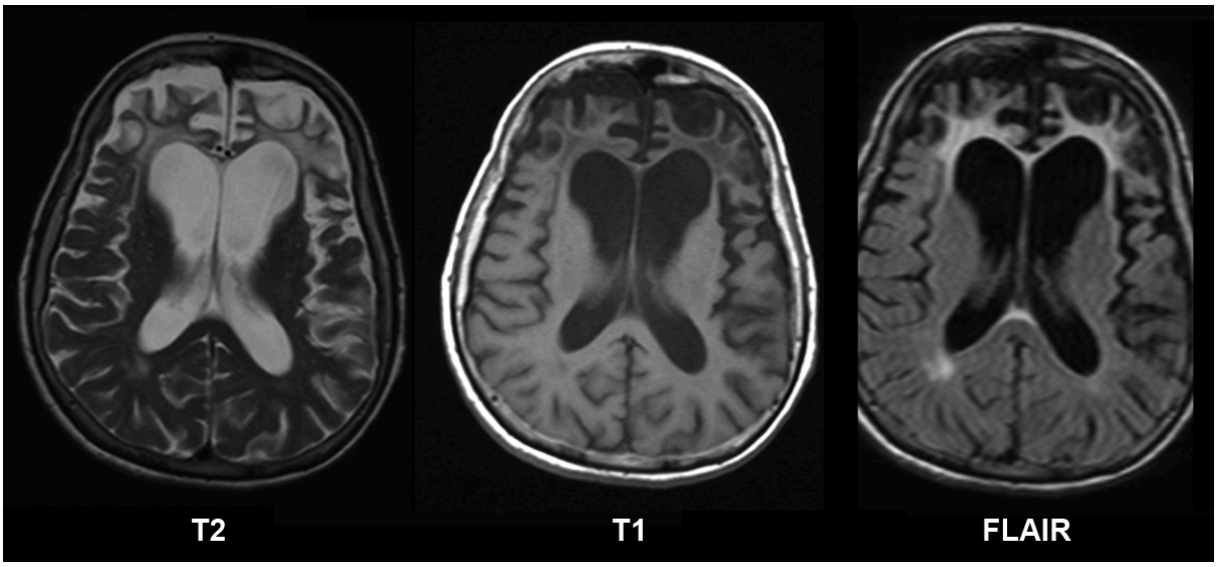
The mechanism is damage to periventricular tracts such as the inferior fronto-occipital fasciculus, producing downstream deafferentation and medial temporal degeneration, with chronic hypoperfusion also accelerating cortical tau pathology.

On biomarkers, medial temporal atrophy and hippocampal volumes correlate with total and phosphorylated tau in cerebrospinal fluid, with plasma phosphorylated tau and with temporal tau uptake on positron emission tomography, and visual scores and ventricular volume correlate with cerebrospinal amyloid beta 1-42.

Neocortical amyloid burden does not correlate directly with the atrophy score in early disease, because amyloid deposition begins in the default-mode network, precuneus and posterior cingulate, rather than in the medial temporal allocortex.

Frontal atrophy across three MRI sequences

Companion to original page 431



MRI, axial images labelled T2, T1 and FLAIR in the source. Frontal cortical and white-matter atrophy in the source case labelled Pick disease. The sequence labels belong to the source montage. MRI demonstrates an atrophy pattern, not the histological diagnosis of Pick disease.

Compare the anterior atrophy pattern with the medial temporal atrophy plate.

Mikhail Kalinin. Pick's disease.png. CC BY-SA 3.0. https://commons.wikimedia.org/wiki/File:Pick%27s_disease.png Licence: https://creativecommons.org/licenses/by-sa/3.0 Unmodified.

ENTITY	STRUCTURAL SIGNATURE	THE DISCRIMINATOR
Alzheimer disease	Medial temporal and hippocampal atrophy with posterior parietal and precuneal volume loss	**The posterior parietal and precuneal component is what separates it**
Behavioural variant frontotemporal dementia	Prominent, often asymmetric atrophy of orbitofrontal, dorsolateral prefrontal and anterior cingulate cortex with the anterior temporal poles	**Parietal integrity preserved early, even where medial temporal atrophy is severe**
Semantic variant primary progressive aphasia	Severe, strikingly asymmetric anterior temporal atrophy, predominantly the left temporal pole, inferior temporal gyrus and amygdala	**Asymmetry and anterior temporal predominance**
Non-fluent agrammatic primary progressive aphasia	Asymmetric left perisylvian atrophy centred on the inferior frontal gyrus	**Left perisylvian rather than temporal pole**
Vascular cognitive impairment	Extensive confluent white matter hyperintensity, lacunar infarcts in basal ganglia or thalamus, cortical territorial infarcts, dilated perivascular spaces, microbleeds	**Graded on the Fazekas scale, which separates periventricular from deep white matter change; in healthy older individuals without neurodegenerative pathology the mean Fazekas score is about 0.82**
Normal pressure hydrocephalus	Ventriculomegaly out of proportion to sulcal enlargement, with the disproportionately enlarged subarachnoid space pattern: high-convexity and parasagittal sulci crowded against the calvarium while Sylvian fissures and basal cisterns are dilated, plus periventricular transudation	**The high convexities. In Alzheimer-related passive enlargement the convexity sulci are widened, matching or exceeding the ventricular dilatation**

Frontotemporal dementia is the second commonest cause of young-onset neurodegenerative dementia. Note the honest caveats: the Fazekas grade descriptors, the knife-edge and insular descriptors, the Hakim-Adams triad label, the Evans index threshold and the callosal angle values are all carried in the library as unverified notebook material rather than sourced figures, so describe the pattern and do not quote the numbers.

The other scales and the brainstem spotters. The Koedam posterior atrophy score grades parietal atrophy, precuneal widening and posterior cingulate enlargement, and is elevated in early-onset Alzheimer disease and posterior cortical atrophy; the global cortical atrophy scale grades gyral thinning and sulcal widening across regions.

On the spotter table, progressive supranuclear palsy gives midbrain atrophy.

- **Ratios.** A reduced midbrain-to-pons ratio, and an elevated parkinsonism index.
- **Midsagittal T1.** The hummingbird sign, with tegmental atrophy and a preserved pons.
- **Axial midbrain sections.** The Mickey Mouse and morning glory signs.

Multiple system atrophy gives the hot cross bun sign, a cruciform pontine hyperintensity in the cerebellar subtype, the middle cerebellar peduncle sign, and the putaminal rim sign in the parkinsonian subtype. Huntington disease is measured by caudate head atrophy using the frontal horn width to intercaudate distance ratio.

Prion disease gives the hockey stick sign, symmetric hyperintensity of pulvinar and dorsomedial thalamic nuclei, and the pulvinar sign confined to the posterior thalamus.

TRAP Four. Grading hippocampal atrophy on **axial** slices, when the scale is validated on coronal sections aligned to the brainstem axis. Ignoring **age adjustment**, so that a score of 2 is called pathological in an eighty-year-old, where 3 or more is required, and normal in a sixty-year-old, where 2 is unequivocally abnormal. Equating medial temporal atrophy with Alzheimer disease, when high scores also occur in frontotemporal dementia and hippocampal sclerosis and the parietal component is the discriminator. And attributing all hippocampal loss to amyloid and tau while ignoring small vessel disease.

HOLD THIS

Two or more under seventy-five, three or more at seventy-five and above, and the score stops discriminating above eighty-five. Small vessel disease mediates a third to two-fifths of the age effect.

WHAT EARNS THE MARKS

- **Plane before score**: grading on the wrong plane loses the mark first
- **Age-adjusted thresholds**: the same score means different things at different ages
- **The vascular part supplied**: candidates omit small vessel disease, so say it
- **Parietal and precuneal loss**: the pattern that points toward Alzheimer disease

SEE ALSO 17 Naming the modality: CT, MRI, windows and sequences 19 Imaging in the primary psychiatric disorders 20 Functional imaging: PET, SPECT, DaTscan, fMRI 21 Spotters: blood, territories, hydrocephalus, infection

19 · Imaging in the primary psychiatric disorders

NEUROIMAGING · TOPIC 19

What does imaging show in schizophrenia, and would you order it?

INVESTIGATIONS TABLE, IMAGING SPOTTER

THE SHAPE OF THE ANSWER

1 What it is for	Exclusion of an organic cause, said before any finding
2 Compartments	Grey falls, white holds, the fluid space expands
3 Timing	When the enlargement appears, and what stays unproven
4 The thalamus	Cognitive dysmetria, and the circuit claim behind it
5 Other disorders	Mood, obsessive-compulsive, and the habit account

Open with what imaging is for, because the examiner is testing judgement before knowledge. Structural imaging is not indicated to confirm a primary psychiatric diagnosis: the morphometric

and functional differences are group-level statistical associations, not individual diagnostic biomarkers.

In Indian practice it is used to exclude secondary organic causes of psychiatric presentation, indicated by new-onset psychosis or mood episodes in later life, focal neurological deficits, atypical presentation, treatment resistance or unexplained cognitive decline, and specifically to exclude space-occupying lesions, normal pressure hydrocephalus, cerebrovascular events, trauma, and the regionally endemic neuroinfections, neurocysticercosis, tuberculoma and chronic meningoencephalitis.

Which modality, and why the literature reads as it does. Both modalities quantify ventricular size, and the commonest definition of ventricular enlargement uses a volume-to-brain index derived from magnetic resonance and computed tomography reconstruction of the lateral and third ventricles.

Historically, ventricular enlargement in schizophrenia was first described with pneumoencephalography in the early 1960s and later confirmed by computed tomography in chronic patients.

Wider use of magnetic resonance imaging then produced the detail the earlier literature could not resolve: smaller total brain volume, reduced hippocampal and thalamic volumes, and reductions in cortical volume and thickness most pronounced in the frontal and temporal lobes. That is the honest answer to why the detailed structural work-up sits with magnetic resonance imaging.

The comparative cost, local availability and radiation arguments, and the use of computed tomography as a rapid emergency screen for haemorrhage or mass effect, are not carried in these sources, so attribute them to departmental practice rather than to a citation.

COMPARTMENT	SCHIZOPHRENIA	COMPARISON	WHAT IT MEANS
Total intracranial volume	Preserved	Preserved	**No overall difference in head size**
Absolute grey matter	713 ml, standard deviation 77	810 ml, standard deviation 81	**Global cortical and subcortical loss**
Relative grey matter	48.9 per cent	51.6 per cent	**A significant proportional deficit**
Relative cerebrospinal fluid	23.6 per cent	20.6 per cent	**Compensatory ex vacuo expansion**
White matter, absolute and relative	415 ml, 27.5 per cent	441 ml, 27.7 per cent	**No significant difference: the deficit is selective for grey matter, with localised differences in the lateral optic radiation of occipital cortex**
Mean thalamic volume	About 4 per cent reduction	Reference	**Quote it with the caveat that only three studies contributed to this effect**
Lateral ventricles in chronic illness	About 18 per cent enlargement	Reference	**From a meta-analysis of more than 2000 subjects with a mean illness duration of 10 years; degree of enlargement correlates with poor outcome**

Regional grey matter reductions are most prominent in the mediodorsal thalamus, medial and orbital prefrontal cortex, premotor regions, inferolateral temporal lobes and medial temporal structures including the hippocampus.

When does the enlargement appear. There is no indisputable evidence of ventricular enlargement in ultra-high-risk individuals or before the overt onset of psychosis. It is demonstrable at first episode, described over 20 years ago and confirmed by cross-sectional and longitudinal meta-analyses showing increases in right, left and total lateral ventricles.

In chronic cohorts it reaches the 18 per cent figure above and correlates with poor outcome, and child-onset schizophrenia shows both baseline enlargement and accelerated progression. Patients maintained on second-generation antipsychotics show less enlargement over time than those on first-generation drugs.

Against a simple neurodegenerative reading, grey matter volume correlates negatively with age but the *rate* of decline across age does not differ significantly from comparison subjects, which argues against an accelerated generalised involutionary process.

The thalamus and cognitive dysmetria. Grey matter depletion localises to the mediodorsal nucleus, especially on the left, and that nucleus connects reciprocally with prefrontal cortex.

Within schizophrenia cohorts, ventromedial prefrontal grey matter loss shows a significant negative relationship with family history of psychosis, correlation 0.71, pointing to genetic influence on prefrontal maturation, while higher educational achievement correlates positively with relative preservation of mediodorsal thalamic grey matter, correlation 0.48, associated with better premorbid functioning and lower negative symptom severity.

Cognitive dysmetria is Andreasen's term for the failure of fluid coordination and synchrony of mental activity produced by structural and functional impairment of cortico-subcortical-thalamic-cerebellar circuitry: because the thalamus filters and relays, structural loss disrupts gating and the timing of signal transfer, which surfaces as hallucinations, persecutory beliefs, executive impairment and formal thought disorder.

DISORDER	STRUCTURAL FINDINGS	FUNCTIONAL FINDINGS
Schizophrenia	Ventricular enlargement of about 18 per cent in chronic illness, global grey matter deficit, mediodorsal thalamic reduction	**Thalamocortical dysconnectivity, prefrontal hypometabolism, cognitive dysmetria**
Bipolar disorder type I	Lateral ventricular enlargement confirmed in more than 1700 patients, widespread cortical grey matter reduction, reduced cortical thickness, disrupted white matter microstructure with elevated hyperintensities	**Altered fronto-limbic activation during emotion regulation and reward processing**
Bipolar disorder type II	Greater subcortical volume preservation and less pronounced ventricular enlargement	**Distinct connectivity alterations during reward and emotional tasks. Hippocampal and cerebellar reductions occur across both subtypes**
Major depressive disorder	Hippocampal volume reduction	**Reduced glucose metabolism and blood flow in left dorsolateral prefrontal cortex, correlating with executive dysfunction, psychomotor retardation and apathy; increased flow in left ventrolateral prefrontal cortex in some cohorts; reduced left hippocampal metabolism correlating negatively with severity; decreased caudate metabolism with raised thalamic metabolism**
Obsessive-compulsive disorder	Cortico-striato-thalamo-cortical alterations with volumetric change in caudate and orbitofrontal cortex	**Raised glucose metabolism and perfusion in orbitofrontal cortex, anterior cingulate, caudate and thalamus in untreated non-depressed patients, rising further on symptom provocation and falling with effective treatment by serotonin reuptake inhibitors or cognitive behavioural therapy**
Concurrent obsessive-compulsive disorder and depression	Mixed volumetric patterns	**Left hippocampal hypometabolism present, and metabolism in thalamus, caudate and hippocampus significantly *lower* than in obsessive-compulsive disorder alone**

The comorbidity study worth quoting. Three figures, in this order.

- **Lifetime prevalence.** Obsessive-compulsive disorder has an estimated lifetime prevalence of 2 per cent to 3 per cent.

- **Depression within it.** 60 per cent to 80 per cent of patients experience a major depressive episode in their lifetime, with about a third presenting with concurrent depression.
- **Conversely.** Obsessions and compulsions occur during depressive episodes in 22 per cent to 38 per cent of primary depressive patients.

A metabolic study across 88 subjects, 27 with the obsessive-compulsive disorder alone, 27 with depression alone, 17 with both and 17 controls, established the pattern in the table: left hippocampal hypometabolism tracks the depressive episode regardless of which diagnosis is primary, and the comorbid group is not a simple sum of the two.

Treatment response follows: sertraline was superior to desipramine for depressive symptoms in the comorbid group, whereas primary unipolar depression generally shows equivalent response between a serotonin reuptake inhibitor and a tricyclic.

The habit account of compulsion. In a functional imaging study of 70 individuals performing a shock-avoidance pedal-pressing task, avoidance behaviour was assessed after the threat was deactivated. Patients with obsessive-compulsive disorder were impaired in discontinuing the pedal-pressing habit once the shock block was removed, and showed significant caudate hyperactivation after threat removal.

Medial orbitofrontal activation occurred during active avoidance but did not correlate with habit formation. The reading is that compulsions reflect autonomous subcortical habit systems in the caudate escaping top-down prefrontal control, rather than purely goal-directed worry.

TRAP Ventricular enlargement is **not** pathognomonic of schizophrenia; it is a non-specific marker of tissue loss seen in ageing, neurodegeneration and bipolar disorder type I. It is **not** proven before the onset of psychosis. Compartments do **not** shrink uniformly: total intracranial and white matter volumes are preserved. And the 4 per cent thalamic figure rests on three contributing studies, so do not attach it to the larger morphometric literature, in which a frequently quoted meta-analysis of 58 morphometric studies reported medial temporal lobe volume differences as the most frequent observation, which is a separate finding.

HOLD THIS

Grey matter falls, white matter and total intracranial volume hold, and the fluid space expands to fill the difference. Imaging in primary psychiatric illness excludes an organic cause; it does not make the diagnosis.

WHAT EARNS THE MARKS

- **Judgement before knowledge**: state the indication before you state the findings
- **Non-specific enlargement**: ventricular size marks tissue loss, it does not diagnose
- **Compartments accounted for**: grey, white and fluid described in one movement
- **Imaging excludes, it does not diagnose**: the sentence the examiner is listening for

SEE ALSO 16 Evoked potentials and the gating measures 17 Naming the modality: CT, MRI, windows and sequences 18 Dementia imaging and the visual scales 20 Functional imaging: PET, SPECT, DaTscan, fMRI

20 · Functional imaging: PET, SPECT, DaTscan, fMRI

NEUROIMAGING · TOPIC 20

Compare PET, SPECT and functional MRI, and tell me what a DaTscan settles.

INVESTIGATIONS TABLE, IMAGING SPOTTER

THE SHAPE OF THE ANSWER

1	**The frame**	Structure is the hardware, function is the software
2	**The grid**	Tracer, resolution, availability and radiation for each
3	**Preparation**	What the patient does before the scan is acquired
4	**The BOLD signal**	The mechanism, stated accurately, with its size
5	**Clinical patterns**	The dementia signatures, and what transporter imaging settles

Structure is hardware, function is software. Computed tomography and magnetic resonance imaging display anatomy, and in early neurodegenerative disease, including early Parkinson disease, macroscopic anatomy is largely normal. Functional imaging maps dynamic physiology: regional cerebral glucose metabolism, regional blood flow, neurotransmitter transporter density, or changes in blood oxygenation.

Positron emission tomography uses radiotracers labelled with positron-emitting isotopes, injected intravenously and detected by circular detector arrays to build three-dimensional activity maps; the standard tracer in neurocognitive evaluation is fluorodeoxyglucose, a glucose analogue, so regional uptake reflects local metabolic activity and hypometabolism marks quiet, dysfunctional or degenerating neuronal populations.

Single-photon emission computed tomography uses gamma-emitting radiopharmaceuticals detected by rotating gamma cameras to measure blood flow or receptor and transporter density; its spatial resolution is moderate against the high resolution of positron emission tomography, but the instrumentation is widely available across Indian tertiary centres while positron facilities are concentrated in specialised nuclear medicine units.

FEATURE	POSITRON EMISSION TOMOGRAPHY	SINGLE-PHOTON EMISSION TOMOGRAPHY, DATSCAN	FUNCTIONAL MAGNETIC RESONANCE IMAGING
Signal	Positron-emitting radiopharmaceutical, for instance fluorodeoxyglucose	Gamma-emitting radiopharmaceutical, ioflupane iodine-123	Endogenous blood-oxygenation-level-dependent contrast
Measures	Glucose metabolism or protein deposition such as amyloid	**Dopamine transporter density or regional blood flow**	**The haemodynamic neurovascular response**
Spatial resolution	High	Moderate	High, with structural reference
Radiation	Yes, comparable to background radiation received over a few years	Yes, low-dose	**None: radiofrequency and magnetic fields only**
Availability in India	Specialised regional nuclear imaging centres	Widely available in major tertiary hospitals	Tertiary neuroimaging and research centres

Preparation, which is what the examiner actually asks. For a fluorodeoxyglucose study the patient fasts, taking only plain water, for 4 to 6 hours, because circulating plasma glucose competes directly with the tracer for cerebral uptake and compromises the scan; significant hyperglycaemia warrants deferral or careful stabilisation.

It is an outpatient procedure: total clinic time is 2 to 4 hours, covering injection, an uptake interval and imaging, while acquisition itself takes 30 to 60 minutes.

Afterwards the patient drinks 8 to 10 glasses of water across the rest of the day to speed renal excretion, and as a routine radiation precaution avoids prolonged close proximity to pregnant women and infants for 6 to 12 hours.

For a dopamine transporter study, drugs with high affinity for the transporter, including central stimulants such as methylphenidate and specific antidepressants, are discontinued for 24 to 48 hours to prevent pharmacodynamic interference. Side effects of the tracer are minimal: occasional headache, dizziness, increased appetite and a transient crawling sensation under the skin.

CONDITION	PATTERN	WHY IT MATTERS CLINICALLY
Alzheimer disease	Hypometabolism or hypoperfusion in posterior temporoparietal cortex and precuneus	**Striatal transporter uptake is preserved, which separates it from Lewy body pathology**
Frontotemporal dementia	Hypometabolism or hypoperfusion in frontal and anterior temporal lobes	**A cholinesterase inhibitor prescribed for Alzheimer disease may worsen behavioural disinhibition here, which is why the distinction is not academic**
Dementia with Lewy bodies	Occipital cortex hypometabolism, with marked loss of striatal transporter uptake	**Occipital hypometabolism correlates with visual hallucinations, and transporter loss separates it from Alzheimer disease**
Atypical parkinsonism	Asymmetric or symmetric striatal dopamine transporter loss	**Confirms neurodegenerative parkinsonism but does not differentiate progressive supranuclear palsy or multiple system atrophy from idiopathic disease**
Essential tremor	Normal, symmetric, comma-shaped bilateral striatal uptake	**This is the validated indication: separating essential tremor from neurodegenerative parkinsonism**
Idiopathic Parkinson disease	Marked asymmetric reduction in striatal uptake, beginning in the posterior and dorsal putamen and spreading across the basal ganglia	**Loss is asymmetric and posterior-dorsal at onset, which is how it is told from age-related decline**
Drug-induced parkinsonism	Presynaptic transporters typically intact, so the scan is normal	**Because the mechanism is postsynaptic D2 blockade, unless an underlying idiopathic disease is being unmasked**

RULE A dopamine transporter scan separates **neurodegenerative parkinsonism from essential tremor**, and does not separate idiopathic Parkinson disease from the atypical syndromes, because presynaptic nigrostriatal degeneration occurs across all of them. Add the humility: a subset of people clinically suspected of early Parkinson disease have normal scans and do not go on to develop progressive symptoms.

The BOLD mechanism, stated accurately. The signal is not sensitive to action potentials; it detects haemodynamic change. Deoxyhaemoglobin is paramagnetic and attenuates signal, oxyhaemoglobin is diamagnetic and does not.

Neural activity raises local blood flow far more than oxygen consumption, for instance finger tapping raises motor cortex flow by about 60 per cent, so the over-supply flushes

deoxyhaemoglobin out and the signal strengthens slightly, by about 1 per cent. Task paradigms alternate 30 second task and rest blocks.

Resting-state and arterial spin labelling. Resting-state imaging measures spontaneous low-frequency fluctuations at rest, and regions whose fluctuations correlate are read as functional networks; altered connectivity is reported in schizophrenia, depression, autism spectrum disorders and Alzheimer disease.

Arterial spin labelling inverts the spins of arterial water before delivery and subtracts tagged from untagged images to leave a quantitative perfusion map. That is its advantage: the blood-oxygenation signal is qualitative, a relative contrast between two states, while spin labelling measures absolute flow in any state and can identify resting hypoperfusion.

Before you order any of it. Screen clinically first.

- **Examination.** Cognitive and motor examination.
- **Laboratory exclusion.** Vitamin B12 deficiency and thyroid disease.
- **Structural imaging.** To exclude a space-occupying lesion, chronic subdural collection, tumour or normal pressure hydrocephalus.
- **Cautions.** Pregnancy and lactation for the radiotracer studies, uncontrolled diabetes for tracer uptake, claustrophobia, and ferromagnetic implants for the magnetic modalities.

ASK AND VERIFY The classic regional patterns in primary psychiatric illness, **frontal hypometabolism or hypofrontality in schizophrenia on positron or single-photon imaging**, and the orbitofrontal and caudate findings in obsessive-compulsive disorder, are not asserted in this chapter's sources, which cover degenerative patterns and generic connectivity only. The orbitofrontal, anterior cingulate, caudate and thalamic hypermetabolism of obsessive-compulsive disorder, and prefrontal hypometabolism in schizophrenia, are sourced in the preceding topic, so cite them there. Do not claim that functional imaging measures action potentials, and do not quote administered radioactivity, tracer half-life, rupee costs, cognitive cut-off scores, repetition and echo times in milliseconds or absolute perfusion values, none of which is sourced.

HOLD THIS

Posterior temporoparietal is Alzheimer disease, frontal and anterior temporal is frontotemporal dementia, occipital is Lewy body disease. The blood-oxygenation signal is an indirect haemodynamic surrogate of about one per cent.

WHAT EARNS THE MARKS

- **The tracer named**: each modality answers a different physiological question
- **Three dementia patterns in order**: posterior temporoparietal, frontal, occipital
- **What the transporter scan cannot do**: it does not separate the atypical syndromes
- **An indirect surrogate**: the blood-oxygenation signal is haemodynamic, not neural

SEE ALSO 17 Naming the modality: CT, MRI, windows and sequences 18 Dementia imaging and the visual scales 19 Imaging in the primary psychiatric disorders 25 Neuroendocrine tests and the status of biomarkers

21 · Spotters: blood, territories, hydrocephalus, infection

NEUROIMAGING · TOPIC 21

Show me what you see on this plain scan.

INVESTIGATIONS TABLE, IMAGING SPOTTER

THE SHAPE OF THE ANSWER

1 **Three questions**	Shape, sutures, and whether it crosses the falx
2 **The collections**	Extradural against subdural, with the density clock
3 **Surgery and herniation**	Thresholds, active bleeding, the herniation signs
4 **Territories**	The arterial patterns and their spotter signs
5 **Hydrocephalus and infection**	The patterns, with Wilson disease drawn

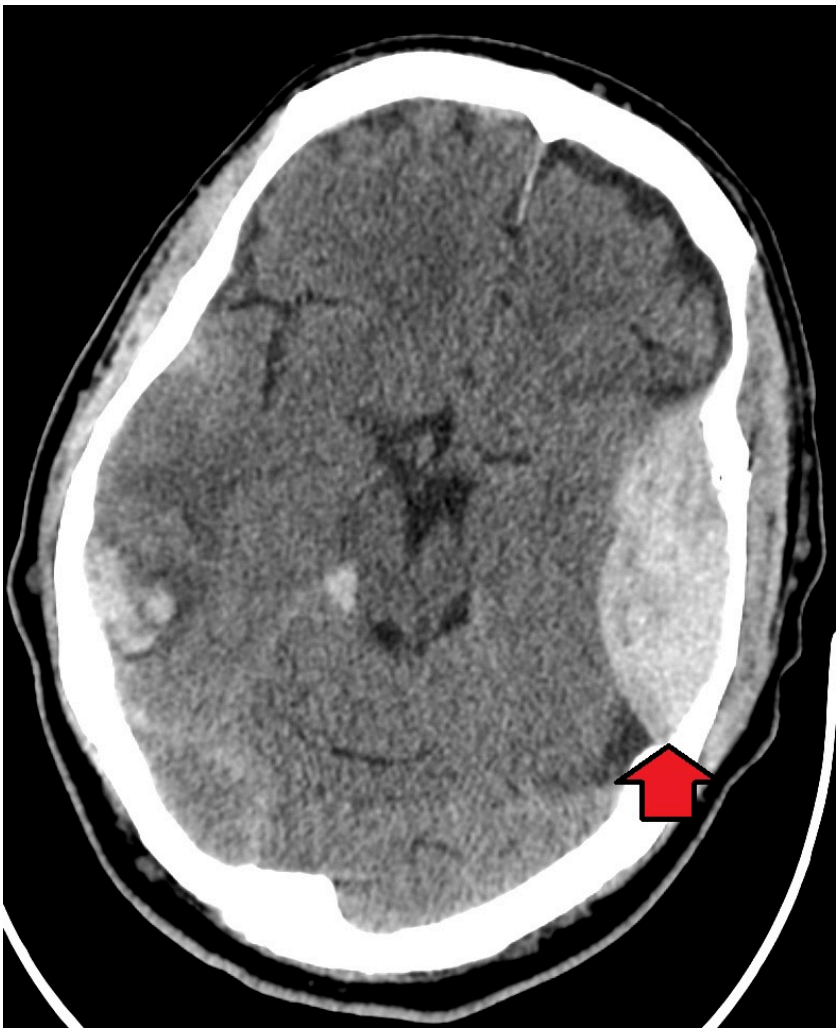
Shape, sutures, and whether it crosses the falx. An extradural collection is bleeding into the potential space between the inner table and the outer periosteal layer of the dura; a subdural collection is blood between the inner meningeal layer of the dura and the arachnoid.

The systematic sequence on an emergency plain scan is parenchyma for symmetry and hypoattenuation, the ventricles and basal cisterns for effacement or shift, the extra-axial spaces for hyperattenuating blood, then bone windows for fractures.

FEATURE	EXTRADURAL HAEMATOMA	SUBDURAL HAEMATOMA
Source	Arterial, the middle meningeal artery in about 75 per cent; a torn dural venous sinus is an uncommon venous source with no frequency given	Venous, from shearing of cortical bridging veins crossing to the dural sinuses
Shape	**Biconvex, lentiform, lemon-shaped**	**Crescentic, concave internally, banana-shaped**
Sutures and dural reflections	**Does not cross sutures, because the periosteum is bound there, but can cross the falx and tentorium**	**Readily crosses sutures across the hemisphere, but is limited by the falx and tentorium**
Fracture association	Associated with a skull fracture in about 75 per cent, and 85 to 90 per cent in some series	Less often associated with fracture, and common after minor trauma
Demographic	Typically adults under 50 years and children, up to 80 per cent male	The elderly with cerebral atrophy, patients on anticoagulation, infants
Course	Loss of consciousness in about 60 per cent, a lucid interval in under 20 per cent, then rapid decline	Insidious decline, fluctuating confusion, progressive mass effect

Extradural blood: lentiform contour

Companion to original page 443



CT, axial plane. A hyperattenuating lentiform extra-axial collection lies on the patient's left, image right. The source identifies an epidural haematoma with a skull fracture. Use the lens-shaped contour and extra-axial position as the visual comparison with the subdural plate. The skull fracture is a source-case finding, not necessarily demonstrated on this view.

Compare the convex inner margin with the crescentic subdural example.

James Heilman, MD. EpiduralHematoma.jpg. CC BY-SA 4.0. https://commons.wikimedia.org/wiki/File:EpiduralHematoma.jpg Licence: https://creativecommons.org/licenses/by-sa/4.0 Unmodified.

Subdural blood with midline shift

Companion to original page 443

CT, axial plane. A left frontoparietal subdural collection is associated with marked midline shift. The source arrow is retained. Assess the displaced midline as evidence of mass effect, separately from recognising the collection. A single slice does not establish the full extent of herniation.

Trace the collection's inner contour and describe mass effect before naming it.

James Heilman, MD. Subduralandherniation.PNG. CC BY-SA 3.0. https://commons.wikimedia.org/wiki/File:Subduralandherniation.PNG Licence: https://creativecommons.org/licenses/by-sa/3.0 Unmodified.

Blood in the basal cisterns

Companion to original page 443

CT, axial plane. Hyperattenuating blood occupies the basal subarachnoid cisterns. The source also identifies widening of the temporal horns. Identify the anatomical compartment occupied by blood before considering the cause. This CT does not by itself identify a ruptured aneurysm.

Locate the blood in the cisterns rather than the extra-axial convexity or parenchyma.

Lipothymia. Subarachnoid haemorrhage.jpg. CC BY-SA 3.0. https://commons.wikimedia.org/wiki/File:Subarachnoid_haemorrhage.jpg Licence: http://creativecommons.org/licenses/by-sa/3.0/ Unmodified.

When the extradural collection goes to theatre. Evacuation by craniotomy is required on any of these grounds.

- **Volume.** Greater than 30 cubic centimetres, regardless of the coma score.
- **Thickness or shift.** Thickness greater than 15 mm, or midline shift greater than 5 mm, in a patient with neurological symptoms or deterioration.
- **Coma with a focal sign.** A comatose patient with a Glasgow Coma Scale score below 9 who has pupillary asymmetry or radiological evidence of herniation.
- **Progressive deterioration.** Any documented progressive neurological deterioration attributable to the haematoma.
- **A posterior fossa collection.** Under 5 per cent of adult and under 10 per cent of paediatric cases, but carrying a high hazard of rapid brainstem compression and tonsillar impaction.
- **Enlargement or active bleeding.** Demonstrated enlargement on repeat imaging, or active contrast extravasation.

Conservative management is for small collections without mass effect, extravasation or shift.

How a subdural collection changes with time. Acute blood is hyperdense to cortical grey matter, reflecting retracted clot in the initial days. Between 3 and 21 days it becomes isodense to parenchyma, and then identification relies on indirect signs: unilateral effacement of cortical sulci, medial displacement of the grey-white junction and ventricular compression.

Beyond 21 days it is hypodense, approaching fluid density through complete liquefaction, which can mimic a hygroma. Mixed density with layered hyperdense sediment under hypodense supernatant, the haematocrit effect, or patchy heterogeneity indicates rebleeding into a chronic collection. Rarely acute blood appears isodense because of severe anaemia, coagulopathy or dilution by fluid after an arachnoid tear.

TRAP The lentiform subdural collection. About 8 per cent to 14 per cent of acute subdural haematomas present with a biconvex configuration mimicking an extradural collection, because pre-existing dural adhesions from previous surgery, burr holes or cranial irradiation compartmentalise the space and restrict hemispheric spread. Four features establish the subdural location: a **peripheral crescentic tail** extending beyond the biconvex core; an **obtuse angle** between the haematoma edge and the inner table; a **distinct dural line** between the outer margin and the calvarium; and **direct continuity** with underlying intracerebral contusions.

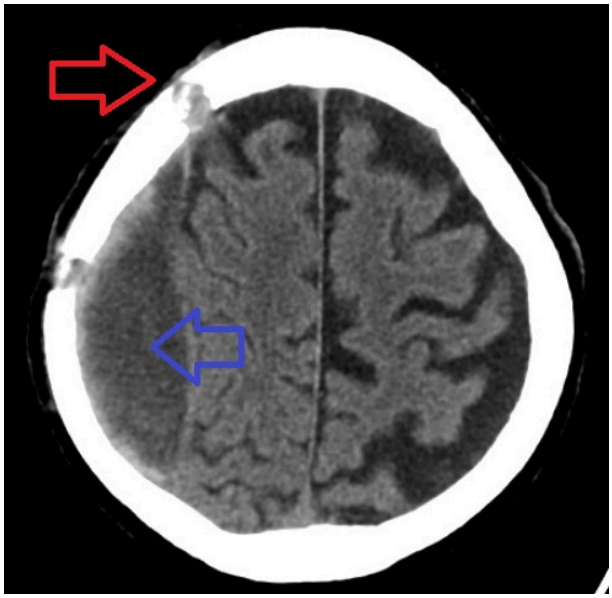
Active bleeding and herniation. The *swirl sign* on a plain scan is irregular low-attenuation unclotted fresh blood swirling within high-attenuation clot, a marker of active hyperacute extravasation indicating rapid enlargement; on angiography the spot sign confirms dynamic arterial bleeding.

Then the herniation syndromes.

- **Subfalcine.** Medial displacement of the cingulate gyrus under the falx, effacing the ipsilateral ventricle and trapping the contralateral foramen of Monro.
- **Uncal, or transtentorial.** Medial displacement of the temporal uncus over the tentorial notch, compressing the third cranial nerve to give an ipsilateral dilated non-reactive pupil, and compressing the posterior cerebral artery.
- **Duret haemorrhage.** Linear flame-shaped haemorrhagic streaks in the paramedian midbrain and upper pons from rapid downward brainstem displacement tearing perforating branches of the basilar artery,

Chronic subdural collection after burr holes

Companion to original page 444



Brain CT, axial plane. A crescentic low-attenuation extra-axial collection is marked by the blue arrow; the red arrow marks an old burr hole. The source identifies a chronic subdural bleed. Retain the arrow colours when describing this treated source case; the burr hole is a prior intervention, not part of the collection.

Contrast low-attenuation chronic blood with the hyperattenuating collection and recognise prior treatment.

Doc James. ChronicSubduralPostBurrHoles.jpg. CC BY-SA 4.0. https://commons.wikimedia.org/wiki/File%3AChronicSubduralPostBurrHoles.jpg
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which carries a grave prognosis.

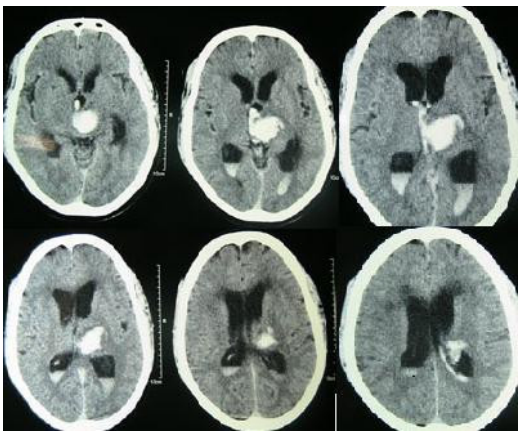
TERRITORY OR PATTERN	WHAT IT INVOLVES	THE SPOTTER SIGN
Middle cerebral artery	Lateral convexity of frontal, parietal and superior temporal lobes, with lenticulostriate supply to basal ganglia and internal capsule	**Hyperdense artery sign for thrombus in the horizontal segment, the dot sign for thrombus in Sylvian branches, insular ribbon loss, and the disappearing basal ganglia sign**
Anterior cerebral artery	Medial surfaces of frontal and parietal lobes	**Contralateral leg-predominant weakness, urinary incontinence and abulia**
Posterior cerebral artery	Occipital lobe, inferomedial temporal lobe, posterior thalamus	**Homonymous hemianopia, alexia without agraphia, thalamic pain syndromes**
Artery of Percheron	A solitary variant branch from one posterior cerebral trunk supplying bilateral paramedian thalami and rostral midbrain	**Acute stupor with vertical gaze palsy and fluctuating delirium, a psychiatric presentation of a vascular event**
Watershed infarcts	Between anterior and middle, or middle and posterior, terminal zones	**Wedge-shaped or rosary-like lesions after profound systemic hypotension**
Cerebral venous sinus thrombosis	Infarcts that do not conform to an arterial distribution, often in young or postpartum women, with high rates of bilateral haemorrhagic transformation	**Dense triangle sign, cord sign for a thrombosed cortical vein, and the empty delta sign in the superior sagittal sinus after contrast**
Hypertensive intracerebral haemorrhage	Deep nuclear locations supplied by penetrating arterioles prone to lipohyalinosis: putamen, internal capsule, thalamus, pons, cerebellar hemispheres	**Deep and central. Volume is estimated with the ABC over 2 formula**
Cerebral amyloid angiopathy	Lobar haemorrhage at the grey-white junction in older patients	**Superficial and lobar, with numerous punctate cortical and subcortical microbleeds on gradient echo or susceptibility imaging, sparing the deep basal ganglia**
Haemorrhagic transformation	Reperfusion into fragile infarcted capillary beds	**The haemorrhage sits inside a pre-existing territorial infarct boundary**

Wilson disease, the sign the examiner wants drawn. An autosomal recessive disorder of hepatic copper excretion caused by mutations in the ATP7B gene on chromosome 13, with copper deposition in lenticular nuclei, thalamus, midbrain and pons.

On axial T2 and fluid-attenuated inversion recovery imaging of the midbrain, the *face of the giant panda*: hyperintensity of the midbrain tegmentum forms the face, the normally hypointense red

Thalamic blood with ventricular extension

Companion to original page 445



CT, serial axial slices. A thalamic haemorrhage extends into the third and lateral ventricles in the source case. Trace blood within the ventricles separately from the parenchymal focus. The small source composite is deliberately displayed at limited width to avoid false detail.

Separate the parenchymal origin from intraventricular extension.

Yadav YR, Mukerji G, Shenoy R, Basoor A, Jain G, Nelson A. Intracerebral hemorrhage.jpg. CC BY 2.0. https://commons.wikimedia.org/wiki/File:Intracerebral_hemorrhage.jpg Licence: https://creativecommons.org/licenses/by/2.0 Unmodified.

Right MCA territory infarct

Companion to original page 445

CT, axial plane. Right MCA territory hypoattenuation in the source infarct case. Describe the territory and attenuation before naming the source diagnosis. This low-resolution teaching image does not support fine vessel or early-ischaemia grading.

Describe the lateral hemispheric territory and compare its attenuation with the other side.

Lucien Monfils. INFARCT.jpg. CC BY-SA 3.0. https://commons.wikimedia.org/wiki/File:INFARCT.jpg Licence: https://creativecommons.org/licenses/by-sa/3.0 Unmodified.

Combined MCA and PCA territory infarcts

Companion to original page 445

CT, axial plane. The source identifies right middle and posterior cerebral artery territory infarcts. Hypoattenuation extends into the posterior territory. This is a combined-territory example, not an isolated posterior cerebral artery infarct. Keep that distinction when using it for a vascular-territory viva.

Recognise that a case can involve more than one arterial territory.

Cerevisae. Right MCA and PCA territories infarct.jpg. CC BY-SA 4.0.
https://commons.wikimedia.org/wiki/File:Right_MCA_and_PCA_territories_infarct.jpg Licence: https://creativecommons.org/licenses/by-sa/4.0
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nuclei form the eyes, preserved hypointensity of the lateral substantia nigra pars reticulata forms the ears, and marked hypointensity of the superior colliculi forms the chin.

It was described by Hitoshi and colleagues in 1991 and was identified in 12 per cent of patients in a 100-patient series.

In the pons the *miniature panda* or panda cub: the central tegmental tract and medial longitudinal fasciculus form the eyes, the aqueduct opening into the fourth ventricle forms the nose and mouth, and the superior cerebellar peduncles form the cheeks. The *bright claustrum* sign is a thin sharply demarcated rim of T2 hyperintensity lateral to the putamen.

Laboratory correlation: decreased serum caeruloplasmin, often below 15 mg/dL and reported at 5.5 to 6.0 in clinical presentations; reduced total serum copper at 60 to 67 mcg/dL against a reference range of 85 to 150; and raised 24-hour urinary copper excretion at 120 to 170 mcg per day against a reference of 50 to 70.

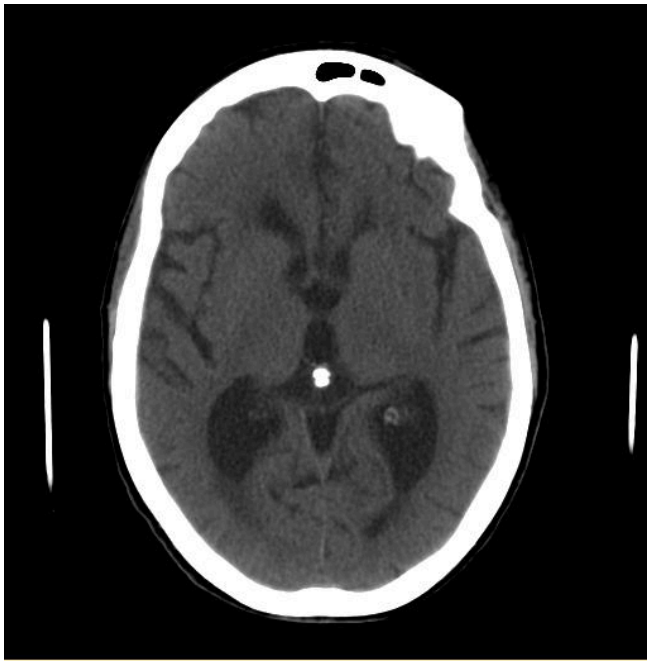
Bilateral Kayser-Fleischer rings on slit-lamp examination confirm copper in Descemet's membrane.

HYDROCEPHALUS PATTERN	IMAGING	THE DISCRIMINATOR
Normal pressure hydrocephalus (communicating)	Ventriculomegaly out of proportion to sulcal widening, with the disproportionately enlarged subarachnoid space pattern and periventricular transudation	**Tight high-convexity sulci with enlarged Sylvian fissures. Imaging alone does not establish shunt responsiveness**
Obstructive (non-communicating)	Selective proximal dilatation with normal distal calibre, for instance aqueductal stenosis dilating the lateral and third ventricles while the fourth stays normal	**A level of obstruction can be named, with periventricular capping of high signal around the frontal horns from transependymal seepage**
Ex vacuo	Passive ventricular enlargement secondary to atrophy	**Ventricular dilatation matches progressive cortical sulcal widening, without periventricular oedema or bowing of the corpus callosum**

The bedside confirmatory step before shunt referral is a large-volume cerebrospinal fluid tap test with gait assessment before and after drainage. The drainage volume and the response criteria are unsourced, as are the Evans index threshold and the callosal angle values, so describe the test and do not quote the thresholds.

Ventricular enlargement: state the missing view

Companion to original page 446



Brain CT, axial plane, from a source case of normal pressure hydrocephalus. Ventricular enlargement is visible. This slice alone does not show the high-convexity sulci needed for the full distinction from ex-vacuo enlargement. Relate ventricular size to the rest of the brain and the clinical history; ventricular enlargement alone is insufficient for the diagnosis.

State what further imaging is needed to distinguish communicating hydrocephalus from ex-vacuo enlargement.

RadsWiki (radswiki.net). Normal pressure hydrocephalus 001.jpg. CC BY-SA 3.0.
https://commons.wikimedia.org/wiki/File%3ANormal_pressure_hydrocephalus_001.jpg Licence: https://creativecommons.org/licenses/by-sa/3.0/
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INFECTION OR MASS	PATTERN	THE DISCRIMINATOR
Tuberculous meningitis	Thick gelatinous basal leptomeningeal enhancement filling the interpeduncular, suprasellar and Sylvian cisterns	**Basal, plus its complications: hydrocephalus and arteritis of lenticulostriate perforators producing basal ganglia infarcts**
Tuberculoma	Solitary or multiple nodular or ring-enhancing lesions with a solid caseating centre of low T2 signal and prominent surrounding oedema	**Larger, irregular, thick-walled, with profound oedema and caseous T2 hypointensity**
Neurocysticercosis	Lesions staged by cyst evolution: vesicular, a non-enhancing cyst with an eccentric hyperdense scolex; colloidal, ring enhancement with marked oedema; granular and calcified, non-oedematous calcified specks	**Thin walls with a recognisable eccentric scolex, the hole-with-dot appearance**
Cerebral toxoplasmosis	Multiple ring-enhancing abscesses centred on basal ganglia and the corticomedullary junction	**The eccentric target sign**
Primary central nervous system lymphoma	Periventricular or subependymal solid or ring-enhancing masses	**Dense hyperattenuation on the plain scan from high cellularity, with diffusion restriction**
Meningioma (extra-axial)	Sharply circumscribed, hyperattenuating or isodense unenhanced, with vivid homogeneous enhancement	**A broad dural base with an enhancing dural tail, inward buckling of cortical grey matter, and a preserved fluid-vascular cleft between mass and cortex**
High-grade glioma (intra-axial)	Arises in subcortical white matter with an irregular thick ring-enhancing wall, central necrosis, marked oedema and mass effect	**No fluid cleft and no dural attachment, and it invades across the corpus callosum as a butterfly glioma**

In Indian practice, treatment of central nervous system tuberculosis follows the national programme, which replaced category-based regimens with a standardised daily fixed-dose combination modified on drug-susceptibility testing, with the continuation phase extended for central nervous system disease and adjunctive systemic corticosteroids. Regimen durations are not sourced here, so name the structure and not the months.

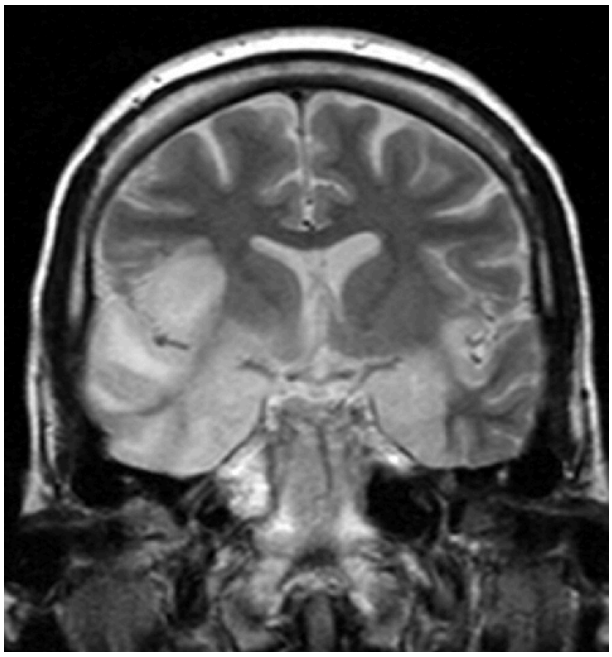
RULE In subacute trauma an **isodense** subdural collection hides in plain sight. Trace the cortical sulci out to the inner table: if the sulci do not reach the bone, there is an extra-axial collection whatever the density looks like.

HOLD THIS

Extradural is biconvex, respects sutures and crosses the falx; subdural is crescentic, crosses sutures and is stopped by the falx. Hyperdense, then isodense from three days to three weeks, then hypodense beyond.

Temporal change in herpes encephalitis

Companion to original page 447



MRI, coronal T2-weighted image. Temporal and insular high signal in the source confirmed herpes simplex encephalitis case. The source labels this sequence T2-weighted, not FLAIR. Signal abnormality must be interpreted with the other sequences and clinical or laboratory findings.

Describe the regional signal change before correlating it with the encephalopathic EEG topic.

dr Laughlin Dawes. Hsv encephalitis.jpg. CC BY 3.0. https://commons.wikimedia.org/wiki/File:Hsv_encephalitis.jpg Licence: https://creativecommons.org/licenses/by/3.0 Unmodified.

Ring enhancement: a mass, not a diagnosis

Companion to original page 447

Contrast-enhanced MRI, axial plane. Irregular ring-like enhancement surrounds a dark central necrotic region in the source glioblastoma case. The diagnosis belongs to the source case; ring enhancement alone is not specific for glioblastoma. The available provenance does not justify a more specific sequence label.

Describe the ring and centre, then use the chapter's differential rather than treating enhancement as an infection diagnosis.

The Armed Forces Institute of Pathology. AFIP-00405558-Glioblastoma-Radiology.jpg. Public domain. https://commons.wikimedia.org/wiki/File:AFIP-00405558-Glioblastoma-Radiology.jpg Licence: https://creativecommons.org/publicdomain/mark/1.0/ Unmodified.

WHAT EARNS THE MARKS

- **Shape and sutures together**: either feature on its own misclassifies the collection
- **The density clock**: hyperdense, then isodense, then hypodense, with the intervals
- **Sulci traced to the inner table**: the manoeuvre that finds an isodense collection
- **Panda face and bright claustrum**: the two signs worth drawing for the examiner

SEE ALSO 17 Naming the modality: CT, MRI, windows and sequences 18 Dementia imaging and the visual scales 20 Functional imaging: PET, SPECT, DaTscan, fMRI 24 Cerebrospinal fluid, neuroinfection and neurosyphilis

Laboratory monitoring and reports

The laboratory table runs two ways. Either you are asked what you would monitor on a drug and how often, which is a protocol question, or a report is put in front of you and you are asked to interpret it, which is a reasoning question.

Both reward the same habit: state the value, grade it, name the mechanism, then say what you would do before the next blood test.

22 · Lithium, clozapine and the metabolic schedule

LABORATORY MONITORING AND REPORTS · TOPIC 22

A patient is starting lithium. What would you check, and how often?

INVESTIGATIONS TABLE, LABORATORY STATION

THE SHAPE OF THE ANSWER

1	Baseline	The panel before the first dose, which candidates skip
2	Levels	Twelve-hour trough, the maintenance range, the intervals
3	Interpreting	Renal and thyroid results, and what a fall means
4	Clozapine	Counts, the revised threshold, and when surveillance stops
5	Metabolic	The schedule, and the cause of death to name

Baseline first, because it is the thing candidates skip. Before lithium, take six baselines.

- **Renal function.** Estimated glomerular filtration rate, blood urea nitrogen, serum creatinine and urinalysis, since the drug is excreted by the kidneys and a baseline is what lets you detect chronic decline.
- **Thyroid function.** Thyroid stimulating hormone with reflexive free thyroxine, because lithium inhibits thyroid hormone synthesis and release.
- **Serum calcium.** To unmask or monitor lithium-associated hyperparathyroidism.
- **The cardiometabolic baseline.** Weight, height, fasting glucose, glycated haemoglobin, lipid profile, liver function and full blood count.
- **Pulse, blood pressure and an electrocardiogram.** Where cardiovascular risk factors exist, or to exclude an arrhythmia or a Brugada pattern.
- **A pregnancy test.** In women of childbearing potential, given the teratogenic risk including cardiac malformation.

QUESTION	THE ANSWER
When is the level drawn	**As a 12-hour post-dose trough. For a once-daily evening dose, the following morning 12 hours later; for twice-daily dosing, 12 hours after the evening dose and immediately before the morning one. A sample drawn earlier captures the distribution phase and must be repeated**
Starting dose in adult practice	Modified-release lithium carbonate 200 to 600 mg once daily at bedtime, and 100 to 250 mg in frail or elderly patients. Bedtime dosing aids adherence, may reduce renal tubular exposure, and makes a standardised morning draw possible
During titration	Check at 7 days after initiation, then weekly until stable, and repeat 7 days after any change of dose, formulation or brand, because brands are not bioequivalent: 200 mg of lithium carbonate corresponds to 5.4 mmol of lithium, which matches 509 mg per 5 ml of lithium citrate liquid
Once stable	**Every 3 months through the first year. After that, at least every 3 months in higher-risk patients, and at least every 6 months in clinically stable low-risk patients**
Who counts as higher risk	Age 65 years or older, co-prescription of interacting medication, suboptimal adherence, unstable symptom control, a preceding plasma level of 0.8 mmol/L or higher, or pre-existing renal, thyroid or calcium abnormality
Target range	**Acute mania and lithium-naive bipolar maintenance 0.6 to 0.8 mmol/L, with 0.8 to 1.0 in selected refractory patients; unipolar depressive augmentation 0.4 to 0.6, with 0.6 to 0.8 in selected refractory depression; permissible maintenance range overall 0.4 to 1.0**
Other monitoring	Estimated glomerular filtration rate, thyroid stimulating hormone, serum calcium and weight every 6 months

Say which source you are quoting if the examiner offers a wider range: secondary manuals cite 0.8 to 1.2 mmol/L, while contemporary primary guidance narrows the target to 0.6 to 0.8 to limit long-term renal toxicity. Quote the primary guideline.

MEASURED LEVEL	CLINICAL PICTURE	ACTION
1.0 to 1.5 mmol/L	Asymptomatic, no signs of toxicity	**Look for a reversible cause: dehydration, a sample drawn under 12 hours post-dose, a new nonsteroidal or diuretic, a brand switch, a dosing error. Recheck at 24 hours because peak tissue distribution may lag by 1 to 2 days. With no reversible cause, reduce the dose, check renal function, encourage fluids and recheck in 5 to 7 days**
Above 1.5 mmol/L, or any clinical sign of toxicity	Early toxicity: coarse tremor, ataxia, nausea, vomiting, diarrhoea, drowsiness	**Withhold lithium immediately, assess mental state and hydration, repeat urgent level with urea, electrolytes and creatinine, and transfer for emergency medical assessment**
Above 2.0 mmol/L	Severe poisoning: convulsions, renal failure, hypotension, coma	**A medical emergency: urgent admission, intravenous hydration to promote diuresis and restore electrolytes, and preparation for haemodialysis**

Immediate medical evaluation is also indicated after an accidental acute ingestion of 50 mg per kg or more of lithium carbonate, or 100 mg per kg or more of lithium citrate. Where lithium is stopped deliberately, taper over at least 4 weeks and preferably over 3 months, with close relapse monitoring for 3 months and extended mood review for 2 years.

Interpreting the renal and thyroid results. Interactions raise the level by reducing clearance: thiazide diuretics, nonsteroidal anti-inflammatory drugs, angiotensin-converting enzyme inhibitors and angiotensin receptor blockers can increase serum lithium by up to 60 per cent, and metronidazole and tetracyclines also impair excretion, as do salt restriction and volume depletion through increased proximal tubular reabsorption.

An estimated filtration rate below 60 ml per minute is common in chronic therapy and does not by itself indicate progressive lithium nephropathy unless there is a continuous decline greater than 4 ml per minute per year, so track serial values.

If the rate declines across 2 or more consecutive tests, increase the frequency of level checks. A persistent rate below 60 with progressive decline, or any unprovoked rate below 30, requires screening for proteinuria and haematuria, cardiovascular assessment and nephrology referral.

New polyuria and polydipsia mandate urine osmolality testing for nephrogenic diabetes insipidus. A raised thyroid stimulating hormone with normal free thyroxine is subclinical hypothyroidism, which matters because it destabilises mood, and is managed by reducing the dose or adding levothyroxine after specialist discussion rather than by stopping lithium abruptly.

PARAMETER	TRADITIONAL RULE	GLOBAL CONSENSUS REVISION
Cessation threshold, general population	**Absolute neutrophil count below 1.5**	**Below 1.0, in units of 10 to the ninth cells per litre**
Cessation threshold in a person with the null variant of the Duffy antigen receptor	Not standardised in the older rules	**Below 0.5**
Twice-weekly monitoring trigger	Count below 2.0	Replaced by individualised clinical risk assessment
Duration of routine neutrophil surveillance	**Indefinite**	**Discontinue routine counts after 2 years, moving to comprehensive adverse drug reaction review every 3 months**

Why the surveillance can stop. The risk of clozapine-induced agranulocytosis is heavily front-loaded. Peak incidence of severe neutropenia is in the first month of exposure, reaching 0.9 per cent; weekly incidence peaks at 9 weeks at 0.128 per cent and falls to a rolling weekly average of 0.001 per cent by 2 years.

Cumulatively, 38 per cent of neutropenic events occur by 1 month, 56 per cent by 2 months, 84 per cent by 4.5 months, with Latin American data reporting 87.9 per cent within 18 weeks, 89 per cent by 12 months, 89 per cent by 24 months and 96 per cent by 36 months, with no further increase at 48 months.

The mean time to onset of acute severe neutropenia is 8.4 days, which reflects rapid marrow granulocyte arrest and is why monitoring less often than every 4 weeks has no utility for early detection.

On rechallenge, among 4,719 patients registered after cessation in a national non-rechallenge database, 1,296 (27 per cent) underwent rechallenge, and of 435 meeting revised criteria only 29 individuals (7 per cent) had a recurrent neutropenic event within 6 months.

TRAP The clozapine mortality paradox. Trainees answer this station entirely on agranulocytosis. **Gastrointestinal hypomotility is the leading cause of death** in patients taking clozapine, progressing through severe obstipation to functional ileus, bowel ischaemia and fatal perforation, so ask about bowel frequency, stool consistency and abdominal distension at every contact. Clozapine also carries the highest rate of pneumonia of all antipsychotics, frequently fatal, with aspiration from sedation and sialorrhoea a major pathway; the most severe metabolic burden of the commonly used agents; frequent tachycardia and postural hypotension; and sialorrhoea, nocturnal enuresis, daytime over-sedation, reflux and obstructive sleep apnoea to screen for. Long-term registries show a small excess of haematological malignancy against other antipsychotics, 61 against 41 cases per 100,000 person-years.

PARAMETER	BASELINE	4 AND 8 WEEKS	12 WEEKS	THEN
Personal and family history	Yes	No	No	**Annually**
Weight, height, body mass index, waist circumference	Yes	Yes	Yes	**Quarterly**
Blood pressure	Yes	No	Yes	**Annually**
Fasting plasma glucose	Yes	No	Yes	**Annually**
Fasting lipid profile	Yes	No	Yes	**Every 5 years, unless abnormal**

The consensus metabolic schedule for second-generation antipsychotics, with frequency increased if clinical status deteriorates or abnormalities emerge. In young populations up to 80 per cent experience notable weight gain, the majority of it within the first 12 weeks of exposure. The annual incidence of new type 2 diabetes is 0.4 per cent in paediatric recipients aged 10 to 18 years against 4 per cent or higher in adults. Real-world adherence to the schedule is poor: full monitoring rates of 4.7 per cent for body mass index, 6.5 per cent for fasting glucose and 0.8 per cent for total cholesterol, with partial rates of 44.8, 29.4 and 22.4 per cent, and clinicians monitor higher-risk drugs more closely, so that initiating olanzapine against aripiprazole carries more than fourfold higher odds of full body mass index monitoring and over fivefold for glucose.

ASK AND VERIFY The examiner will invite you to state therapeutic plasma levels for **sodium valproate and carbamazepine**, along with sampling times, toxic thresholds, auto-induction kinetics and the surveillance intervals for liver function, blood counts, ammonia and serum sodium. None of that is carried in the library's sources for this chapter. Say so explicitly, which demonstrates source discipline, and carry your own verified figures from a named formulary.

HOLD THIS

Twelve-hour trough, 0.6 to 0.8 for bipolar maintenance, every three months in the first year. A filtration rate below sixty is common; a fall of more than four per year is the finding that matters.

WHAT EARNS THE MARKS

- **Baseline before the first dose**: the omission the examiner is waiting to hear
- **Twelve hours, stated as such**: the sampling time is part of the number
- **Rate of fall, not one value**: a filtration rate is read as a trajectory
- **Hypomotility above agranulocytosis**: the leading cause of death on clozapine

SEE ALSO 23 Reading the report they hand you 25 Neuroendocrine tests and the status of biomarkers 26 Genetic testing and pharmacogenomics 27 Toxicology, urine screens and alcohol

23 · Reading the report they hand you

LABORATORY MONITORING AND REPORTS · TOPIC 23

Here is the report. What do you make of it?

INVESTIGATIONS TABLE, LABORATORY STATION

THE SHAPE OF THE ANSWER

1 **Grade first**	Say how abnormal the value is before explaining it
2 **A low sodium**	Two causes, separated on urine osmolality
3 **Creatine kinase**	The massive value without the syndrome around it
4 **Named traps**	Valproate and ammonia, folate and vitamin B12
5 **Provenance**	Name the owner of a reference interval that is not yours

Grade the abnormality before you explain it. Hyponatraemia is a serum sodium below 135 mmol/L, graded as mild at 130 to 135, moderate at 125 to 129 and severe below 125. Mild cases give non-specific headache, nausea, vomiting and dizziness; moderate to severe cases produce lethargy, confusion, delirium, seizures and coma.

In hospitalised cohorts admitted primarily for hyponatraemia the mean sodium was 121 milliequivalents per litre, with 77 per cent below 125.

PARAMETER	SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION	PSYCHOGENIC POLYDIPSIA
Volume status	Euvolaemic	Euvolaemic or hypervolaemic
Serum sodium	Below 135 mmol/L	Below 135 mmol/L
Serum osmolality	Low	Low
Urine osmolality	**Inappropriately concentrated, relatively high**	**Maximally dilute, low**

Urine osmolality is the discriminator candidates forget to ask for. The mechanism of the drug-induced form is stimulation of hypothalamic 5-HT2 and 5-HT2C receptors promoting antidiuretic hormone release, with serotonin-noradrenaline reuptake inhibitors additionally blocking the noradrenaline transporter, altering renal haemodynamics and potentiating the hormone. Absolute cut-offs for urine osmolality and urine sodium are unsourced here, so describe the direction rather than quoting a threshold.

Which antidepressants, and when. Antidepressant therapy carries an overall hyponatraemia event rate of 6.03 per cent and more than doubles the odds, with an odds ratio of 2.164.

Selective serotonin reuptake inhibitors give an event rate of 5.98 per cent with an odds ratio of 2.158, and serotonin-noradrenaline reuptake inhibitors 6.13 per cent with an odds ratio of 2.270; head to head, the latter carry higher risk for any hyponatraemia, odds ratio 1.271, and for clinically relevant hyponatraemia, odds ratio 1.291.

The highest-risk agents are fluoxetine among the first class, event rate 6.51 per cent and odds ratio 3.579, influenced by a long elimination half-life of 4 to 6 days and active metabolites, and venlafaxine among the second, event rate 5.66 per cent and odds ratio 2.683.

Paroxetine sits in the moderate to high band, having induced hyponatraemia in 12 per cent of elderly patients within a mean duration of 9 days. Lower-risk agents are sertraline, citalopram, duloxetine and mirtazapine, and switching to mirtazapine is frequently viable; tricyclics also have lower risk than either class.

The vulnerable groups are the elderly, women, who represent 80 per cent of hospitalisations among new users with a mean age of 76 years, patients of low body weight, and those on

concurrent thiazides or angiotensin-converting enzyme inhibitors, with ischaemic heart disease, diabetes, cerebrovascular disease, congestive heart failure and alcoholism adding vulnerability.

The critical window is within 2 to 4 weeks of starting, that is within about 14 to 30 days, and the risk falls substantially toward baseline non-user risk by 3 to 6 months, after some 85 to 180 days. In geriatric patients, measure sodium at baseline and check again at 1 to 2 weeks.

Mild asymptomatic cases are managed by fluid moderation or restriction, electrolyte monitoring and consideration of a lower-risk agent; severe symptomatic hyponatraemia below 125 mmol/L needs medical transfer, and the correction rate and the hypertonic saline regimen are unsourced here, so state the principle that over-rapid correction risks osmotic demyelination and take the rate from a named medical reference.

FEATURE	NEUROLEPTIC MALIGNANT SYNDROME	ISOLATED ANTIPSYCHOTIC-INDUCED ELEVATION OR RHABDOMYOLYSIS
Fever	**Present**	**Absent**
Muscle tone	**Severe lead-pipe rigidity**	**Normal tone, with myalgia possible**
Autonomic stability	Marked instability: tachycardia, labile pressure, diaphoresis	Vital signs stable
Mental state	Delirium, stupor, mutism, encephalopathy	Clear sensorium, or baseline psychosis
Creatine kinase	Markedly elevated	**Can reach massive levels: a reported case at 26,152 units per litre with a white cell count of 22.7 and an absolute neutrophil count of 16.6**

Massive creatine kinase, without the syndrome. Acute psychiatric excitement and physical restraint cause moderate elevations, and second-generation antipsychotics can cause substantial elevations without the full clinical picture; a comparison trial confirmed that the atypical agents produce higher elevations than conventional ones, and the interval from initiation to elevation spans from 5 days to 2 years.

So assess the cardinal features of the malignant syndrome first, hyperthermia, rigidity, altered sensorium and autonomic instability; if they are absent, this is isolated drug-induced rhabdomyolysis, remembering that a high enzyme level is itself a vulnerability for a subsequent malignant syndrome.

Manage it aggressively.

- **Stop or reduce the suspected agent.** For instance reducing ziprasidone from 240 to 160 mg per day in the reported case.
- **Hydrate.** Give vigorous intravenous hydration.
- **Monitor.** Monitor renal function and maintain urine output to protect against tubular necrosis.

Under hydration the level falls rapidly, from 26,152 to about 11,000 units per litre within a day or two and below 500 within the week.

REPORT	WHAT TO SAY	THE TRAP INSIDE IT
Liver function, transaminitis on valproate	Valproate, carbamazepine, phenothiazines and duloxetine frequently produce transient asymptomatic elevations. A rise of about three times the upper limit of normal with systemic symptoms, nausea, anorexia, right upper quadrant pain or jaundice, or with rising direct bilirubin, is drug-induced liver injury and the drug stops	**An aspartate to alanine aminotransferase ratio above two to one with a raised gamma-glutamyl transferase points to alcohol-related hepatocellular injury rather than psychotropic toxicity. The specific numeric cut-offs beyond the threefold figure are unsourced, so give the principle**
Serum ammonia, confusion on valproate	Valproate-induced hyperammonaemic encephalopathy: fluctuating lethargy, cognitive dulling, asterixis, ataxia or paradoxical worsening of seizures, through inhibition of carbamoyl phosphate synthetase I and mitochondrial carnitine depletion. Stop valproate, give L-carnitine, avoid protein loading	**Transaminases are characteristically normal or minimally raised, which is what misleads. Do not wait for an abnormal liver panel before checking ammonia. Reference ranges and the carnitine dose are unsourced here**
Renal function on long-term lithium	Urea, creatinine and estimated filtration rate, with nephrogenic diabetes insipidus presenting as polyuria and polydipsia with dilute urine through downregulation of aquaporin-2 channels. Keep single nightly dosing, avoid dehydration, remove nephrotoxic co-prescriptions, and involve a nephrologist as the rate falls	**Thiazides reduce lithium clearance and can precipitate toxicity. The action thresholds to quote are the sourced ones in the previous topic, not the ranges carried unsourced here**
Thyroid function on lithium	Lithium concentrates in the thyroid and inhibits hormone synthesis, iodotyrosine coupling and colloid release. A raised stimulating hormone with normal free thyroxine is subclinical hypothyroidism; with a reduced free thyroxine it is overt, and may present as fatigue, weight gain, constipation, depression or rapid cycling	**The action is to add levothyroxine, not to stop the lithium. Reference intervals and the target range for replacement are unsourced here**
Vitamin B12 and folate	Deficiency causes cognitive impairment, delirium, treatment-resistant depression, apathy, paranoia, peripheral neuropathy and subacute combined degeneration of the cord	**Giving folate alone to a patient with unrecognised B12 deficiency corrects the anaemia while the neurological degeneration progresses. Verify B12 status before or alongside folate. The numeric cut-offs are unsourced here**

REPORT	WHAT TO SAY	THE TRAP INSIDE IT
Serum prolactin	Dopamine D2 blockade in the tuberoinfundibular pathway removes tonic inhibition. Symptoms are galactorrhoea, amenorrhoea, gynaecomastia, sexual dysfunction and reduced bone mineral density, and the potent D2 antagonists are the usual agents	**Rule out pregnancy and primary hypothyroidism first, since thyrotropin-releasing hormone stimulates prolactin. Marked elevation warrants imaging of the sella to exclude a prolactinoma, and management is a prolactin-sparing agent or low-dose adjunctive partial agonism. The numeric bands and doses are unsourced here**

RULE Say the provenance out loud when a number is not yours to give. Several reference intervals in this territory, prolactin, vitamin B12, folate, ammonia, transaminases and thyroid, are **not carried in the library's sources**, so name the direction of the abnormality, the mechanism and the action, and offer to confirm the interval against the reporting laboratory's own range, which is the honest clinical answer anyway.

HOLD THIS

Urine osmolality separates the two causes of a low sodium; a massive creatine kinase without fever or rigidity is rhabdomyolysis, not the malignant syndrome. Confusion on valproate with normal transaminases means check the ammonia.

WHAT EARNS THE MARKS

- **Grading before explanation**: the sequence itself earns the mark
- **Urine osmolality**: one measurement separates the two low-sodium causes
- **Ammonia on valproate**: drowsiness with a normal liver panel is the indication
- **Vitamin B12 before folate**: replacing folate alone risks cord degeneration

SEE ALSO 22 Lithium, clozapine and the metabolic schedule 24 Cerebrospinal fluid, neuroinfection and neurosyphilis 25 Neuroendocrine tests and the status of biomarkers 27 Toxicology, urine screens and alcohol

24 · Cerebrospinal fluid, neuroinfection and neurosyphilis

LABORATORY MONITORING AND REPORTS · TOPIC 24

When would you tap, and how do you read this fluid report?

INVESTIGATIONS TABLE, LABORATORY STATION

THE SHAPE OF THE ANSWER

1	**Indication**	When you would tap, and what you exclude first
2	**The grid**	Appearance, cells, protein, glucose ratio, confirmation
3	**The ratio**	Why a simultaneous blood glucose is mandatory
4	**Tuberculous disease**	Why the microbiology fails you, and what follows
5	**Neurosyphilis**	Staging, general paresis, the test tiers, follow-up

Say when you would tap, and what you would exclude first. In psychiatric practice a lumbar puncture is indicated in acute organic brain syndromes, rapid-onset cognitive decline, late-onset psychosis, first-episode catatonia, unexplained delirium, refractory status epilepticus, or psychiatric symptoms with fever, headache, meningism, focal deficits or cranial neuropathies.

Contraindications at the bedside are local skin or soft tissue infection at the puncture site, severe coagulopathy or thrombocytopenia, and a space-occupying lesion with mass effect or obstructive hydrocephalus; fundoscopy or plain neuroimaging must precede the tap wherever focal deficits, papilloedema or impaired consciousness suggest impending herniation.

CONDITION	APPEARANCE	CELLS PER MICROLITRE AND DIFFERENTIAL	PROTEIN	GLUCOSE AND RATIO	CONFIRMATORY TEST
Normal	Clear, opening pressure 6 to 25 cm of water	Under 5, lymphocytes and monocytes	Under 40 mg/dL, that is 0.15 to 0.45 g/L	**At least 2.5 mmol/L or above 50 per cent of blood glucose, ratio about 0.6**	No organisms
Bacterial meningitis	Turbid, cloudy, purulent, pressure often above 20 to 30	100 to 10,000, often above 1000, neutrophil predominant	Above 1.0 g/L, often markedly raised	**Low, often below 2.2 mmol/L, ratio below 0.4**	Gram stain, culture, multiplex polymerase chain reaction
Tuberculous meningitis	Clear or slightly cloudy, with a fibrin cobweb clot on standing	50 to 1000, lymphocyte predominant, mixed early	1.0 to 5.0 g/L	**Low, ratio below 0.3 to 0.4**	Acid-fast staining, mycobacterial culture, rapid nucleic acid amplification, interferon-gamma on fluid
Viral meningitis or encephalitis	Clear, pressure normal or mildly raised	10 to 1000, usually under 250 to 500, lymphocyte predominant with neutrophils in the first 24 to 48 hours	Normal or mildly raised, usually under 1.0 to 1.5 g/L	**Usually normal, ratio above 0.5 to 0.6**	Viral polymerase chain reaction panels and viral IgM serology
Fungal meningitis	Clear or cloudy, pressure often raised in cryptococcal disease	20 to 500, may be low in immunocompromise, lymphocyte predominant	Raised, often 0.5 to 2.5 g/L	**Low or normal-low, ratio often below 0.5**	Cryptococcal antigen, India ink, fungal culture on a large-volume sample, fungal polymerase chain reaction
Autoimmune encephalitis	Clear	Mild lymphocytic pleocytosis or a normal count	Normal or mildly raised	**Normal**	Specific neuronal autoantibody panels in paired serum and fluid, with oligoclonal bands

The autoimmune row is carried in the library as unverified notebook material rather than sourced values, so describe the pattern as mild or normal and let the antibody panel carry the diagnosis.

Why a simultaneous blood glucose is mandatory. Glucose crosses the blood-fluid barrier by facilitated diffusion and therefore lags behind peripheral glycaemia, so severe systemic hypoglycaemia can produce an artificially low absolute fluid glucose with no infection at all. Interpret the ratio, normally about 0.6.

A ratio below 0.4 indicates active glycolytic consumption by proliferating organisms and infiltrating neutrophils, impaired hexose transport or heightened anaerobic metabolism, which is the bacterial pattern; below 0.3 to 0.4 is typical of tuberculous disease; below 0.5 is frequent in fungal disease; and above 0.5 to 0.6 with preserved utilisation is the viral pattern.

Tuberculous meningitis, and why the microbiology fails you. Five tests, five limitations.

- **Acid-fast smear.** Ziehl-Neelsen staining is notoriously insensitive, because the disease is paucibacillary.
- **Conventional culture.** Takes up to 8 weeks, and wants a high-volume tap of 30 mL or more for optimal recovery.
- **Conventional fluid polymerase chain reaction.** Sensitivity of only about 50 per cent.
- **Rapid automated nucleic acid amplification.** Detects the organism and rifampicin resistance within hours.
- **Interferon-gamma release assay.** On fluid it supports active central nervous system disease, whereas a positive serum assay reflects prior peripheral exposure without proving central infection.

Because delay increases morbidity and mortality, presumptive antitubercular therapy starts on strong clinical and biochemical suspicion without waiting for culture.

Neurosyphilis, staged. Following infection with the microaerophilic spirochete, about a third of individuals manifest clinical signs of systemic illness. Primary syphilis is a solitary painless chancre with regional lymphadenopathy appearing 10 days to 3 months after inoculation, and in 60 to 70 per cent that remains the only primary manifestation.

Secondary syphilis is haematogenous and lymphogenous dissemination with constitutional symptoms, generalised lymphadenopathy and maculopapular or pustular rash, and central seeding occurs early: the organism is recovered from cerebrospinal fluid in 30 per cent of secondary-stage patients and asymptomatic pleocytosis is identified in 40 per cent, while spontaneous clearance in most individuals means only 5 to 10 per cent develop late neurosyphilis.

Early syphilis is defined as duration up to 2 years by one authority, while European and American guidelines designate disease lasting 1 year or more as late.

Early neurosyphilis occurs months to a few years after infection as syphilitic meningitis with cranial neuropathies of the third, seventh, eighth or optic nerves, as uveitis, or as meningovascular disease causing cerebral infarction in young adults without vascular risk factors, with gummas as circumscribed meningeal granulomas.

Late neurosyphilis emerges years to decades later as tabes dorsalis, with posterior column and dorsal root degeneration giving sensory ataxia, lightning pains and autonomic dysfunction, and as general paresis.

General paresis, the general paralysis of the insane, is a chronic progressive meningoencephalitis that typically begins 10 to 30 years after primary infection, with cortical neuronal loss and gliosis producing six groups of features.

- **The cognitive picture.** Cognitive deficits, executive dysfunction and memory loss advancing to profound dementia.
- **Personality change.** Impaired judgement, emotional lability and social disinhibition.
- **Psychotic episodes.** The classically described grandiose and persecutory delusions and hallucinations.
- **Speech and tremor.** Dysarthria, tongue tremor described as trombone tongue, and facial tremor.
- **Pupils.** Pupillary irregularity including Argyll Robertson pupils, which accommodate but do not react to light.
- **Late features.** Seizures, upper motor neuron reflex changes, terminal incontinence and marasmus.

Atypical presentations can mirror temporal lobe encephalitis, autoimmune encephalitis or herpes simplex encephalitis, which is precisely why it belongs on a psychiatric differential.

TEST	WHAT IT MEASURES	THE LIMITATION THAT DECIDES ITS USE
Non-treponemal tests, VDRL and RPR	Cardiolipin-cholesterol-lecithin antibodies; serum titres fluctuate with disease activity and fall after successful therapy	**The rapid plasma reagin test cannot replace the VDRL test in cerebrospinal fluid**
Treponemal tests, TPHA, TPPA, TPLA, FTA-ABS	Antitreponemal antibodies directly	**High sensitivity but reactive for life regardless of cure, so unsuitable for monitoring response**
CSF-VDRL	The standard criterion for active disease is a reactive result with a raised cell count above 5 per microlitre and protein above 40 mg/dL	**Specificity is high, approaching complete in late disease, but sensitivity is only 30 to 85.7 per cent, so a non-reactive result does not exclude the diagnosis**
Intrathecal antibody index	Local central production of specific antibody, correcting for passive diffusion across a disrupted barrier	**High sensitivity, but raised values can persist for years after microbiological cure**
Treponemal polymerase chain reaction on fluid	Direct nucleic acid detection	**Sensitivity 31.7 to 63.7 per cent and specificity 39.9 to 98 per cent: not recommended to confirm or exclude**
CXCL13 chemokine	Significantly raised in neurosyphilis and falling rapidly after treatment	**Not recommended routinely: no large trials, and it rises in Lyme neuroborreliosis and central nervous system lymphoma**

The diagnostic tiers are worth reciting in order.

Probable neurosyphilis needs reactive serum TPHA or TPPA with reactive serum FTA-ABS, plus any two of the following.

- **Course.** A subacute or chronic progressive course of neurological or psychiatric symptoms with phases of deterioration and partial remission.
- **Fluid findings.** Pathological findings with mononuclear or mixed pleocytosis, barrier disruption shown by a raised albumin quotient, or an IgG-dominant intrathecal response.
- **Response to treatment.** Objective clinical improvement or resolution of fluid abnormalities after recommended antitreponemal therapy.

Proven adds demonstrated local intrathecal antibody production against the organism. *Confirmed* adds a reactive CSF-VDRL.

In a patient with human immunodeficiency virus infection and reactive treponemal serology, central invasion is more frequent, and a tap is pragmatically indicated if at least two of four criteria are met: a CD4 count of 200 cells per microlitre or below, untreated infection, a detectable viral load, or a serum VDRL titre above 1:64.

A tap is also indicated with neurological, psychiatric, cognitive, otological or ophthalmological symptoms, or where cardiovascular or gummatous tertiary disease is suspected.

Treatment and follow-up. First line is aqueous crystalline penicillin G intravenously, given as 4 doses of 6 million international units per day, or 5 doses of 5 million, or 3 doses of 10 million, equivalent to 3 to 4 million units every 4 hours, for 14 days with a minimum of 10 days.

Because high-dose parenteral penicillin lowers the seizure threshold, anticonvulsant prophylaxis is indicated where there is a seizure history or epileptiform discharges on the record.

Two alternatives, each with a caveat.

- **Ceftriaxone.** Intravenous ceftriaxone 2 g once daily for 14 days, noting that initial 4 g loading regimens lack supporting evidence.
- **Doxycycline.** Oral doxycycline 200 mg twice daily for 28 days, which achieves adequate penetration but is not endorsed by the major guidelines for want of randomised trials, and is contraindicated in pregnancy and in children up to 8 years because of dental discoloration.

The Jarisch-Herxheimer reaction is an acute self-limiting systemic inflammatory response to the release of treponemal endotoxins and lipoproteins on rapid lysis, emerging 12 to 24 hours after the first dose.

It is common in secondary syphilis but occurs in only 1 to 2 per cent of patients with neurosyphilis. It presents with high fever, rigors, headache, myalgia, tachycardia, transient pressure lability, leukocytosis and acute exacerbation of focal deficits or seizures.

Manage it with monitored bedside care and anti-inflammatory analgesia; the infusion is not interrupted, and routine prophylactic corticosteroids are not recommended.

Cure is not tracked on treponemal tests, which stay reactive. Repeat the tap at 3 to 6 months if the clinical response is favourable, documenting a fall in pleocytosis and protein, then every 6 months until the cell count normalises, which is expected within 2 years.

Serum IgM antitreponemal antibodies decline over 6 to 12 months and disappear within 18 months, and the CSF-VDRL titre usually falls by 3 to 4 dilutions within the first year, with conversion to non-reactive as definitive laboratory evidence of cure.

Retreat with an alternative intravenous regimen if pleocytosis fails to fall significantly by 6 months, if it persists at 2 years, or if a confirmed fourfold, that is two-dilution, rise occurs in non-treponemal titres in serum or fluid.

Successful therapy arrests infection without reversing established structural damage. Persistent sequelae affect 41.8 per cent of patients.

- **Cognitive.** Residual cognitive impairment in 28.8 per cent, including memory deficits in 10.1 per cent.
- **Motor and seizure.** Pyramidal damage in 18.6 per cent, seizures in 16.9 per cent and ataxia in 16.9 per cent.
- **Cranial nerve and the rest.** Cranial nerve deficits in 15.2 per cent, visual impairment in 11.8 per cent, and speech disturbance or hemiparesis in 6.7 per cent each.

TRAP Six that lose this station. Excluding neurosyphilis because the **CSF-VDRL is non-reactive**, when sensitivity is 30 to 85.7 per cent. Ordering a **treponemal test to document cure**, when the antibodies persist for life. **Delaying antitubercular therapy** while waiting for a culture that takes up to 8 weeks. Reading isolated mild pleocytosis in a patient with human immunodeficiency virus as proof of neurosyphilis, when the virus itself causes pleocytosis and barrier disruption. **Stopping penicillin** for a Jarisch-Herxheimer reaction, which is a lysis response and not an IgE-mediated anaphylaxis. And correcting a traumatic tap by the traditional one-white-cell-per-five-hundred-red-cells formula, which is unreliable: look instead for xanthochromia, which takes 6 to 12 hours to develop in true subarachnoid haemorrhage.

HOLD THIS

Interpret the glucose as a ratio, about 0.6 normally, below 0.4 in bacterial and below 0.3 to 0.4 in tuberculous disease. A negative fluid VDRL excludes nothing, and cure is measured on cells and non-treponemal titres.

WHAT EARNS THE MARKS

- **Glucose read as a ratio**: the absolute value on its own is uninterpretable
- **A non-reactive fluid VDRL excludes nothing**: the sensitivity figure makes the point for you
- **Treponemal tests cannot show cure**: antibodies persist, so follow cells and titres
- **Antituberculous treatment not delayed**: the microbiology is too slow to wait for

SEE ALSO 14 Encephalopathy, the named periodic patterns, and drugs
21 Spotters: blood, territories, hydrocephalus, infection 23 Reading the report they hand you
25 Neuroendocrine tests and the status of biomarkers

25 · Neuroendocrine tests and the status of biomarkers

LABORATORY MONITORING AND REPORTS · TOPIC 25

What does the dexamethasone suppression test interrogate, and is it still useful?

INVESTIGATIONS TABLE, LABORATORY STATION

THE SHAPE OF THE ANSWER

1 What it tests	Feedback on the axis, before any cut-off is quoted
2 The protocols	Dose, timing, sampling, threshold, indication
3 The numbers	Sensitivity, specificity, and why it failed as a test
4 Confounders	What manufactures a false positive result
5 The low-dose test	Hypersuppression, and the personality-disorder question

Say what the test interrogates before you quote a cut-off. The dexamethasone suppression test evaluates the functional integrity and negative feedback sensitivity of the hypothalamic-pituitary-adrenocortical axis.

Dexamethasone is a potent synthetic glucocorticoid acting principally at the anterior pituitary to suppress adrenocorticotropic hormone synthesis and release, so that reduced circulating hormone removes the drive to the adrenal cortex and suppresses endogenous cortisol the following morning.

The crucial physiological caveat is that peripherally administered dexamethasone does not achieve substantial glucocorticoid receptor occupancy within the human brain, so the response predominantly reflects *pituitary* receptor responsiveness and feedback sensitivity rather than central suprahypothalamic drive; when limbic mechanisms overdrive the axis, or when receptor desensitisation develops, suppression is attenuated and shorter, giving early escape or frank non-suppression.

PROTOCOL	DOSE AND TIMING	SAMPLING	THRESHOLD	INDICATION
Standard overnight test	1.0 mg orally between 11:00 PM and midnight	8:00 AM to 9:00 AM	**Serum cortisol above 5 micrograms per decilitre indicates non-suppression**	The historical marker for melancholic depression
Low-dose overnight test	0.5 mg orally at 11:00 PM	8:00 AM to 8:15 AM	**Continuous percentage suppression, with no fixed binary cut-off**	Detecting enhanced feedback sensitivity, that is hypersuppression, in post-traumatic stress disorder
Classic two-day low-dose test	0.5 mg orally every 6 hours for 8 doses, over 48 hours	2 to 6 hours after the eighth dose	**Cortisol above about 1.4 to 2.2 micrograms per decilitre**	Endocrine screening, separating Cushing syndrome from a pseudo-Cushing state
Overnight high-dose test	8 mg orally at 11:00 PM	8:00 AM to 9:00 AM	**Suppression of more than 50 to 80 per cent from baseline**	Separating pituitary Cushing disease from ectopic hormone secretion

Carroll's original protocol included additional afternoon and late-evening samples, but a morning draw is the standard baseline in subsequent research. Plasma dexamethasone is measured alongside serum cortisol, which is how a false non-

suppression from inadequate drug levels is detected.

The numbers, and why the test failed as a diagnostic test. In the psychiatric literature non-suppression in major depression was defined as a post-dexamethasone cortisol above 5 micrograms per decilitre, and on that threshold the 1.0 mg test showed non-suppression in about 40 per cent to 60 per cent of patients, most prominently in severe melancholic or psychotic presentations.

Endocrinology uses far stricter thresholds to screen for autonomous hypercortisolism, a post-suppression cut-off of 1.7, or 1.4 to 2.2, micrograms per decilitre, with values below 1.4 to 1.7 indicating preserved suppression.

In Cushing disease cohorts the 1.0 mg test carries an 8 per cent false-negative rate and a 30 per cent false-positive rate across normal controls, psychiatric illness, obesity and systemic medical conditions.

The test failed in psychiatry on both blades: inadequate sensitivity, leaving 40 to 60 per cent of depressive cases undetected, and poor specificity, since non-suppression is shared by mania, dementia, acute bereavement, severe weight loss, anorexia nervosa and general medical illness. It is also a static suppression challenge, assessing feedback shut-off rather than dynamic regulatory reserve.

The confounders that manufacture a false positive. Hepatic enzyme induction accelerates dexamethasone clearance so that pituitary concentrations are not reached: phenytoin, phenobarbitone, carbamazepine, rifampicin and chronic heavy alcohol intake, which is why plasma dexamethasone is measured.

Raised corticosteroid-binding globulin in high-oestrogen states, oral contraception and pregnancy, artificially elevates total plasma cortisol despite a normal unbound fraction, which is why late-night salivary cortisol or 24-hour urinary free cortisol are preferred in pregnancy. Acute emotional distress, sleep deprivation, physical trauma, obesity, acromegaly and Graves disease elevate the morning value.

In the other direction, reduced clearance in severe renal or hepatic impairment prolongs the half-life and produces misleading suppression.

PARAMETER	PSEUDO-CUSHING STATE	TRUE CUSHING SYNDROME
Clinical phenotype	Mild or absent cushingoid stigmata	Moderate to severe progressive signs: striae, myopathy, plethora
24-hour urinary free cortisol	Mildly elevated, under 3 times the upper limit of normal	Markedly elevated, above 3 times the upper limit
Overnight 1.0 mg test	**Variable**	**Variable, and it may also fail to suppress, which is why this test does not discriminate**
Two-day low-dose dexamethasone with corticotropin-releasing hormone	**Cortisol suppresses below 1.4 micrograms per decilitre, with the hormone given at 1 microgram per kilogram intravenously 2 to 6 hours after the last dose**	**Cortisol remains above 1.4 after stimulation**
Insulin tolerance test	Preserved hormone and cortisol rise during hypoglycaemia	Blunted or absent response to hypoglycaemia
Natural history	Spontaneous remission after psychiatric treatment or alcohol detoxification	Relentless progression requiring neurosurgical or adrenal intervention

Pseudo-Cushing is functional hypercortisolism mimicking the syndrome, secondary to depression, chronic alcohol dependence or severe obesity, driven by sustained hypothalamic overdrive of corticotropin-releasing hormone rather than autonomous tumour activity. In Indian de-addiction practice the alcohol-related form is common, and a few weeks of supervised abstinence normalises the axis, which spares the patient an invasive pituitary or adrenal work-up.

AXIS MEASURE	MAJOR DEPRESSIVE DISORDER	POST-TRAUMATIC STRESS DISORDER
Basal cortisol	Elevated, with increased 24-hour urinary free cortisol	**Lower, with decreased 24-hour urinary free cortisol**
Lymphocyte glucocorticoid receptor density	Decreased	**Increased**
Circadian pattern	Chaotic, lacking tight rhythmic control	**Tightly regulated, with an enhanced signal-to-noise ratio**
Feedback sensitivity	**Decreased: non-suppression on the 1.0 mg test**	**Enhanced: hypersuppression, detected on the 0.5 mg test**

Why the dose is halved. The 1.0 mg dose was designed to find *decreased* feedback sensitivity, and in healthy people it suppresses so completely that a floor effect hides heightened responsiveness. At 0.5 mg healthy individuals suppress only partially, so hypersuppression becomes visible, while that dose is insensitive to the decreased sensitivity of depression.

In a controlled study of 52 outpatients with personality disorders, 12 women and 40 men aged 19 to 60 years, those with comorbid post-traumatic stress disorder showed significantly greater adjusted mean percentage suppression after 0.5 mg, 83.6 per cent with a confidence interval of 67.7 to 99.4, against 63.0 per cent with an interval of 54.3 to 71.8, $p = 0.03$.

Morning post-dexamethasone cortisol was significantly lower in those with the disorder, 1.39 against 4.42 micrograms per decilitre, mirroring prior studies reporting 1.09 to 1.78 against 3.22 to 4.78 in controls.

Depression status had no significant effect on the low-dose test, 76.9 per cent suppression against 73.1 per cent, $p = 0.85$, confirming that the low-dose paradigm selectively detects enhanced sensitivity.

Borderline personality disorder has no independent signature. The early literature using the 1.0 mg test produced conflicting results because it examined depression and did not assess comorbid post-traumatic stress disorder. About 70 per cent of individuals with personality disorders report severe childhood trauma and about 30 per cent meet criteria for post-traumatic stress disorder.

In the controlled analysis, a diagnosis of borderline personality disorder had no significant main effect on cortisol suppression, $p = 0.16$, and no significant interaction with the stress disorder, $p = 0.64$, while controlling for the borderline diagnosis left the stress disorder significantly associated with enhanced suppression, $p = 0.006$.

So the variable patterns historically reported in borderline personality disorder reflect opposing comorbidities: melancholic depression driving non-suppression and post-traumatic stress disorder driving hypersuppression.

Glucocorticoid receptor antagonism. Severe depression with psychotic features shows robust axis hyperactivity and high rates of non-suppression, which was the rationale for mifepristone, a synthetic steroid acting at high concentrations as a competitive glucocorticoid receptor antagonist, with the aim of blocking excessive glucocorticoid action centrally and resetting feedback control. Open-label results looked promising; controlled trials did not follow.

In a multicentre trial of about 110 patients per group, 7 days of mifepristone followed by 21 days of treatment as usual significantly improved psychotic symptoms but failed to separate from placebo on depressive symptoms, and a subsequent phase III trial failed on every primary endpoint, confounded by a placebo response reaching 95 per cent in both arms.

Receptor antagonism remains an experimental concept rather than an approved therapy in mood disorders.

ASK AND VERIFY The natural follow-up to "why was the test abandoned" is whether it retains **prognostic or treatment-monitoring value**, specifically whether persistent non-suppression despite clinical improvement predicts early relapse and whether normalisation correlates with sustained remission. Neither claim is carried in the library's sources, so it is not asserted here. What is sourced is narrower: the loss of reliability, validity and specificity as a diagnostic instrument has not reduced the test's usefulness as a challenge modality for studying neuroendocrine-immune interaction.

Biomarkers, and the honest answer. The precedent is old: in 1906 Alzheimer correlated the presenile dementia of a patient who developed symptoms at age 51 and died at 55 with post-mortem plaques and tangles.

Despite advances in imaging, transcriptomics, proteomics and metabolomics, no clinically validated diagnostic biomarker exists for routine psychiatric practice, and diagnosis under the current manuals remains descriptive and syndromic, resting on history and mental state examination.

In depression the candidates are pro-inflammatory cytokines, C-reactive protein, neopterin, markers of oxidative stress, reduced brain-derived neurotrophic factor, and neuroendocrine and metabolomic predictors of rapid response to ketamine or scopolamine, all lacking individual diagnostic specificity and overlapping with ordinary stress, obesity, infection and medical disease.

In schizophrenia they are cerebrospinal protein expression clusters, peripheral gene expression profiling and markers of gestational hypoxia and placental oxidative stress, useful in developmental research and not diagnostic. In bipolar disorder, genetic risk profiles, volumetric markers and circadian transcriptomics cannot separate unipolar from bipolar depression at the point of care.

For suicidality, whole-blood gene expression panels validated in male cohorts are promising exploratory work without the sensitivity or specificity for bedside risk stratification.

CATEGORY	DEFINITION	WORKED EXAMPLE
State biomarker	Correlates with the acute episode or symptom severity and normalises in remission	**Acute cytokine elevation, and acute cortisol hypersecretion in active melancholia**
Trait biomarker	Persists independently of episode status, present premorbidly, during the episode and in euthymia, reflecting genetic vulnerability or enduring structural change	**Shortened rapid-eye-movement latency in depression**
Endophenotype	An intermediate biological phenotype between genomic variation and clinical syndrome, which must be heritable, trait-related, cosegregating with illness in families, and detectable in unaffected relatives at higher rates than in the general population	**Reduced P300 amplitude in the schizophrenia spectrum**
Research Domain Criteria	A research framework classifying mental disorders by functional neurobiological domains, such as negative valence, positive valence and cognitive systems, across levels of analysis from genes and molecules to circuits, physiology and behaviour	**It explicitly bypasses the descriptive diagnostic categories to seek biological validity**

TRAP Three. Reading non-suppression on the 1.0 mg test as proof of melancholia, when the false-positive rate is about 30 per cent in the medically ill, stressed or obese and the test misses 40 to 60 per cent of depressive cases. Confusing the two protocols: the standard dose looks for reduced suppression in depression, the low dose for enhanced suppression in post-traumatic stress disorder. And claiming that peripheral markers such as C-reactive protein, interleukin-6 or brain-derived neurotrophic factor can diagnose depression in clinic; they cannot, and saying so is the correct answer.

HOLD THIS

One milligram finds non-suppression in depression; half a milligram finds hypersuppression in post-traumatic stress disorder. Borderline personality disorder has no independent axis signature, and no psychiatric biomarker is clinically validated.

WHAT EARNS THE MARKS

- **Mechanism before the number**: what is being suppressed, and at which level
- **Two directions on one axis**: non-suppression against hypersuppression
- **The false-positive rate named**: the figure that ended its diagnostic career
- **No validated psychiatric biomarker**: the honest closing sentence of this station

SEE ALSO 15 Sleep staging, polysomnography and depression 22 Lithium, clozapine and the metabolic schedule 23 Reading the report they hand you 26 Genetic testing and pharmacogenomics

26 · Genetic testing and pharmacogenomics

LABORATORY MONITORING AND REPORTS · TOPIC 26

A child has intellectual disability. Which genetic test would you order first?

INVESTIGATIONS TABLE, LABORATORY STATION

THE SHAPE OF THE ANSWER

1 **The order**	Microarray first, karyotype for a recognisable syndrome
2 **Yields**	What each test detects, and its yield in each indication
3 **Tubes**	Purple top for the array, green top for the karyotype
4 **Fragile X**	The repeat bands, and when the test is indicated
5 **Pharmacogenomics**	The HLA alleles, the enzymes, the panel question

First-tier means first-tier, and the order is the answer. Intellectual disability has an estimated population prevalence of 3 per cent, and autism spectrum disorder a reported prevalence of 1 in 68 children, about 1.47 per cent, with an identifiable genetic aetiology in approximately 20 per cent to 25 per cent of autism.

Conventional G-banded karyotyping detects gross aneuploidy and large rearrangements and yields a diagnosis in only about 3 per cent of patients evaluated for an unexplained neurodevelopmental condition.

Chromosomal microarray analysis, by array comparative genomic hybridisation or single-nucleotide polymorphism platforms, detects submicroscopic copy-number variants and areas of homozygosity.

The published yield is 10 per cent to 20 per cent in unexplained non-dysmorphic intellectual disability or developmental delay, and 7 per cent to 10 per cent in autism. In clinical paediatric cohorts, pathogenic or likely pathogenic findings appear in 15 per cent to 29 per cent of isolated intellectual disability and 5 per cent to 9 per cent of autism.

Professional consensus therefore places microarray as the first-tier investigation, superseding karyotyping, which is reserved for clinically recognisable chromosomal syndromes or a suspected balanced translocation.

INVESTIGATION	WHAT IT DETECTS	YIELD IN INTELLECTUAL DISABILITY	YIELD IN AUTISM	TUBE
G-banded karyotype	Aneuploidy and large translocations	About 3 per cent	About 3 per cent	**Lithium heparin, green top, because viable dividing cells are needed for culture**
Chromosomal microarray	Submicroscopic copy-number variants, microdeletions and duplications, areas of homozygosity	10 to 20 per cent in the literature, 15 to 29 per cent clinically	7 to 10 per cent in the literature, 5 to 9 per cent clinically	**EDTA, purple top, because intact genomic DNA is needed and cell division is not**
Fragile X testing	CGG trinucleotide repeat expansion	About 2 per cent in the literature, 2.5 per cent clinically	0.5 to 2 per cent in the literature, and 0 per cent clinically in unselected cohorts	**EDTA, purple top**

Requisitioning a karyotype in an EDTA tube, or a microarray in a heparin tube, gets the sample rejected. That is a real viva question and a real laboratory failure.

Fragile X syndrome is the commonest inherited cause of intellectual disability, an X-linked disorder caused in 99 per cent of cases by an unstable CGG repeat expansion in the untranslated region of the FMR1 gene, leading to hypermethylation and transcriptional silencing.

The sourced repeat bands are the premutation at 55 to 200 repeats, carried by mothers of affected boys and associated with premature ovarian insufficiency and with fragile X-associated tremor and ataxia syndrome in maternal relatives, and the full mutation above 200 repeats.

The phenotype to look for falls into three groups.

- **Physical.** Relative macrocephaly, an elongated face, prominent ears with cupping of the upper pinnae, more discernible after puberty, joint hypermobility and post-pubertal macroorchidism.
- **Developmental and behavioural.** Developmental and speech delay ranging from mild articulation difficulty to absent expressive speech, hyperactivity and impulsivity, hand flapping, tactile defensiveness and autistic social communication deficits.
- **Pedigree clues.** Maternal intellectual disability, premature ovarian insufficiency, or tremor and ataxia in elderly maternal relatives.

Molecular confirmation historically used triplet-primed polymerase chain reaction with fragment analysis, with sensitivity above 99 per cent, and in modern services isolated repeat testing has been folded into broad sequencing pipelines, while imprinting disorders escape standard sequencing and need targeted methylation analysis and multiplex ligation-dependent probe amplification.

The population prevalence of males with a full mutation is about 1 in 4,000 to 1 in 7,000, and about 20 per cent of males with the syndrome meet criteria for autism.

The practical point: in unselected cohorts without physical features or family history the yield is 0 per cent in autism and 2 to 2.5 per cent in isolated intellectual disability.

A pre-test clinical checklist based on dysmorphic features and maternal pedigree reduces unnecessary requests by 60 to 80 per cent without missing true positives, which is why this is best rationalised as a targeted second-tier test.

ASK AND VERIFY Examiners ask candidates to place a borderline repeat count in one of four bands. Only two are sourced, the premutation at 55 to 200 and the full mutation above 200. The **normal upper limit and the intermediate or gray-zone band are unsourced** and are not printed here; confirm them against a cytogenetics or college reference before quoting.

CHARACTERISTIC	HLA-B*1502	HLA-A*3101
Phenotype	Severe cutaneous reactions: Stevens-Johnson syndrome and toxic epidermal necrolysis	Broad hypersensitivity: drug reaction with eosinophilia and systemic symptoms, maculopapular eruption, severe cutaneous reactions, hepatic injury
Ancestral distribution	**East and Southeast Asia 10 to 15 per cent, with a multi-ethnic Singapore frequency of 14.87 per cent; South Asia including India 2 to 4 per cent; below 1 per cent in Japan and Korea; rare in Europeans, among whom severe reactions occur in 1 to 6 per 10,000 new users**	**Present across both European and Asian populations, which is the trap**
Risk magnitude	About an 80-fold increase for carbamazepine-induced severe cutaneous reactions	A significant increase in systemic hypersensitivity reactions
If positive and drug-naive	**Do not prescribe carbamazepine**	**Do not prescribe carbamazepine unless no alternative exists; if none, monitor more frequently and stop at the first sign of rash**
If already treated for more than 3 months without reaction	**Continuation may cautiously be considered**	**Continuation may cautiously be considered**

The mechanism is class I presentation of drug antigens to cytotoxic T cells. The temporal rule is the one candidates get wrong: severe cutaneous reactions manifest almost exclusively within the initial months of exposure, so established tolerance past 3 months substantially reduces the likelihood of an acute allele-mediated reaction.

The other anticonvulsants. Oxcarbazepine carries an estimated 30-fold increased risk of severe cutaneous reaction in carriers, and testing is recommended by clinical consortia in Asian populations before prescribing, although regulatory mandates vary.

Lamotrigine carries only about a 2-fold increase, and because the baseline incidence of severe cutaneous reaction with lamotrigine is low, of the order of 1 in 3,000 to 1 in 6,000, a twofold elevation falls within baseline variability and does not change clinical decisions, so pre-treatment allele testing is not recommended; slow titration remains the risk mitigation.

Valproate has no actionable cytochrome P450 guideline, but POLG, the mitochondrial DNA polymerase gamma gene, must be tested before initiation where a hereditary neurometabolic condition is suspected, because of fatal hepatotoxicity, and screening for the urea cycle genes OTC and CPS1 is indicated if a urea cycle disorder is suspected, to prevent hyperammonaemic encephalopathy.

Lithium and pregabalin are eliminated unchanged by the kidney and have no hepatic pharmacogenomic interaction.

CLASS AND DRUG	ENZYME	CONSEQUENCE IN A POOR METABOLISER	GUIDANCE
Citalopram, escitalopram	CYP2C19	Raised levels with concentration-dependent QTc prolongation and torsades de pointes risk	**Reduce the maximum dose or choose an agent not reliant on this enzyme; in ultra-rapid metabolisers accelerated clearance gives low levels and non-response**
Sertraline	CYP2C19 with CYP2B6	Altered exposure	**Adjust the initial dose on clinical response**
Amitriptyline, clomipramine, imipramine, doxepin	CYP2C19 with CYP2D6	Raised parent tertiary amine levels with anticholinergic and cardiac toxicity, because demethylation to the active secondary amine is impaired	**Reduce the dose or monitor levels**
Clobazam, diazepam	CYP2C19	Prolonged elimination half-life and sedative duration	**Anticipate prolonged sedation**
Paroxetine, venlafaxine, fluvoxamine, vortioxetine, nortriptyline, desipramine	CYP2D6	Raised systemic exposure and side-effect burden; ultra-rapid metabolisers risk therapeutic failure	**Actionable guidelines exist. Agomelatine, bupropion, fluoxetine, mirtazapine and trazodone have none**
Aripiprazole, brexpiprazole, haloperidol, pimozide, risperidone, zuclopenthixol	CYP2D6	Higher plasma levels with extrapyramidal symptoms, excessive sedation and QTc prolongation	**Actionable guidance for these 6 antipsychotics; pimozide labelling requires genotyping before higher doses because of fatal arrhythmia risk. Clozapine and olanzapine have no actionable guidance for this enzyme, and quetiapine has guidance for CYP3A4 rather than CYP2D6**
Atomoxetine	CYP2D6	Peak plasma concentrations about 10-fold higher than in extensive metabolisers, with severe sedation and tachycardia against a baseline sedation rate of about 1 in 20 patients	**Start at a standard or reduced dose and titrate slowly with close haemodynamic monitoring**

Ancestral variation is pronounced: Asian populations show lower baseline activity of both enzymes than Caucasian populations, and sequencing in Asian cohorts finds actionable phenotypes in 51.0 to 77.2 per cent for CYP2C19 and 31.1 to 47.2 per cent for CYP2D6.

Commercial combinatorial panels. These analyse pharmacokinetic genes together with candidate pharmacodynamic genes using proprietary, non-transparent algorithms to produce traffic-light prescribing recommendations.

The trial evidence in depression is negative or inconclusive on primary outcomes.

- **One Canadian trial.** No significant difference in 8-week response and remission between guided care and treatment as usual.
- **A larger trial.** The per-protocol analysis found no significant difference in symptom reduction at 8 weeks, with modest secondary findings.
- **A veterans trial.** Reduced prescriptions of drugs with predicted gene interactions, but showed no significant difference in remission or response at 24 weeks.
- **Two further trials.** One of them in Han Chinese patients, showing no significant differences at 8 weeks.

International consensus is therefore that routine pre-emptive testing and direct-to-consumer panels should not be used in standard practice, with testing reserved for documented treatment resistance after multiple adequate first-line trials, unusually severe or unexpected adverse reactions at standard or sub-therapeutic doses, and pre-treatment screening for specific established risks, the carbamazepine allele and the valproate polymerase gene.

TRAP Four. Ordering **fragile X testing reflexively in unselected autism**, where the yield is 0 to 2 per cent against 7 to 10 per cent for microarray. Generalising the carbamazepine allele warning to **lamotrigine**. **Stopping carbamazepine** in a patient who has tolerated it for more than 3 months and is found incidentally positive. And assuming the allele epidemiology is interchangeable: one allele is concentrated in Asian populations, the other spans European and Asian populations alike.

HOLD THIS

Microarray first, purple top; karyotype only for a recognisable syndrome, green top. Eighty-fold for carbamazepine, thirty-fold for oxcarbazepine, twofold and not worth testing for lamotrigine.

WHAT EARNS THE MARKS

- **First-tier named as first-tier**: the sequence of tests is itself the answer
- **Yield figures attached**: each test carries its yield in its own indication
- **Allele to drug, not to class**: the carbamazepine warning does not generalise
- **Tube colours given**: a small detail the examiner uses to test familiarity

SEE ALSO 22 Lithium, clozapine and the metabolic schedule 25 Neuroendocrine tests and the status of biomarkers 27 Toxicology, urine screens and alcohol

27 · Toxicology, urine screens and alcohol

LABORATORY MONITORING AND REPORTS · TOPIC 27

The urine screen is positive. How would you interpret it?

INVESTIGATIONS TABLE, LABORATORY STATION

THE SHAPE OF THE ANSWER

1 **Validate**	Temperature, creatinine and pH, before any interpretation
2 **Screen against confirm**	Immunoassay presumptive, mass spectrometry confirmatory
3 **Windows**	The detection windows, and the cross-reacting agents
4 **Cannabinoids**	The myths, with the numbers that settle them
5 **Alcohol**	Conversions, clinical bands, specimen handling

Validate the specimen before you interpret it. Urine is the commonest matrix because collection is non-invasive and metabolites persist at higher concentrations for longer than in blood, but before any result is interpreted the specimen must be shown not to have been tampered with, substituted or diluted.

Four objective parameters, documented on receipt.

- **Temperature.** Between 90 and 100 degrees Fahrenheit measured within 4 minutes of voiding, where an out-of-range value indicates synthetic urine, added liquid or substitution.
- **Acidity.** A pH between 4.5 and 8, where below 3 or above 11 indicates adulteration with bleach, vinegar or detergent.
- **Specific gravity.** Between 1.002 and 1.030, where a value below 1.002 suggests pre-collection water diuresis or dilution.
- **Creatinine.** Above 20 mg/dL, which is what distinguishes physiologically dilute urine from substituted fluid.

Collection requires 20 to 50 mL, typically 25 mL. Tetrahydrozoline eye drops added to a sample can directly produce a false-negative cannabinoid result.

SUBSTANCE	TYPICAL URINE DETECTION WINDOW
Unmetabolised ethanol	7 to 12 hours
Ethyl glucuronide and ethyl sulfate	**3 to 5 days, at a urinary cut-off of 100 ng/mL or above, which is why they are used to verify abstinence**
Amphetamine and methamphetamine	48 hours
Barbiturates	**24 hours for short-acting agents, up to 3 weeks for phenobarbital**
Benzodiazepines	**Up to 3 days for short-acting agents, up to 30 days for diazepam**
Cocaine metabolite, benzoylecgonine	2 to 4 days

SUBSTANCE	TYPICAL URINE DETECTION WINDOW
Cannabis metabolite	**3 days after single use, 3 to 7 days in some series; 5 to 7 days with moderate use; 10 to 15 days with daily heavy use; more than 30 days with chronic long-term heavy use**
Synthetic cannabinoids	72 hours acutely, longer with chronic use
Morphine, codeine, heroin as morphine	48 to 72 hours
Hydromorphone, oxycodone	2 to 4 days
Methadone	3 days
Phencyclidine	8 days

Windows vary with lipophilicity, route, volume of distribution, hepatic metabolism, urinary pH and pattern of use. Alternative matrices: saliva detects ethanol for 6 to 12 hours, sweat patches track excretion from 24 hours before placement across 7 to 10 days of wear, and hair ethyl glucuronide on a proximal segment reflects consumption over months.

Screening against confirmation. Initial screening uses immunoassays with antibodies directed at broad drug classes or selected metabolites, and they behave like a lock that related chemical structures can enter without turning, which is cross-reactivity, so every immunoassay result is presumptive and preliminary.

Confirmation uses gas chromatography with mass spectrometry, or liquid chromatography with tandem mass spectrometry, which fragments molecules into mass-to-charge fingerprints for definitive identification and quantification.

Standardised workplace cut-offs exist to prevent false positives from trace environmental exposure or food: cannabis metabolites screen at 50 ng/mL and confirm at 15; cocaine metabolites screen at 150 and confirm at 100; opiates screen and confirm at 2000; the heroin-specific metabolite 6-acetylmorphine at 10; phencyclidine at 25; and amphetamines screen at 500 and confirm at 250 each.

The federal opiate cut-off was raised in 1998 from 300 to 2000 ng/mL specifically to eliminate false positives from dietary poppy seed. Clinical addiction programmes frequently choose lower thresholds to detect non-adherence or low-level relapse.

SCREEN TARGET	COMMON CROSS-REACTING AGENTS
Amphetamines	**Bupropion, aripiprazole, trazodone, fluoxetine, desipramine, chlorpromazine, thioridazine, promethazine, atomoxetine, selegiline, amantadine, labetalol, ranitidine, metformin, ofloxacin, pseudoephedrine, phenylephrine, brompheniramine**
Benzodiazepines	**Sertraline, efavirenz, oxaprozin**

SCREEN TARGET	COMMON CROSS-REACTING AGENTS
Opioids	**Quetiapine, amisulpride, sulpiride, tramadol, tapentadol, dextromethorphan, diphenhydramine, ofloxacin, levofloxacin, rifampicin, verapamil, quinine, poppy seed**
Methadone	**Quetiapine, chlorpromazine, clomipramine, thioridazine, diphenhydramine, doxylamine, verapamil**
Buprenorphine	**Amisulpride, sulpiride, tramadol**
Phencyclidine	**Venlafaxine, lamotrigine, tramadol, ketamine, dextromethorphan, diphenhydramine, doxylamine, ibuprofen, imipramine, meperidine, thioridazine**
Tricyclic antidepressants	**Quetiapine and cyclobenzaprine on urine screens; carbamazepine, cyproheptadine, diphenhydramine and hydroxyzine on serum assays**
Barbiturates	**Ibuprofen, naproxen**
Cocaine	**Coca leaf tea**

The cannabinoid myths, with the numbers that settle them. Screening targets the inactive metabolite at a cut-off of 50 ng/mL. Efavirenz at 600 mg daily produces high rates of false-positive results, in one report 28 of 30 patients, through cross-reactivity from its major urinary glucuronide metabolite.

Oral pharmaceutical delta-9-tetrahydrocannabinol produces presumptive positives in 30 to 45 per cent of users. Nonsteroidal anti-inflammatory interference, though historically cited, is rare with modern reformulated assays: in clinical trials acute ibuprofen at 1200 mg in one day and chronic naproxen at 1000 mg daily for 30 days each yielded a single cross-reaction among 102 participants.

Proton pump inhibitor labelling carries a warning, but volunteer trials show no measurable interference on rapid test strips. Certain commercial infant cleansers added to a sample produce dose-dependent false positives. Passive inhalation in confined spaces rarely exceeds 20 ng/mL, observed in only 2 of 80 urine samples, and so fails to breach the 50 ng/mL cut-off.

And daily ingestion of hemp seed oil yields peak urinary metabolite concentrations of about 5.2 ng/mL, below both the screening and the confirmation cut-offs.

CONVERSION OR PARAMETER	VALUE
Percentage to mass	**A concentration of 0.05 per cent weight by volume equals 0.05 g/dL, that is 50 mg/dL**
Serum report to blood concentration	**Shift the decimal three places left: a serum level of 100 mg/dL equals 0.10 g/dL. Serum or plasma runs about 1.2 times higher than whole blood**

CONVERSION OR PARAMETER	VALUE
SI conversion	1 mmol/L of blood ethanol equals 4.61 mg/dL
Absorption	Small intestine primarily, stomach secondarily; peak at 30 minutes fasting, delayed up to 3 hours by food
Elimination	**More than 90 per cent by hepatic oxidation, zero-order at intoxicating concentrations, clearing at 10 to 30 mg/dL per hour, about one standard drink an hour**
One standard drink	12 ounces of beer, 5 ounces of wine or 1.5 ounces of 80-proof spirits, raising the level by 15 to 25 mg/dL
Breath to blood ratio	**2100 to 1: 2100 mL of expired alveolar air contains the ethanol of 1 mL of blood, measured on end-expiratory air after deep inspiration**
Urine kinetics	Peaks 45 to 60 minutes after ingestion, and during elimination runs about 1.3 to 1.5 times higher than blood; an uncapped container loses 10 to 25 per cent of ethanol per hour
Tolerance marker	**Functioning without overt intoxication above 150 mg/dL demonstrates substantial neuroadaptation and strongly indicates chronic dependence**

Women reach higher peak levels than men for an equivalent dose, through a higher percentage of body fat, lower total body water and lower gastric alcohol dehydrogenase activity. Blood testing remains clinically reliable within 6 to 12 hours of the last drink.

The clinical bands, remembering that symptoms overlap and vary with tolerance and rate of drinking. From 10 to 50 mg/dL, mild relaxation, loss of shyness and subtle euphoria without overt incoordination.

From 50 to 90, reduced alertness, impaired judgement, divided-attention deficits and lengthened reaction time, with measurable driving impairment from about 70 mg/dL and flushing, reduced peripheral vision and blunted reflexes above 80. From 100 to 150, evident central depression with slurred speech, ataxia, gross motor impairment and emotional lability as euphoria gives way to dysphoria and nausea.

From 160 to 250, severe confusion, prominent dysphoria, vomiting, inability to walk unassisted, and anterograde amnesia becoming likely. From 250 to 300, severe impairment of almost all brain functions with little physical control, emotional numbing, reduced pain responsiveness, vomiting with a greatly increased chance of asphyxiation, and loss of consciousness listed at 300.

From 300 to 350, stupor in those still conscious with little comprehension of place or activity, and reported deaths. From 350 to 400, the level of surgical anaesthesia, possible coma, and respiratory and heart rate slowing toward arrest. Above 400, coma with death possible from respiratory depression or arrest.

Specimen handling, which is where forensic cases are lost. Disinfect the venepuncture site with a non-alcoholic antiseptic, aqueous povidone-iodine or benzalkonium chloride. Use tubes containing sodium fluoride, grey top, which inhibit glycolysis and microbial fermentation, keep them airtight to prevent evaporation of a volatile analyte, and separate serum within 4 hours on unpreserved specimens.

On breath testing, alveolar air concentration is directly proportional to pulmonary arterial blood concentration, and artifactual elevations arise from residual mouth alcohol, for instance ethanol-containing mouthwash, or from raised breath ketones in diabetic or starvation ketoacidosis.

On urine, glucosuric urine contaminated with Candida species can generate ethanol in vitro through bladder fermentation, which is distinguished by a negative blood ethanol with absent urinary ethyl glucuronide and sulfate.

TRAP Four. Reporting a **serum** figure as a blood alcohol concentration, when serum runs about 1.2 times higher, so a serum report of 96 mg/dL reflects a whole blood level of about 80. Assuming a standard **opiate** screen covers the semisynthetic and synthetic opioids: it detects morphine, codeine and the heroin metabolite, and misses oxycodone, hydromorphone, buprenorphine, fentanyl and methadone unless targeted assays are ordered. Accepting a negative **benzodiazepine** screen in a clonazepam or lorazepam overdose, since kits are calibrated against oxazepam or nordiazepam while these are excreted as glucuronide conjugates at low free concentrations. And accepting a passive-exposure defence, which does not survive confirmatory testing.

ASK AND VERIFY Two items here are unsourced and are not printed. **Widmark's formula** and its body-water distribution factor for men and women, which the examiner asks for when estimating expected concentration from an ingested dose or back-extrapolating to an earlier time; what is sourced for that arithmetic is the elimination rate of 10 to 30 mg/dL per hour and the rise of 15 to 25 mg/dL per standard drink, so a back-calculation is elapsed time multiplied by the elimination rate. And the **Indian statutory driving limit** under the Motor Vehicles Act: name the Act and the section from the Bare Act, and do not quote the threshold from a clinical reference.

HOLD THIS

Validate the specimen, treat every immunoassay as presumptive, confirm by mass spectrometry. Breath to blood is 2100 to 1, serum is 1.2 times whole blood, and a comfortable patient above 150 mg/dL is tolerant, not sober.

WHAT EARNS THE MARKS

- **Presumptive until confirmed**: the sentence that protects the report you sign
- **Serum against whole blood**: the conversion factor, and what it does to a figure
- **Opiate is not opioid**: the standard screen misses the synthetic agents
- **Comfort at a high level is tolerance**: the clinical inference, not a claim of sobriety

SEE ALSO 22 Lithium, clozapine and the metabolic schedule 23 Reading the report they hand you 26 Genetic testing and pharmacogenomics

Quick review

The discriminators of this note in one table	
METHOD	Form carries the diagnosis, content carries the biography. Primary means psychologically irreducible and non-understandable, not first in time. Understanding grasps meaning from within, explaining establishes causes from without.
DELUSION AND ITS NEIGHBOURS	Jaspers: conviction, incorrigibility, impossibility, and those are the outside of it. Delusion and overvalued idea are ego-syntonic and unresisted; the obsession is ego-dystonic and resisted. Overvalued idea is an idealised value fused with identity.
PERCEPTION	Pseudohallucination sits in inner subjective space and is involuntary; true hallucination sits in external space with full conviction. Functional is same-modality and simultaneous, reflex is cross-modal, extracampine is outside the sensory field and diagnostically neutral. Pareidolia grows with attention; completion and affect illusions dissolve.
THOUGHT AND SPEECH	Circumstantiality arrives, tangentiality does not. Poverty of speech is quantity, poverty of content is information. Verbigeration has no trigger; palilalia repeats the patient's own terminal words with falling volume. Bizarre proverb responses carry the specificity, literal ones do not.
POSSESSION AND FIRST RANK	Eleven symptoms in the ICD-10 list, seven of them ego-boundary disorders. Not pathognomonic, and their absence in a new psychosis should send you looking for an organic cause. Somatic passivity needs the sensation plus deliberate external infliction.
CATATONIA	Three signs of twelve on DSM-5, two of fourteen on the screening scale. Catalepsy is placed, posturing is assumed; mitgehen yields to light touch, gegenhalten resists proportionally. Challenge dose then a fifty per cent fall. Antipsychotics are withheld.
CONSCIOUSNESS AND MEMORY	Arousal from the reticular activating system, content from cortex. Organic amnesia keeps identity and loses new learning; dissociative amnesia does the reverse and violates Ribot's law. Confabulation needs amnesia plus a frontal deficit. Approximate answers show the question was understood.
EMOTION AND SELF	Mood is sustained and reported, affect is cross-sectional and observed. Euphoria, elation, exaltation, ecstasy. Anhedonia is hedonic capacity, apathy is motivation. Reality testing separates depersonalisation from passivity: "as if" against a delusion of control.
INSIGHT	Three dimensions: awareness, relabelling, treatment need. Six bedside grades, with grade V intellectual and grade VI emotional. Insight and symptoms are semi-independent. Rising insight needs active screening for depression and suicidality.

The discriminators of this note in one table	
CULTURE AND EPONYMS	Dhat, possession, ascetic, gilhari and koro in a Delhi clinic series, and depression is the commonest comorbidity. Capgras is the same face with a changed identity, Fregoli a changed face with the same identity. Lesion-related misidentification is right hemisphere in 92 per cent and right frontal in 63 per cent.
SUICIDE IN INDIA	Rate 12.3 per 100,000 with 171,418 deaths, hanging 60.9 per cent, poisoning 25.0 per cent, two thirds aged 18 to 45, two thirds earning under a lakh. Section 115 presumes severe stress and bars trial; Section 309 was not repealed.
EEG NORMAL	Alpha 8 to 13 Hz posteriorly, reactive, asymmetry under 1 Hz. Bipolar montages localise by phase reversal, referential by maximal amplitude. Input 1 more negative deflects upward. A spike without background disruption or an aftercoming slow wave is probably a benign variant.
EEG ABNORMAL	Spike under 70 milliseconds, sharp wave 70 to 200. Regular 3 Hz on a normal background is typical absence; under 2.5 Hz with an abnormal background is not. Hypsarrhythmia above 200 microvolts with chaotic multifocal spikes. Prion periodicity 1 to 2 seconds, panencephalitis 4 to 10.
SLEEP	Four stages since 2007. Latency 70 to 90 minutes, cycles about 90 minutes, N2 about half the night. Depression shortens REM latency as a trait and raises REM density as a state; narcolepsy reaches REM within 15 minutes.
EVOKED POTENTIALS	Both schizophrenia and bipolar disorder prolong the P300 latency; only schizophrenia flattens the amplitude. P50 is sensory gating on scalp recording, prepulse inhibition is sensorimotor gating on facial electromyography, and they do not correlate.
IMAGING VOCABULARY	Attenuation and density for one modality, signal intensity for the other. Fluid dark on T1, bright on T2, suppressed on FLAIR; white matter brighter than grey on T1 and darker on FLAIR. Bright diffusion needs a dark coefficient map.
DEMENTIA IMAGING	Medial temporal atrophy scored on coronal sections at the anterior pons: 2 or more abnormal under 75 years, 3 or more at 75 and above, and the score stops discriminating above 85. Small vessel disease mediates 30 to 42 per cent of the age effect. Parietal and precuneal loss points to Alzheimer disease.
PSYCHIATRIC IMAGING	Grey matter falls, white matter and total intracranial volume hold, fluid space expands. Ventricular enlargement is non-specific and unproven before onset. Imaging excludes an organic cause and does not make the diagnosis.
FUNCTIONAL IMAGING	Posterior temporoparietal for Alzheimer disease, frontal and anterior temporal for frontotemporal dementia, occipital for Lewy body disease. Transporter imaging separates neurodegenerative parkinsonism from essential tremor and not the atypical syndromes from idiopathic disease.

The discriminators of this note in one table	
SPOTTERS	Extradural biconvex, respects sutures, crosses the falx; subdural crescentic, crosses sutures, stopped by the falx. Hyperdense, then isodense from three days to three weeks, then hypodense. Panda face in the midbrain, bright claustrum lateral to the putamen.
LITHIUM AND CLOZAPINE	Twelve-hour trough, 0.6 to 0.8 mmol/L for bipolar maintenance, three-monthly in the first year. A filtration rate below 60 is common; a fall above 4 per year matters. Neutrophil threshold revised to 1.0, routine counts stopped after 2 years, and hypomotility kills more often than agranulocytosis.
READING REPORTS	Urine osmolality separates the two low-sodium causes. A massive creatine kinase with neither fever nor rigidity is rhabdomyolysis. Drowsiness on valproate with a normal liver panel is an indication for serum ammonia. Folate given without checking B12 risks cord degeneration.
FLUID AND NEUROSYPHILIS	Interpret glucose as a ratio, about 0.6 normally, below 0.4 bacterial, below 0.3 to 0.4 tuberculous. A non-reactive fluid VDRL excludes nothing; treponemal tests cannot show cure. General paresis begins 10 to 30 years after infection.
NEUROENDOCRINE AND GENETICS	One milligram finds non-suppression in depression, half a milligram hypersuppression in post-traumatic stress disorder, and no psychiatric biomarker is validated. Microarray first in purple, karyotype in green; eighty-fold for carbamazepine, twofold for lamotrigine.
TOXICOLOGY	Validate the specimen, treat the immunoassay as presumptive, confirm by mass spectrometry. Breath to blood 2100 to 1, serum 1.2 times whole blood, elimination 10 to 30 mg/dL per hour, and comfort above 150 mg/dL is tolerance.
UNSOURCED FIGURES	Named here rather than guessed: Schneider's second-rank list, Cotard's stages and folie a deux's definition, the hallucinosis definition and the command-hallucination risk step, the Korsakoff eponym, the Glasgow Coma Scale, the statutory sections for modified electroconvulsive therapy and for capacity and admission, Hounsfield anchors, the Evans index and callosal angle, prolactin, vitamin B12, folate, ammonia, transaminase and thyroid reference ranges, valproate and carbamazepine levels, the fragile X normal and gray-zone bands, Widmark's formula, and the statutory driving limit. Carry your own verified figure, or say you will verify it.

HOLD THIS, LAST

Both tables reward the same three-beat answer: name the phenomenon in its own vocabulary, define it in one sentence, then separate it from the thing it is most often confused with. Where the number is not yours to give, say whose it is.

PART VII

VII

Viva Tables II: Psychopharmacology and Somatic Treatments

The drug table: mechanism, dose, level, monitoring, adverse effect, interaction, switch; then ECT, neuromodulation and the emergencies.

01 Antipsychotics and clozapine

02 Mood stabilisers

03 Antidepressants and treatment resistance

04 Anxiolytics, stimulants and cognition

05 Addiction pharmacology

06 Adjuncts, special populations, interactions and basics

07 ECT and neuromodulation

08 Emergencies and new drugs

Viva Tables II: Psychopharmacology and Somatic Treatments

Eight sections · 41 topics · one complete notes document

A drug table is not a recitation. The examiner names a drug or a syndrome and listens for one discriminating line: the mechanism that explains the adverse effect, the level that decides the next step, the monitoring interval that shows you have prescribed this before.

Answer in the order the table is built. Class, mechanism, dose, level, monitoring, adverse effect, interaction, special population, switch. Every grid below runs in that order. The **hit** cell is the discriminator: cover the rest of the row and rebuild it from that cell alone.

Where a figure a candidate is expected to quote is absent from the sourced material, the question is printed as a trap with the words *verify before the exam* and no number, because a wrong milligram is a factual error an examiner can act on.

Antipsychotics and clozapine

1 · Classification and the four dopamine tracts

ANTIPSYCHOTICS · TOPIC 01

Classify the antipsychotics for me, and then tell me what D2 blockade does in each dopamine tract.

PSYCHOPHARMACOLOGY VIVA TABLE | LONG CASE DRUG REVIEW

THE SHAPE OF THE ANSWER

1 **Three classifications**	Generational, chemical, mode of action. Name all three first.
2 **Four tracts**	One benefit, three prices, each with its own adverse effect.
3 **Occupancy numbers**	Response near 65%, movement risk from 78% upward.
4 **What atypicality means**	Affinity ratio plus dose, not a clean receptor dichotomy.

Name three classifications before naming any drug.

- **Generational.** First-generation agents are defined by epoch and potent D2 antagonism (haloperidol, chlorpromazine, trifluoperazine, fluphenazine, thioridazine, flupenthixol, zuclopenthixol). Second-generation agents are defined clinically by lower extrapyramidal and prolactin liability (clozapine, risperidone, olanzapine, quetiapine, ziprasidone, asenapine, lurasidone, paliperidone, amisulpride). Third-generation agents are D2 and D3 partial agonists with 5-HT1A partial agonism (aripiprazole, brexpiprazole, cariprazine).
- **Chemical.** Tricyclics, that is phenothiazines by side chain, thioxanthenes, and the seven-membered "epines", against non-tricyclics, that is butyrophenones, diphenylbutylpiperidines, substituted benzamides, benzisoxazoles, benzisothiazoles and phenylpiperazines.
- **Neuroscience-based Nomenclature.** By mode of action, so haloperidol and amisulpride are D2 antagonists, olanzapine adds 5-HT2, clozapine and risperidone add alpha-2, quetiapine adds noradrenaline reuptake inhibition through norquetiapine, and aripiprazole with cariprazine are D2 and 5-HT1A partial agonists.

TRACT	PROJECTION	EFFECT OF D2 BLOCKADE
Mesolimbic	Ventral tegmental area to ventral striatum	**Therapeutic: positive symptoms fall. Cost: reward blunting, secondary apathy**
Mesocortical	Ventral tegmental area to prefrontal cortex	**Already hypodopaminergic: worsens secondary negative symptoms and cognition**
Nigrostriatal	Substantia nigra to caudate and putamen	**Disinhibits cholinergic interneurons: dystonia, parkinsonism, akathisia, tardive dyskinesia**
Tuberoinfundibular	Hypothalamus to pituitary stalk	**Disinhibits lactotrophs: galactorrhoea, amenorrhoea, gynaecomastia. Spared by clozapine, quetiapine, aripiprazole**

Occupancy ties the table together. Response needs approximately 65% striatal D2 occupancy, and extrapyramidal risk escalates sharply at 78% to 80%.

- **The dose makes the atypical.** Risperidone makes the point: 2 mg/day gives roughly 65% occupancy, and 6 mg/day pushes toward 80%. Atypicality is therefore partly a dosing artefact.
- **Pharmacological definition.** Higher normalised affinity for 5-HT2A than D2, because striatal 5-HT2A blockade disinhibits local dopamine and displaces excess D2 blockade while mesolimbic blockade is kept.

RULE **Receptor breadth does not buy efficacy.** Across 35 antipsychotics, breadth showed no significant correlation with effect size for positive or negative symptoms, and the best agents include the broadest (clozapine) and the most selective (amisulpride).

TRAP Calling chlorprothixene, loxapine and pipamperone typical on receptor grounds. All three show 5-HT2A affinity exceeding D2, so the typical and atypical dichotomy does not map cleanly onto receptor profiles.

HOLD THIS

Mesolimbic blockade is the benefit; the other three tracts are the price. Cortex costs cognition, nigrostriatum costs movement, the tuberoinfundibular tract costs prolactin.

WHAT EARNS THE MARKS

- **Three classifications named before any drug**: it shows you hold a framework rather than a drug list.
- **The tract bound to its adverse effect**: mesocortical to cognition, nigrostriatal to movement, tuberoinfundibular to prolactin.
- **The two occupancy figures**: 65% for response and 78% to 80% for extrapyramidal risk convert the table into a dosing decision.
- **Atypicality stated as a ratio plus a dose**: the candidate who says the dichotomy is imperfect is ahead of the one who defends it.

SEE ALSO 02 Potency, equivalents and the percentage method 03 Extrapyramidal syndromes and tardive dyskinesia 04 Prolactin, metabolic risk, QTc, anticholinergic load

2 · Potency, equivalents and the percentage method

ANTIPSYCHOTICS · TOPIC 02

This patient is on two antipsychotics. Is this high-dose therapy, and how did you decide?

LONG CASE PRESCRIPTION AUDIT | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1 **Define potency**	Milligrams to effect, against efficacy as the ceiling.
2 **Quote equivalents**	The chlorpromazine 100 mg benchmark set, then its limits.
3 **Use the percentage method**	Each drug as a share of its own maximum, then sum.
4 **Show the arithmetic**	Include the as-required doses in the sum.
5 **Governance**	Who signs, what is documented, what is monitored.

Potency is a milligram statement, efficacy a ceiling statement. Maximal antipsychotic response is comparable across the potency range.

- **High potency.** Haloperidol, trifluoperazine, fluphenazine and pimozide bind D2 tightly, are dosed at 2 mg to 20 mg/day, and carry high movement liability with little off-target load.
- **Low potency.** Chlorpromazine, thioridazine, promazine and levomepromazine are dosed at 100 mg to 1000 mg/day and carry sedation, orthostatic hypotension and antimuscarinic toxicity with little acute extrapyramidal risk.
- **Chlorpromazine equivalents, the benchmark set.** Against 100 mg of chlorpromazine: haloperidol 2 mg, fluphenazine 2 mg, pimozide 2 mg, thiothixene 4 mg, trifluoperazine 5 mg, perphenazine 10 mg, loxapine 10 mg, thioridazine 100 mg. On the same convention a high-potency agent needs under 5 mg to match, and a low-potency agent above 40 mg.

Why equivalence is retired. It assumes one uniform mechanism of striatal blockade, which fails for newer agents; occupancy saturates rather than rising linearly; and sensitivity shifts, with more resistance in acute psychosis and more receptor sensitivity as symptoms remit.

- **The replacement.** The percentage method: express each drug as a percentage of its own licensed maximum and sum the percentages. High-dose therapy means a single drug above its licensed maximum, or concurrent drugs whose summed percentages cross the licensed ceiling.

WORKED EXAMPLE	ARITHMETIC	VERDICT
Oral combination	Clozapine 700 mg of a 900 mg maximum is 78%; amisulpride 400 mg of 1200 mg is 33%	**Sum 111%, high-dose therapy**
Oral plus as-required	Clozapine 400 mg of 900 mg is 44%; haloperidol 5 mg thrice, that is 15 mg of 20 mg, is 75%	**Sum 119%, high-dose therapy**

Denominators worth carrying: amisulpride 1200 mg, aripiprazole 30 mg, cariprazine 6 mg, chlorpromazine 1000 mg, clozapine 900 mg, flupentixol 18 mg, haloperidol 20 mg, lurasidone 148 mg, olanzapine 20 mg, paliperidone 12 mg, quetiapine 750 mg (800 mg in mania or modified release), risperidone 16 mg, sulpiride 2400 mg, trifluoperazine 50 mg, zuclopenthixol 150 mg.

TRAP Omitting the as-required prescription from the sum. Discretionary doses prescribed without auditing the cumulative percentage are the commonest route into inadvertent high-dose therapy, and the arithmetic above is the sourced example.

Governance, because the examiner asks who signs. The consultant decides, the indication is documented, consent is taken, escalation uses titration intervals of at least 1 week, and the regimen is reviewed at least every 3 months and continued only on documented objective benefit.

- **Cardiac monitoring.** Baseline electrocardiography is mandatory with cardiology review if the QTc exceeds 440 ms, repeated at steady state after each increment and then every 6 to 12 months.
- **The rest of the schedule.** Urea, electrolytes and liver function every 6 months; blood pressure lying and standing, pulse and temperature every 3 months; and rating scales at weeks 0, 6 and 12 and then annually.

HOLD THIS

Quote the percentage method, not a conversion ratio. Haloperidol is more potent than chlorpromazine and no more efficacious.

WHAT EARNS THE MARKS

- **Potency separated from efficacy in one sentence**: the commonest muddle in this table.
- **The percentage method offered before the ratio**: it is the current standard and the ratio is the retired one.
- **The as-required dose counted in the sum**: this is the arithmetic that turns a legal prescription into high-dose therapy.
- **Named governance steps**: consultant decision, documented indication, consent, review at 3 months.

SEE ALSO 01 Classification and the four dopamine tracts 04 Prolactin, metabolic risk, QTc, anticholinergic load 38 Rapid tranquillisation and acute agitation

3 · Extrapyramidal syndromes and tardive dyskinesia

ANTIPSYCHOTICS · TOPIC 03

This man became restless three weeks after starting haloperidol. What is it, and how would you treat it?

SHORT CASE, MOVEMENT EXAMINATION | OSCE STATION ON ADVERSE EFFECTS

THE SHAPE OF THE ANSWER

1 Timing first	Four syndromes, four onset windows, hours to years.
2 Name the scale	Simpson-Angus, Barnes, Abnormal Involuntary Movement Scale.
3 Treat by syndrome	Anticholinergic helps two, does nothing for one, harms one.
4 Exclude the emergency	Rigidity with fever and autonomic change is a different answer.
5 Rechallenge	Wait, correct the risks, choose a low-liability agent.

Four syndromes, four onset windows. Lead with timing and the rest follows. Liability is highest with high-potency first-generation agents, lowest with clozapine and quetiapine, and dose-dependent with risperidone and paliperidone above 6 mg/day.

SYNDROME	ONSET	FEATURES AND PREVALENCE	SCALE	MANAGEMENT
Acute dystonia	**Hours (heuristic 3 hours), minutes if parenteral**	Torticollis, oculogyric crisis, retrocollis, trismus, laryngeal spasm; about 10%	General scales	Parenteral anticholinergic; response in 5 minutes intravenously, 20 minutes intramuscularly
Pseudoparkinsonism	**Days to weeks (heuristic 3 days)**	Cogwheel rigidity, pill-rolling tremor, bradykinesia, masked facies; about 20%	Simpson-Angus	Reduce dose, switch agent, trihexyphenidyl or amantadine, review every 3 months
Akathisia	**Days to months (heuristic 3 months)**	Subjective inner restlessness with pacing and rocking; about 25% with first-generation drugs	Barnes	Reduce dose, switch, propranolol, benzodiazepine. Anticholinergics do not help alone
Tardive dyskinesia	**Months to years (heuristic 3 years)**	Choreoathetoid tongue, lip and limb movements; 3% to 5% a year with first-generation, 0.5% to 1% with second-generation drugs	Abnormal Involuntary Movement Scale	Stop the anticholinergic, switch to clozapine, VMAT2 inhibitor

COMMON TRAP

Akathisia read as worsening psychosis: the dose goes up, the akathisia worsens and suicide risk rises with it. The discriminator is subjective, a distressing inner restlessness felt mainly in the legs, against delusion-driven agitation without limb-focused tension. A trial of low-dose propranolol separates them.

Neuroleptic malignant syndrome. The tetrad is generalised lead-pipe rigidity, unexplained hyperthermia, autonomic instability and fluctuating altered sensorium.

- **Markers.** Creatine kinase often above 1000 units/L, leucocytosis, myoglobinuria and deranged transaminases.
- **Risk profile.** High-potency agents, rapid dose change either way, abrupt anticholinergic withdrawal, polypharmacy, dehydration, agitation and being a young man.

Management, in sequence. Stop all antipsychotics, transfer to intensive care, hydrate to keep urine output above 100 mL/hour against myoglobinuric tubular necrosis, cool externally, correct electrolytes and acidosis.

- **Then the drugs.** Dantrolene 1 to 2.5 mg/kg intravenously up to 10 mg/kg/day, bromocriptine 2.5 mg every 8 hours titrated to 15 to 20 mg/day, and lorazepam 1 to 2 mg for agitation. ECT rescues where drugs fail to arrest progression within 48 to 72 hours.

- **Rechallenge.** Wait at least 5 days and preferably 2 weeks after resolution and normalisation of creatine kinase, correct modifiable risks, avoid high-potency agents and depots, and select quetiapine, clozapine or aripiprazole titrated slowly.

TRAP The starting dose for a post-crisis rechallenge is a figure candidates volunteer and the sources do not carry. Give the principle, below the usual initial dose with very slow titration, and **verify before the exam**.

MNEMONIC

HOURS, DAYS, MONTHS, YEARS

Dystonia in hours, pseudoparkinsonism in days, akathisia by months, tardive dyskinesia after years, each attached to its scale: general, Simpson-Angus, Barnes, Abnormal Involuntary Movement Scale. Crude, and it gets four syndromes out in the right order under pressure.

HOLD THIS

Anticholinergics rescue dystonia and parkinsonism, do nothing for akathisia, and worsen tardive dyskinesia. One class, three answers, decided by which syndrome is in front of you.

WHAT EARNS THE MARKS

- **Onset window quoted before the treatment**: timing is what separates the four syndromes at the bedside.
- **Akathisia distinguished from psychotic agitation**: the subjective leg-focused restlessness, and the propranolol trial.
- **The anticholinergic rule stated three ways**: rescue, useless, harmful, according to syndrome.
- **Urine output target and dantrolene dose**: they show the malignant syndrome has been managed, not just recognised.

SEE ALSO 04 Prolactin, metabolic risk, QTc, anticholinergic load 27 Anticholinergics, propranolol and the burden scale 41 New and pipeline drugs

4 · Prolactin, metabolic risk, QTc, anticholinergic load

ANTIPSYCHOTICS · TOPIC 04

She has galactorrhoea on risperidone and has gained eight kilograms. What do you do, and what do you check first?

LONG CASE, ADVERSE EFFECT REVIEW | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1 Rank the drugs	Highest to neutral, for each of the four liabilities.
2 Exclude the mimics	Pregnancy, hypothyroidism, renal failure, prolactinoma.
3 Monitoring schedule	Baseline, 4 and 8 weeks, 12 weeks, then quarterly.
4 Act on a threshold	Switch at 5% to 7% gain; QTc bands decide the cardiac step.
5 Name the switch	Aripiprazole, and say why from the receptor profile.

Rank the drugs before describing the effect.

- **Prolactin ranking.** Highest with haloperidol, fluphenazine, trifluoperazine, chlorpromazine, risperidone and paliperidone, the last two sustained and dose-dependent; moderate to low with asenapine,

iloperidone, ziprasidone, lurasidone and cariprazine; minimal with clozapine and olanzapine; absent with quetiapine; and aripiprazole is neutral or lowering.

- **Consequences.** Galactorrhoea, oligomenorrhoea, amenorrhoea and infertility in women, gynaecomastia, erectile dysfunction and oligospermia in men.
- **Steps, in order.** Exclude pregnancy, hypothyroidism, renal failure and prolactinoma; monitor if asymptomatic; reduce dose; switch to aripiprazole or quetiapine; or add low-dose aripiprazole where switching risks decompensation. Dopamine agonists are avoided because they risk exacerbating psychosis.

METABOLIC TIER	DRUGS	WEIGHT, GLUCOSE, LIPIDS
High	Clozapine, olanzapine	**Severe; 56% on olanzapine gained more than 7% of body weight in CATIE; ketoacidosis risk; triglyceride rise averaging 40.5 mg/dL**
Moderate	Quetiapine, risperidone, paliperidone	**Moderate; 14% on risperidone gained more than 7%; moderate insulin resistance and lipid rise**
Low	Aripiprazole, ziprasidone, lurasidone, asenapine, cariprazine, brexpiprazole	**Minimal weight gain, low glucose effect, neutral lipids**

Monitoring: baseline history, weight, body mass index, waist, blood pressure, fasting glucose and lipids; weight and body mass index at 4 weeks and 8 weeks; at 12 weeks add blood pressure, glucose and lipids; then quarterly weight and glucose with annual waist, pressure and lipids.

Switch once gain exceeds 5% to 7% of baseline. Lurasidone needs a meal of at least 350 kcal, ziprasidone at least 500 kcal.

QTC	ACTION
Under 440 ms in men, 470 ms in women	Proceed; repeat annually if indicated
Abnormal but under 500 ms	**Correct potassium, calcium and magnesium (potassium under 3.5 mmol/L especially), stop non-essential prolonging drugs, reduce dose or switch to aripiprazole, repeat and refer if still abnormal**
Above 500 ms	**Stop the drug; telemetry if syncope has occurred; urgent cardiology referral**
High-risk patient	Repeat at steady state, about 5 half-lives, then monthly for 6 months and every 6 to 12 months

Risk ranking: strong for pimozide, haloperidol (particularly intravenous), chlorpromazine, thioridazine and high-dose therapy; moderate for quetiapine and amisulpride; low for clozapine, olanzapine, risperidone, paliperidone, asenapine, fluphenazine, flupentixol, sulpiride; none at therapeutic dose for aripiprazole, lurasidone, zuclopenthixol. Borderline sits at 430 ms to 450 ms, prolonged above 450 ms.

Anticholinergic load. Low-potency phenothiazines, clozapine, olanzapine and quetiapine block muscarinic receptors potently; high-potency first-generation drugs, aripiprazole and lurasidone barely at all.

- **Peripheral picture.** Dry mouth, blurred vision, urinary hesitancy or retention, constipation, reduced bronchial secretion, sinus tachycardia.

- **Central picture.** Impaired concentration and recall, confusion, restlessness, visual hallucination and frank delirium, with the elderly and the organically impaired most vulnerable.

RULE **Aripiprazole is the pivot in three of these four tables.** Prolactin-neutral, metabolically low, negligible QTc effect, minimal anticholinergic affinity. When asked what to switch to, that is usually the answer, and the reason is the receptor profile.

HOLD THIS

Risperidone for prolactin, clozapine and olanzapine for metabolism, pimozide and thioridazine for QTc, clozapine again for anticholinergic load. Four tables, four worst offenders.

WHAT EARNS THE MARKS

- **A ranked list, not a single drug name**: the table is asking for gradation across the class.
- **The mimics excluded before the drug is blamed**: pregnancy and hypothyroidism are the two that cost candidates the mark.
- **A number attached to the action**: 5% to 7% weight gain triggers the switch, 500 ms stops the drug.
- **Aripiprazole justified from its receptor profile**: prolactin-neutral, metabolically low, negligible QTc, minimal muscarinic affinity.

SEE ALSO 03 Extrapyramidal syndromes and tardive dyskinesia 27 Anticholinergics, propranolol and the burden scale 32 Children, adolescents and the elderly

5 · Clozapine: workup and ancestry-adjusted titration

ANTIPSYCHOTICS · TOPIC 05

You have decided on clozapine. Take me through the workup and the first two weeks of titration.

LONG CASE MANAGEMENT PLAN | OSCE STATION ON CLOZAPINE INITIATION

THE SHAPE OF THE ANSWER

1 Justify the indication	Define resistance by trials, dose, duration, adherence.
2 Baseline workup	Four domains: physical, haematology, cardiac, metabolic.
3 Titrate by ancestry	Start 12.5 mg, target 175 to 300 mg on this subcontinent.
4 Levels and metabolism	Trough 350 to 600 ng/mL, CYP1A2, smoking and caffeine.
5 Consent and access	Capacity documented, and the tertiary-tier supply problem.

Clozapine is examined as a process. Indications are treatment-resistant schizophrenia and reduction of recurrent suicidal behaviour in schizophrenia or schizoaffective disorder, with refractory tardive dyskinesia and persistent aggression added in Indian practice.

- **Resistance, defined.** Persistent positive symptoms despite at least 2 adequate trials of different antipsychotics at sufficient dose and duration with documented adherence. It affects about 20% of patients, and only 10% to 20% of eligible candidates receive the drug.

BASELINE DOMAIN	DOCUMENTED BEFORE THE FIRST DOSE
Physical	Pulse, sitting and standing blood pressure, temperature, respiratory rate, weight, waist
Haematology	**Full blood count with neutrophil count, at least 1500/mm3, or at least 1000/mm3 in benign ethnic neutropenia, plus blood group**
Cardiac	**Electrocardiogram, echocardiogram for baseline ejection fraction, troponin, C-reactive protein**
Metabolic	Fasting glucose, lipids, liver function, renal parameters, electrolytes

POINT	INDIAN OR EASTERN ASIAN ANCESTRY	WESTERN LABEL
Start	**12.5 mg at bedtime**	12.5 mg once or twice daily
End of week 1	50 mg daily, increments of 6.25 mg to 25 mg	100 mg daily, increments of 25 mg to 50 mg
End of week 2	100 mg daily	200 mg daily, targeting 300 to 450 mg
Target	**175 to 300 mg daily**	300 to 600 mg daily, label maximum 900 mg

Eastern Asian ancestry, spanning Pakistan through East Asia and including the subcontinent, carries lower baseline CYP1A2 activity, so equivalent concentrations are reached on about half the Western dose. Geriatric patients target 12.5 to 200 mg daily.

Levels and metabolism. Trough above 350 ng/mL gives the most robust response; the reference range is 350 to 600 ng/mL; above 600 ng/mL seizure liability rises without therapeutic gain.

- **Which enzyme.** Metabolism is mainly CYP1A2 with minor CYP2C19, CYP2D6 and CYP3A4 contributions. Smoke induces CYP1A2 and drives levels down, so abrupt cessation or heavy caffeine raises them. Routine pharmacogenomic testing is not recommended.

Contraindications and cautions. The labelled contraindication is hypersensitivity.

- **Boxed warnings to screen and document.** Severe neutropenia; orthostatic hypotension, bradycardia and syncope; seizures; myocarditis, pericarditis, cardiomyopathy and mitral incompetence; and increased mortality in elderly patients with dementia-related psychosis.
- **Labelled precautions.** Eosinophilia, QT prolongation, metabolic change, neuroleptic malignant syndrome, hepatotoxicity, fever, pulmonary embolism, anticholinergic toxicity and reduced gastrointestinal motility.

TRAP The classic package-insert list a viva expects, that is myeloproliferative disorder or severe marrow depression, paralytic ileus, uncontrolled epilepsy, severe central depression or coma, and concurrent marrow-suppressant drugs, is not carried by the sourced material. Quote it from the current Indian product insert and **verify before the exam**.

INDIAN PRACTICE NOTE

Capacity is assessed and consent documented under the Mental Healthcare Act 2017, through the voluntary route or the supported admission provisions; the numbered section is not carried by the sourced material, so name the route rather than the number.

On the essential medicines list clozapine sits at the tertiary tier only, which is exactly the access problem for district hospital psychiatrists.

HOLD THIS

Start at 12.5 mg, target 175 to 300 mg, keep the trough between 350 and 600 ng/mL. Half the Western dose, because of half the CYP1A2 activity.

WHAT EARNS THE MARKS

- **Resistance defined with four components**: two trials, adequate dose, adequate duration, documented adherence.
- **Baseline echocardiogram and troponin offered unprompted**: it shows you expect myocarditis, not only agranulocytosis.
- **The ancestry-adjusted target**: 175 to 300 mg, on roughly half the Western dose, with CYP1A2 as the reason.
- **Consent route named rather than a section number**: precision about what the sourced material carries is itself a mark.

SEE ALSO 06 Clozapine hazards and rechallenge 28 Interactions: CYP450, smoking, grapefruit, binding 33 Essential psychotropics in India

6 · Clozapine hazards and rechallenge

ANTIPSYCHOTICS · TOPIC 06

Three weeks into clozapine he is tachycardic and breathless, and the blood count is normal. What is happening?

SHORT CASE, CLOZAPINE COMPLICATION | EMERGENCY VIVA TABLE

THE SHAPE OF THE ANSWER

1 **Name five hazards**	Neutropenia, myocarditis, bowel, seizures, sialorrhoea.
2 **Read the count**	Four bands, four actions, with the neutropenia thresholds.
3 **Separate myocarditis**	Weeks two to three, persistent tachycardia, protein then troponin.
4 **Stop or continue**	Two numbers decide it, then cardiology and imaging.
5 **Rechallenge and augment**	Conditions for rechallenge; ECT before a second antipsychotic.

Five hazards, and the examiner's favourite is the one the blood test misses. Severe neutropenia, a count below 500/mm3, is the historic reason for restricted distribution, and structured monitoring has brought real-world agranulocytosis below 1%.

- **Monitoring interval.** Weekly with differential through months 0 to 6, fortnightly through months 6 to 12, then monthly for the duration of therapy.
- **The regulatory point examiners now probe.** The centralised registry was removed in February 2025, and labelling still mandates that testing.

NEUTROPHIL COUNT	ACTION
At least 1500/mm3, or 1000/mm3 in benign ethnic neutropenia	Continue. After an interruption of 30 days or more, monitor as newly initiated
1000 to 1499/mm3	**Continue, test 3 times weekly until at least 1500/mm3**
500 to 999/mm3	**Interrupt, haematology opinion, daily counts until above 1000/mm3, then 3 times weekly**
Below 500/mm3	**Stop, reverse isolation if febrile, haematology, colony-stimulating factor if infected**

FEATURE	BENIGN TITRATION PHENOMENON	MYOCARDITIS
Frequency and timing	Up to 66% get mild flu-like symptoms, settling over 4 to 6 weeks	**Onset between days 14 and 22, with 83% of cases in that window**
Heart rate	Transient rise of 10 to 20 beats per minute	**Persistent, at or above 120, or a rise above 30**
Protein and troponin	Mild elevation only	**C-reactive protein rises with symptoms, often above 50 mg/L; troponin follows 1 to 5 days later**
Eosinophils	Not the marker	**Peak 7 days after troponin, so useless as an early screen**

Reported incidence spans 0.015% to 8.5%, around 0.7% prospectively and roughly 3% worldwide, with case fatality of 10% to 50%. Rapid titration and concurrent valproate are risks, the latter carrying a 2.5-fold increase.

Surveillance and the cessation threshold. Baseline troponin, C-reactive protein, electrocardiogram and echocardiogram; across days 1 to 28 daily pulse, postural blood pressure, temperature and respiratory rate with direct enquiry; laboratory troponin and protein on days 7, 14, 21, 28, 35 and 42.

- **The two numbers that stop the drug.** Troponin above twice the upper limit, or protein above 100 mg/L, because prolonged exposure after symptom onset carries significantly increased mortality. Subthreshold findings allow cautious continuation with daily markers.
- **Then what.** Urgent echocardiogram, cardiology opinion, consideration of cardiac magnetic resonance imaging, an angiotensin-converting-enzyme inhibitor with a beta blocker, and a loop diuretic for congestion. Function recovers in about 70% of survivors within 5 days.

SHORT CASE, AND THE QUESTION LADDER

A man in his twenties, three weeks into titration on a synthetic ward record, reports fatigue and breathlessness; pulse persistently above 120, low-grade fever, C-reactive protein well above 100 mg/L, troponin three times the upper limit.

1. What is the diagnosis, and what single feature separates it from benign titration fever?
2. Which marker rose first, and which would mislead you if you waited for it?
3. Continue or stop, and on which two numbers does that rest?
4. Which investigations and which two cardiac drugs follow?
5. Is rechallenge permissible, and on what conditions?
6. Why does the haematological protocol give no protection here?

Bowel, seizures and saliva. M1 and M3 antagonism suppresses peristalsis, and impaction, ileus, ischaemia and perforation carry high mortality with a twofold increased ileus risk. Prescribe an osmotic laxative with a stimulant early, and avoid bulk-forming agents, because non-absorbable mass in an aperistaltic colon precipitates obstruction.

- **Seizures are dose and concentration dependent.** Above 300 to 450 mg/day and trough levels above 350 mcg/L, with a crude trial incidence of 3.5%, that is 61 of 1743 patients.
- **Seizure management.** Withhold for 24 hours, draw a 12-hour trough, restart at approximately 50% of the previous dose and titrate slowly; if seizures recur add valproate and avoid carbamazepine, which induces CYP enzymes and adds marrow suppression.
- **Sialorrhoea, paradoxical in an anticholinergic drug.** It reflects alpha-2 antagonism with impaired swallowing: use sublingual atropine 1% drops, 1 to 3 at bedtime titrated to 3 drops three times daily, or intraoral ipratropium 0.06% spray on the same schedule, then clonidine or amisulpride, then glycopyrrolate.
- **Excluded option.** Systemic tertiary anticholinergics, because of fatal ileus risk.

Rechallenge after severe neutropenia. It is cautioned against in the United Kingdom and permitted in revised United States labelling on a documented prescriber and haematologist judgement, treating the patient as newly initiated.

- **Rechallenge after myocarditis.** Cautioned against, yet case series document 63% to 75% success with one fatality, and it requires complete recovery, normal echocardiography, cardiology involvement, consent, inpatient admission, micro-dose titration and daily markers.
- **Clozapine-resistant illness.** Where 30% to 70% respond incompletely, optimise first (adherence, trough above 350 ng/mL, adequate duration), then ECT as the first-line augmentation, then aripiprazole.

RULE **ECT before a second antipsychotic.** Antipsychotic augmentation as a class does not separate from placebo in meta-analysis; ECT is the sourced first-line option.

HOLD THIS

Weeks two to three, tachycardia, protein first and troponin after: myocarditis, with a normal blood count. Stop at troponin twice the upper limit or protein above 100 mg/L.

WHAT EARNS THE MARKS

- **Myocarditis named with a normal blood count in front of you**: the haematological protocol gives no protection here.
- **The marker sequence in order**: protein with symptoms, troponin 1 to 5 days later, eosinophils too late to screen.
- **A neutrophil band matched to its action**: continue, test three times weekly, interrupt, or stop.
- **An osmotic plus stimulant laxative, and bulk-forming agents refused**: the bowel hazard kills and is the one candidates skip.

SEE ALSO 05 Clozapine: workup and ancestry-adjusted titration 34 ECT: indications, workup and anaesthesia 28 Interactions: CYP450, smoking, grapefruit, binding

7 · Long-acting injectables: test dose, loading, overlap

ANTIPSYCHOTICS · TOPIC 07

He relapses whenever he stops tablets. Which injectable, and how do you start it safely?

LONG CASE MAINTENANCE PLAN | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1 Why an injectable	Non-adherence becomes a documented event.
2 Pick from what exists here	Name the preparations actually supplied in India.
3 Test dose or oral cover	Oil-based depots need a test dose; atypicals need tolerability.
4 Load and overlap	Paliperidone deltoid loading, and who needs overlap.
5 The one syndrome	Post-injection delirium belongs to olanzapine pamoate.

The advantage no oral drug has is that non-adherence becomes visible. A missed injection is a documented event, separating pharmacological non-response from covert non-adherence.

- **Added advantages.** Loss of first-pass metabolism, a predictable dose to concentration relationship, lower peak-to-trough fluctuation, regular clinical contact and protection against oral overdose.
- **The numbers to quote.** Continuous depot prophylaxis gave 92% two-year survival without hospitalisation against 54% with targeted intermittent treatment, and relapse on oral maintenance was 30% against 65% on placebo. First and second-generation injectables show similar overall efficacy.

PREPARATION	START	MAINTENANCE AND INTERVAL	STEADY STATE
Haloperidol decanoate	50 mg	50 mg to 300 mg every 4 weeks	**10 to 12 weeks**
Fluphenazine decanoate	12.5 mg	12.5 mg to 50 mg every 2 to 3 weeks	Not carried by the sources
Flupenthixol decanoate	20 mg	50 mg every 4 weeks up to 400 mg weekly	**6 to 12 weeks**
Zuclopenthixol decanoate	100 mg	200 mg every 3 weeks up to 600 mg weekly	**10 to 12 weeks**
Risperidone microspheres	25 mg	25 mg to 50 mg every 2 weeks	Not carried by the sources

PREPARATION	START	MAINTENANCE AND INTERVAL	STEADY STATE
Paliperidone palmitate, monthly	**234 mg then 156 mg into the deltoid a week apart**	39 mg to 234 mg monthly, standard 117 mg	**Rapid, from the loading**
Olanzapine pamoate	150 mg to 300 mg	Every 2 weeks, or 300 mg to 405 mg monthly	Rapid
Aripiprazole monohydrate	400 mg	300 mg to 400 mg monthly	**12 weeks**

Dose increases inside the accumulation phase are illogical: at least 2 further doses at the scheduled interval come first. Available in India are haloperidol, fluphenazine and flupenthixol decanoate, risperidone microspheres and monthly paliperidone palmitate; olanzapine pamoate is largely unavailable because of the observation requirement, and aripiprazole lauroxil is not available.

Test dose, and why there are two reasons. A test dose is mandatory before maintenance dosing of any oil-based first-generation depot: susceptibility to acute extrapyramidal reaction, and hypersensitivity to the base oil. Observe 4 to 10 days.

- **Sourced test doses.** Flupentixol 20 mg, haloperidol 25 mg, zuclopenthixol 100 mg, fluphenazine 12.5 mg.
- **Second-generation injectables.** No oil test dose, but oral tolerability is established first, for example oral risperidone or paliperidone 3 mg daily for at least 2 days.
- **Oral overlap.** At least 3 weeks for risperidone microspheres, 14 days for aripiprazole monohydrate, 21 days for aripiprazole lauroxil, a cross-taper over weeks to months for first-generation depots, and none for paliperidone palmitate or olanzapine pamoate.

Paliperidone: read the units before the dose. Doses are stated as palmitate salt (39, 78, 117, 156, 234 mg) or as milligram equivalents of paliperidone (25, 50, 75, 100, 150 mg eq), the conversion factor being 1.56.

- **Loading.** 234 mg into the deltoid, then 156 mg into the deltoid a week later, within 2 days either side, with monthly maintenance beginning 4 weeks after the second dose, standard 117 mg, into deltoid or gluteal muscle.
- **Why the deltoid.** It gives higher peak concentration and faster absorption, which is why loading goes there.
- **Conversion from oral risperidone.** 1 mg/day to 39 mg monthly, 2 mg to 78 mg, 3 mg to 117 mg, 4 mg to 156 mg, 6 mg to 234 mg.

TRAP Giving paliperidone loading into the gluteal muscle, or adding an unnecessary oral overlap to paliperidone while omitting the mandatory three weeks for risperidone microspheres. The two errors are mirror images and cost the same mark.

Post-injection delirium and sedation syndrome belongs to olanzapine pamoate alone: the poorly soluble pamoate salt entering a vascular channel dissolves rapidly and spikes.

- **Frequency and picture.** Approximately 0.07% of injections, within minutes to 1 hour, mimicking oral overdose with sedation, confusion, agitation, ataxia and dysarthria.
- **The protocol.** Gluteal injection only, observation by a healthcare professional in a registered facility for at least 3 hours, and confirmation that the patient is alert, oriented and accompanied before discharge.
- **After stopping any depot.** Active drug persists 3 to 6 months, so withdrawal reactions are rare, relapse is delayed, and adverse effects can persist for months.

HOLD THIS

Test dose for oil, oral cover for microspheres, deltoid for paliperidone loading, three hours for olanzapine pamoate. Four rules, four drugs, and the examiner asks which belongs to which.

WHAT EARNS THE MARKS

- **A preparation that is actually available here**: naming an unavailable depot in an Indian viva reads as textbook recall.
- **Test dose offered for the oil, not for the atypical**: it shows the reason is the vehicle, not the molecule.
- **Deltoid loading for paliperidone, with no oral overlap**: the mirror-image error is the classic loss of a mark.
- **Three hours of observation for olanzapine pamoate**: the syndrome is rare, and the protocol is what is being examined.

SEE ALSO 02 Potency, equivalents and the percentage method 26 Kinetic basics the examiner opens with 33 Essential psychotropics in India

Mood stabilisers

8 · Lithium: kinetics, levels and the sampling rule

MOOD STABILISERS · TOPIC 08

When would you draw this patient's lithium level, and what number are you aiming for?

LONG CASE, MONITORING QUESTION | OSCE STATION ON LITHIUM COUNSELLING

THE SHAPE OF THE ANSWER

1 Kinetics in one line	Renal, unbound, unmetabolised, distributed in body water.
2 Indications	Acute mania, maintenance, augmentation, anti-suicidal effect.
3 Timing before number	Twelve-hour trough, plain red-top tube.
4 Targets by group	Acute, maintenance, and the lower elderly band.
5 Workup and review	Renal, thyroid, calcium, electrocardiogram, pregnancy test.

Lithium is the one psychotropic whose kinetics fit in a sentence. No hepatic metabolism, negligible protein binding, entirely renal excretion, distribution approximating total body water. **Every interaction and every route into toxicity follows from that.**

- **Licensed and practised indications.** Acute manic and mixed episodes and maintenance of bipolar I disorder, both from 7 years of age; used across both poles in practice, to prevent recurrence in recurrent unipolar depression, and as an augmenting agent in refractory depression, with an established anti-suicidal effect.
- **Mechanism, with the caveat.** The label states that the mood-stabilising mechanism is unknown: name membrane effects, monoamine transmission and phosphatidylinositol metabolism, add neurogenesis and the glycogen synthase kinase 3 and cyclic AMP response element-binding protein pathways, then add the caveat.

PARAMETER	VALUE
Time to peak	0.25 to 3 hours immediate-release; 2 to 6 hours sustained-release
Protein binding and metabolism	**Negligible binding; no cytochrome or phase II metabolism at all**
Volume of distribution	0.7 to 1 L/kg, approximating total body water
Central equilibration	Slow, up to 24 hours to brain tissue equilibrium
Excretion	**Freely filtered, 80% passively reabsorbed proximally alongside sodium**
Half-life	18 to 36 hours with normal renal clearance

Because lithium shares proximal reabsorption with sodium, any state or drug producing volume depletion or sodium deficit increases reabsorption and precipitates toxicity. That one line answers most interaction questions. Atomic weight 6.94; paediatric clearance 0.59 L/h at 20 kg, 0.79 L/h at 30 kg, 1.17 L/h at 50 kg.

GROUP	START AND INCREMENT	ACUTE TARGET	MAINTENANCE TARGET
Adults and children above 30 kg	300 mg three times daily, increasing by 300 mg every 3 days	**0.8 to 1.2 mEq/L**	**0.8 to 1.0 mEq/L**
Children 20 to 30 kg	300 mg twice daily, increasing by 300 mg weekly	0.8 to 1.2 mEq/L, usual dose 600 to 1500 mg daily	0.8 to 1.0 mEq/L, usual dose 600 to 1200 mg daily
Older adults	A lower initial dose, titrated against weekly levels	**0.4 to 0.8 mmol/L**	**0.4 to 0.8 mmol/L**
Creatinine clearance under 30 mL/min	**Avoid**	Avoid	Avoid

The sampling rule, a whole question on its own. Draw a 12-hour trough, exactly 12 hours after the previous evening dose and immediately before the morning dose; an 8 to 12 hour window is acceptable, and 12 hours is standard because immediate-release and extended-release preparations give identical profiles at that point.

- **Tube and early schedule.** Use a serum separator or plain red-top tube. Steady state is reached within 5 days; take the first level after 3 days and then twice weekly until stable.
- **Long-term review.** Levels once stable and on any dose change or new interacting drug, creatinine and urea regularly, thyroid-stimulating hormone at baseline, at 3 months and then every 6 to 12 months, calcium periodically, and counselling for normal salt intake with 2500 to 3000 mL of fluid daily during stabilisation.
- **Pre-treatment workup.** Renal function with urinalysis, thyroid function, electrolytes, calcium, blood pressure with an electrocardiogram, full blood count, a pregnancy test, and a medication audit.

CLASSIC TRAP An asymptomatic, clinically well patient with a level reported in the toxic range. Ask which tube was used: lithium heparin in a green-top tube falsely elevates the assay, and the clinical picture decides the next step.

HOLD THIS

Twelve hours, red top, 0.8 to 1.2 for mania against 0.8 to 1.0 for maintenance, and 0.4 to 0.8 in the elderly. Say the timing before the number and you sound like a prescriber.

WHAT EARNS THE MARKS

- **Timing stated before the target number**: it is the sampling time that makes the number interpretable.
- **Renal-only handling said early**: it lets you derive the interactions instead of memorising them.
- **Separate acute, maintenance and elderly targets**: one band for all three loses the mark.
- **The tube named**: it is the discriminator in the well patient with an alarming level.

SEE ALSO 09 Lithium: organ toxicity and intoxication 10 Lithium in pregnancy and the anti-suicidal claim 40 Lithium, benzodiazepine and paracetamol overdose

9 · Lithium: organ toxicity and intoxication

MOOD STABILISERS · TOPIC 09

She is on lithium and has a coarse tremor with vomiting. How do you grade this and what do you do?

EMERGENCY VIVA TABLE | SHORT CASE, DRUG TOXICITY

THE SHAPE OF THE ANSWER

1 System by system	Renal, thyroid, neurological, cardiac, skin and gut.
2 Grade the intoxication	Three concentration bands, each with its own picture.
3 Resuscitate and correct	Volume first, and cap sodium correction.
4 Dialysis on severity	Severity decides, then recheck for rebound.
5 Explain the interactions	Sodium depletion raises the level. One mechanism, whole table.

SYSTEM	EFFECTS	THE EXAMINABLE DETAIL
Renal	Nephrogenic diabetes insipidus, renal tubular acidosis, chronic kidney disease	**Chronic tubulointerstitial nephropathy is the histology; nephrotic syndrome remits on cessation, while long-standing diabetes insipidus may be partly irreversible**
Thyroid and parathyroid	Hypothyroidism with goitre, paradoxical hyperthyroidism, hyperparathyroidism with hypercalcaemia	**Levothyroxine can be added without stopping lithium; parathyroid disease may not resolve on discontinuation**
Central nervous system	Fine postural tremor, hyperreflexia, twitching, ataxia, dysarthria, downbeat nystagmus, choreoathetosis, cognitive slowing	**Pseudotumor cerebri with papilloedema can cause visual loss if unrecognised**

SYSTEM	EFFECTS	THE EXAMINABLE DETAIL
Cardiovascular	Sinus node dysfunction with bradycardia and sinoatrial block, T-wave flattening or inversion, QTc prolongation, ventricular tachyarrhythmia	**Lithium can unmask Brugada syndrome**
Skin and gut	Psoriasis, folliculitis, alopecia, xerosis, ulcers; anorexia, nausea, diarrhoea, metallic taste	Weight gain with oedema of wrists and ankles
With antipsychotics	Encephalopathic syndrome: weakness, lethargy, fever, tremulousness, confusion, extrapyramidal signs, leucocytosis	**Shares features with neuroleptic malignant syndrome and can leave permanent cerebellar and cognitive damage**

The intoxication ladder. At 1.5 mEq/L or above: coarse tremor, vomiting, diarrhoea, slurred speech, ataxia, drowsiness, weakness.

- **Moderate.** Above 2.0 to 2.5 mEq/L: clonus, delirium, hyperreflexia, fasciculation, prominent extrapyramidal signs.
- **Severe.** Above 3.0 to 3.5 mEq/L: seizures, stupor, coma, ventricular arrhythmia, collapse, irreversible cerebellar atrophy; above 3.5 mmol/L is considered potentially fatal.
- **A single spike counts.** Even one level above 1.0 mmol/L is linked to an acute decline in filtration over 3 months.
- **Management.** Stop the drug, secure airway and cardiac monitoring, restore volume with isotonic saline, and where hyponatraemia coexists cap correction at 10 to 12 mEq/L in 24 hours or 18 mEq/L in 48 hours against osmotic demyelination.
- **Decontamination and clearance.** Lavage may be considered for early massive ingestion; charcoal does not bind lithium. Haemodialysis is the treatment of choice in severe poisoning, with levels rechecked afterwards for rebound as the ion leaves tissue.

TRAP The concentration at which dialysis becomes mandatory is volunteered confidently and the sources do not carry it: there the indication is framed by severity, not a cutoff. Answer on severity, that is severe poisoning with seizures, coma or renal failure, and **verify before the exam**.

INTERACTING CLASS	DIRECTION AND MECHANISM
Thiazide and loop diuretics	**Level rises: natriuresis drives compensatory proximal reabsorption of sodium with lithium**
Non-steroidal anti-inflammatory drugs	**Level rises: prostaglandin inhibition cuts renal blood flow and filtration**
Angiotensin-converting-enzyme inhibitors and receptor blockers	**Level rises: altered glomerular perfusion**
Metronidazole	Level rises: impaired tubular elimination
Acetazolamide, theophylline, aminophylline, bicarbonate, urea	**Level falls: alkalinisation and osmotic transit increase excretion**
Antipsychotics	Extrapyramidal reactions, neuroleptic malignant syndrome, encephalopathic syndrome
Calcium channel blockers	**Neurotoxicity (ataxia, tremor, tinnitus, vomiting) even at levels within range**
Serotonergic drugs, iodide, neuromuscular blockers	Serotonin syndrome; additive antithyroid action; prolonged paralysis

NEAR NEIGHBOURS

A fine, high-frequency postural tremor is expected on therapy; an irregular coarse tremor with ataxia, dysarthria or twitching is early intoxication. In nephrogenic diabetes insipidus, water deprivation leaves urine persistently dilute and fluid restriction produces dehydration, hypernatraemia and severe toxicity, whereas in psychogenic polydipsia restriction concentrates the urine and normalises osmolality.

HOLD THIS

Anything that depletes sodium raises lithium. The tubule cannot tell sodium from lithium, and that is the whole interaction table.

WHAT EARNS THE MARKS

- **A concentration band attached to each clinical picture**: grading is what the question is really testing.
- **Isotonic saline before any thought of dialysis**: volume repletion is the step available in any setting.
- **The sodium correction cap**: 10 to 12 mEq/L in 24 hours protects against osmotic demyelination.
- **Charcoal refused, and rebound anticipated**: two details that separate a read answer from a practised one.

SEE ALSO 08 Lithium: kinetics, levels and the sampling rule 31 Renal, hepatic, cardiac, epilepsy, HIV 40 Lithium, benzodiazepine and paracetamol overdose

10 · Lithium in pregnancy and the anti-suicidal claim

MOOD STABILISERS · TOPIC 10

She is on lithium and eight weeks pregnant. What do you tell her, and what do you change?

LONG CASE, PERINATAL PSYCHIATRY | OSCE COUNSELLING STATION

THE SHAPE OF THE ANSWER

1 State the risk honestly	Ebstein anomaly, and the small absolute risk.
2 Fetal surveillance	Echocardiography and anomaly scan at 16 to 20 weeks.
3 Follow the clearance	Requirement rises, then reverses at delivery.
4 Delivery and neonate	Fluid balance, the floppy baby picture, feeding advice.
5 The anti-suicidal claim	Registry evidence, the null trial, then discontinuation risk.

The pregnancy answer is about clearance and timing, not only Ebstein anomaly. Early registries suggested a high risk of Ebstein anomaly, that is apical displacement of the tricuspid valve with right ventricular hypoplasia, after first-trimester exposure; later prospective work indicates that cardiac malformations occur but the absolute risk is small.

- **Fetal surveillance.** Refer for specialised fetal echocardiography and detailed anomaly scanning between 16 and 20 weeks.
- **Around delivery.** The label position is that the drug is stopped or reduced 2 to 3 days before the expected delivery date, because at delivery vascular volume contracts and clearance falls back to pre-pregnancy rates.
- **Neonatal toxicity.** The floppy baby picture gives hypotonia, cyanosis, hypothermia, lethargy, bradycardia and respiratory distress, and neonatal elimination may take up to 14 days.

PHASE	WHAT IS DONE
First and second trimesters	Trough drawn 12 hours post-dose, with filtration rate, every 4 weeks
From 36 weeks to labour	Trough and filtration rate weekly
Intrapartum	Deliver in a consultant-led unit; stopping at the onset of labour is not advised, because abrupt cessation raises postpartum relapse risk; strict fluid balance, a level 12 hours after the preceding dose, hydration if indicated, and no anti-inflammatory drugs
Neonate and postpartum	Serum lithium shortly after birth; restart maintenance promptly, with a level at 5 to 7 days

Requirements rise across pregnancy as filtration rises, then reverse at delivery. Relative infant dose in milk has been reported up to 42%, and newborn filtration is only 30% to 40% of adult capacity, so the ion accumulates; breastfeeding is advised against, and formula feeding is the standard recommendation.

EVIDENCE	WHAT IT SHOWS
Systematic review	Across 28 clinical studies (18 prospective, 10 retrospective) and 16 ecological studies, long-term lithium substantially reduces suicide and attempts; annual incidence of completed suicide or attempt was 0.435% on lithium against 2.63% without
Mechanism and dose-response	**Regarded as intrinsic and independent of mood stabilisation, probably through reduced aggression and impulsivity, with long-term maintenance better than short-term use**
The 2021 veterans trial	Randomised placebo-controlled across 29 centres, 519 veterans, 52 weeks, stopped for futility: hazard ratio 1.10 (95% confidence interval 0.77 to 1.55; P = 0.61), with 127 events (24.5%), 65 on lithium and 62 on placebo
Why it is not the last word	**Mean levels at 3 months were only 0.54 mEq/L in bipolar disorder and 0.46 mEq/L in depression, only 48.1% ever reached target, adherence was documented in 17%, and the sample was predominantly unipolar with heavy comorbidity**

RULE **Taper lithium; do not stop it.** Abrupt cessation produces a rebound spike in attempts and completed suicide that can exceed the pre-treatment rate within weeks to months, which is the clinical corollary of the anti-suicidal claim.

HOLD THIS

A null trial with a mean level of 0.54 mEq/L and 17% adherence tests delivery, not the drug. Quote the trial, then its levels, then the discontinuation surge.

WHAT EARNS THE MARKS

- **Relative risk separated from absolute risk**: the candidate who quotes only the early registries frightens the patient needlessly.
- **A monitoring interval for each phase**: every 4 weeks, weekly from 36 weeks, then at birth.
- **Abrupt cessation refused**: postpartum relapse risk is the reason, and it is the safer answer.
- **The null trial critiqued on levels and adherence**: 0.54 mEq/L and 17% adherence test delivery, not the molecule.

SEE ALSO 08 Lithium: kinetics, levels and the sampling rule 29 Pregnancy: risk drug by drug 30 Lactation: what crosses, what is advised against

11 · Valproate: toxicity and the childbearing rules

MOOD STABILISERS · TOPIC 11

A woman of 24 with mania. Would you give valproate, and what has to be in place if you do?

LONG CASE, PRESCRIBING DECISION | OSCE CONSENT STATION

THE SHAPE OF THE ANSWER

1 **Indication and dose**	Mania, and the band most patients sit in.
2 **Levels have a small role**	Titrate against symptoms, not concentration.
3 **Organ toxicity**	Liver, marrow, ovary, metabolism and bone.
4 **Quote the teratogenic risk**	One in ten structural, three to four in ten developmental.
5 **The childbearing rules**	Test, contraception, counselling, annual form.

Approved for acute mania as monotherapy or adjunct, and as a maintenance alternative when lithium cannot be used. Start 700 mg to 750 mg/day divided and work to 800 mg to 1500 mg/day.

- **Where real prescribing sits.** Audit data show 70.3% of patients on 700 mg to 1500 mg daily, 20.9% on 1500 mg to 2000 mg, 7.6% on 700 mg or less and 0.6% above 2000 mg.
- **Levels have a limited role.** Concentration correlates poorly with mood-stabilising efficacy and psychiatric cut-offs remain unproven, so titrate against symptoms and tolerability. Real-world practice does not even measure it, with levels recorded in only 22.8% of files.

SYSTEM	PATTERN AND INCIDENCE	MONITORING
Hepatic	**Idiosyncratic fatal necrosis, 1 in 20,000, with 95% of cases in the first 6 months; asymptomatic transaminase rise in about 10%**	Baseline liver function, repeated on clinical signs, with a recheck at 1 month if enzymes rise
Haematological	Thrombocytopenia 12% to 18%; leucopenia 0.4% to 5%; rarely pancytopenia	Baseline full blood count with differential and platelets
Endocrine	**Polycystic ovary syndrome, hyperandrogenism and menstrual abnormality, at almost twofold the risk of other antiepileptics**	Screen for menstrual irregularity, hirsutism, acne and weight change
Metabolic and bone	Weight gain, hyperphagia, insulin resistance; reduced bone density beyond 10 years of treatment	Fasting glucose, lipids, weight; vitamin D and bone density in long-term users
Neurological and skin	Fine postural tremor, drowsiness, ataxia; alopecia, usually reversible	Clinical examination

Periodic schedule, distinct from the symptom-triggered recheck: liver function and full blood count with platelets at baseline, then level, count, liver function, cholesterol and glucose at 6 months and annually. Fatal hepatotoxicity risk factors are age 3 years and below, congenital metabolic disorders, severe organic brain pathology and polytherapy. Unlike carbamazepine, valproate does not induce gamma-glutamyl transferase.

RISK	WITH EXPOSURE	COMPARATOR
Major congenital malformations	**10.93% (95% confidence interval 8.91% to 13.13%)**	2% to 3% unexposed
Dose dependence	**3.3% below 1100 mg daily against 30.2% at 1100 mg or above**	Risk climbs steeply above 1000 mg/day

RISK	WITH EXPOSURE	COMPARATOR
Neural tube, cardiac, craniofacial	12.7-fold neural tube defects; 2.5 to 5.7-fold cardiac; 5.2-fold cleft palate	Spina bifida, septal defects, fetal valproate syndrome
Urogenital and skeletal	4.8-fold hypospadias; 6.8-fold craniosynostosis; 2.2-fold polydactyly	Elevated in male fetuses
Neurodevelopment	**30% to 40% probability of a neurodevelopmental disorder; autism odds ratio 1.7 to 4.4; intellectual disability 2.4 to 4.5; attention deficit 1.4 to 1.8**	Intelligence quotient reduction linked to doses above 800 mg/day, worse at 1500 mg/day

The childbearing rules, examined as rules. Valproate is not prescribed to a woman of childbearing potential unless lithium or lamotrigine are ineffective or not tolerated.

- **The prevention framework.** Where unavoidable it runs under a pregnancy prevention framework: documented negative pregnancy test, highly effective contraception, counselling on teratogenic, dysmorphic and cognitive risk, and an annual risk acknowledgement form.
- **Folic acid, stated precisely.** It does not mitigate the major anatomical malformations, although it may modestly reduce neurodevelopmental risk, and the dose is 4 mg to 5 mg daily against 400 mcg for the general population.
- **If pregnancy occurs.** The drug is not stopped abruptly where that risks severe relapse or self-harm: hold the lowest effective dose, below 1000 mg/day as monotherapy, and add alpha-fetoprotein screening with high-resolution ultrasonography at weeks 16 to 18.
- **For men.** A cohort reported increased neurodevelopmental disorder in children of fathers taking valproate in the 3 months before conception; regulators judged it unsubstantiated, and precautionary advice is contraception for patient and partner during treatment and for 3 months after, with no sperm donation over the same period.

TRAP Stopping valproate for an isolated asymptomatic twofold transaminase rise, which occurs in about 10% of patients. Reduce the dose and repeat liver function after 1 month, reserving cessation for symptomatic cases or progressive decompensation.

HOLD THIS

Roughly one in ten exposed pregnancies carries a major malformation, three to four in ten a neurodevelopmental disorder. Folic acid does not fix it, and 1100 mg/day multiplies the structural risk nearly tenfold.

WHAT EARNS THE MARKS

- **The alternatives named before valproate**: lithium or lamotrigine first is the decision the examiner is listening for.
- **Two teratogenic figures, structural and developmental**: quoting only malformation rates understates the harm.
- **Folic acid correctly limited**: it does not cover the anatomical risk, and saying so shows you read the evidence.
- **The dose threshold**: risk climbs steeply above 1000 mg/day, so the dose is part of the consent conversation.

SEE ALSO 12 Carbamazepine, oxcarbazepine, lamotrigine 29 Pregnancy: risk drug by drug

31 Renal, hepatic, cardiac, epilepsy, HIV

12 · Carbamazepine, oxcarbazepine, lamotrigine

MOOD STABILISERS · TOPIC 12

How would you start lamotrigine in a patient already on valproate, and what would you warn her about?

PSYCHOPHARMACOLOGY VIVA TABLE | OSCE STATION ON TITRATION

THE SHAPE OF THE ANSWER

1 What lamotrigine does	Prevents depressive relapse; it is not an antimanic.
2 Halve for valproate	Glucuronidation inhibited, half-life doubled, dose halved.
3 Rash rules	Weeks two to eight, mucosa and fever, stop and no rechallenge.
4 Interaction directions	Inducer doubles, the pill halves, stopping the pill doubles.
5 Carbamazepine screening	HLA-B*1502 before starting in Asian lineage.

Lamotrigine stabilises from below, preventing depressive relapse rather than treating mania, and its whole prescribing story is the titration. Target 200 mg daily in bipolar disorder, with some patients needing 400 mg; bioavailability 98%, peak at 1 to 3 hours, half-life 24 to 30 hours in monotherapy, and levels are not routinely checked in psychiatry.

WEEK	MONOTHERAPY	WITH VALPROATE	WITH AN INDUCER
Weeks 1 and 2	25 mg once daily	25 mg every other day	50 mg once daily
Weeks 3 and 4	50 mg once daily	25 mg once daily	50 mg twice daily
Week 5 onward	100 mg daily, then 50 mg steps every 1 to 2 weeks	Increase by 25 mg to 50 mg every 1 to 2 weeks	Increase by 50 mg to 100 mg every 1 to 2 weeks
Target	200 mg daily	100 mg to 200 mg daily	Up to 400 mg daily or higher

Outpatient escalation is 25 mg every 2 weeks; inpatient escalation of up to 25 mg per week has been used where daily skin inspection is feasible. Valproate inhibits glucuronidation and doubles the half-life to roughly 56 hours, which is why both the starting dose and the rate are halved.

DRUG	ENZYME EFFECT	CONSEQUENCE
Valproate	**Inhibits glucuronidation**	Doubles lamotrigine half-life; raises clozapine levels
Carbamazepine	**Potent inducer of CYP3A4, CYP1A2 and glucuronosyltransferase**	Halves lamotrigine; lowers antipsychotic and contraceptive levels; induces gamma-glutamyl transferase, so contraceptive failure needs a barrier method or an intrauterine device
Oxcarbazepine	**Moderate to weak CYP3A4 inducer; a prodrug reduced to the 10-monohydroxy metabolite**	Modestly reduces CYP3A4 substrate levels, with far less broad-spectrum interaction
Oestrogen-containing contraceptive	Induces lamotrigine glucuronidation	**Levels fall about 50% within 1 week of starting; stopping the pill can double them; during the inactive 7-day interval levels rise transiently without a routine change**

Severe cutaneous reactions. Stevens-Johnson syndrome and toxic epidermal necrolysis occur most often within the first 8 weeks, specifically between 2 and 8 weeks.

- **Prodrome and rash.** Constitutional first, with fever, sore throat, malaise and headache, followed by a macular or morbilliform rash coalescing across face and trunk; mucosal involvement with epidermal detachment separates it from a benign exanthem.
- **Risks.** Exceeding the starting dose, rapid titration, adding lamotrigine to valproate without halving it, and recent viral illness or vaccination.
- **Action.** Stop the drug at the first unexplained rash, mucosal ulceration or fever, transfer for barrier nursing and fluid management, and do not rechallenge.
- **Prevention advice.** Avoid starting within 2 weeks of a viral illness or vaccination, and advise against new foods, cosmetics or soaps during the first 3 months.

The immunogenetics, for the distinction candidate. Endogenous peptide-loaded HLA-B*1502 presents unchanged carbamazepine directly to cytotoxic T lymphocytes, without intracellular processing or metabolism.

- **Binding detail.** Binding favours the 5-carboxamide group on a tricyclic ring, in a preferred conformation with the side chain of Arg62 in the B pocket, and three groove residues are critical: Asn63, Ile95 and Leu156.
- **Why the allele family matters.** Asn63 is shared across the HLA-B75 family, whose other members can also present the drug, while HLA-B62 and HLA-B72 members cannot.
- **The practical consequence.** Screening before starting carbamazepine is recommended in people of Asian lineage, including Indian populations.

COMMON TRAP

Starting lamotrigine monotherapy as emergency treatment for severe, acutely suicidal bipolar depression. The mandatory slow titration takes roughly 6 weeks to reach target, and the patient is unprotected throughout, so bridging with another agent is needed.

TRAP Psychiatric dose ranges for carbamazepine and oxcarbazepine, and the carbamazepine therapeutic serum range, are not carried by the sourced material. Answer mechanism, interaction and monitoring, and **verify before the exam** against a formulary.

HOLD THIS

Valproate halves the lamotrigine dose, carbamazepine doubles it, the pill halves the level and stopping the pill doubles it. Four directions, one enzyme system, and the rash is the cost of getting any of them wrong.

WHAT EARNS THE MARKS

- **The valproate column quoted, not the monotherapy one**: 25 mg on alternate days is the answer to the question asked.
- **The rash window given as two to eight weeks**: it tells the patient when to watch and when to relax.
- **Mucosal involvement named as the discriminator**: it separates a serious reaction from a benign exanthem.
- **Bridging cover offered for acute suicidality**: six weeks to target means the patient is unprotected meanwhile.

SEE ALSO 11 Valproate: toxicity and the childbearing rules 28 Interactions: CYP450, smoking, grapefruit, binding 18 Resistant depression: staging, STAR*D, augmentation

Antidepressants and treatment resistance

13 · Classes, mechanisms and the latency question

ANTIDEPRESSANTS · TOPIC 13

Classify the antidepressants, and tell me why they take two to four weeks to work.

PSYCHOPHARMACOLOGY VIVA TABLE | LONG CASE, CHOICE OF AGENT

THE SHAPE OF THE ANSWER

1 Class by mechanism	Ten classes, each with its defining receptor action.
2 Answer the latency	Transporter in hours, autoreceptor desensitisation in weeks.
3 Class adverse effects	Gut, sleep, sexual, bleeding, early activation.
4 Tricyclics receptor by receptor	M1, H1, alpha-1 and the sodium channel.
5 Choose for this setting	Cost and availability in Indian outpatient practice.

CLASS	AGENTS	MECHANISM
Serotonin reuptake inhibitors	Fluoxetine, sertraline, paroxetine, fluvoxamine, citalopram, escitalopram	Selective transporter inhibition
Dual reuptake inhibitors	Venlafaxine, desvenlafaxine, duloxetine, levomilnacipran, milnacipran	Serotonin and noradrenaline transporter inhibition

CLASS	AGENTS	MECHANISM
Tricyclics, tertiary amines	Amitriptyline, imipramine, clomipramine, doxepin, trimipramine	**Mixed transporter inhibition with potent M1, H1 and alpha-1 antagonism**
Tricyclics, secondary amines	Nortriptyline, desipramine, protriptyline	**Predominantly noradrenergic, with lower anticholinergic affinity**
Irreversible monoamine oxidase inhibitors	Phenelzine, tranylcypromine, isocarboxazid	Irreversible inhibition of both isoforms
Reversible inhibitor of monoamine oxidase A	Moclobemide	**Reversible competitive inhibition, so substrate displaces the drug**
Noradrenaline-dopamine reuptake inhibitor	Bupropion	Dopamine and noradrenaline transporter inhibition, no serotonergic action
Noradrenergic and specific serotonergic agent	Mirtazapine, mianserin	Alpha-2 auto and heteroreceptor antagonism with 5-HT2, 5-HT3 and H1 blockade
Serotonin antagonist and reuptake inhibitor	Trazodone, nefazodone	Potent 5-HT2A antagonism with weak transporter inhibition
Multimodal and melatonergic	Vortioxetine; vilazodone; agomelatine	Multimodal serotonergic action; transporter with 5-HT1A partial agonism; MT1 and MT2 agonism with 5-HT2C antagonism

The latency question, answered mechanistically. The transporter is blocked within hours, yet the therapeutic delay runs 2 to 4 weeks.

- **The autoreceptor step.** Acute somatodendritic serotonin first stimulates presynaptic 5-HT1A autoreceptors, slowing raphe firing; over 2 to 4 weeks those autoreceptors desensitise and downregulate, disinhibiting the neuron and producing sustained terminal release.
- **Class adverse effects.** Nausea, loose stools, insomnia, headache, dizziness, dry mouth, weight change, transient anxiety and sexual dysfunction.
- **Two safety riders.** Platelet serotonin depletion raises bleeding risk, particularly with anti-inflammatory drugs, aspirin or anticoagulants; and under 25 years of age initiation or escalation needs close observation for emergent suicidality.

Dual reuptake inhibitors are preferred with psychomotor slowing, low energy, poor concentration or comorbid neuropathic pain, adding sweating, sustained blood pressure elevation, tachycardia, constipation, anorexia and insomnia, with a hepatotoxicity precaution for duloxetine.

- **Tricyclics, receptor by receptor.** M1 blockade gives dry mouth, blurred vision, constipation, retention, tachycardia, memory impairment and angle-closure glaucoma; H1 blockade gives sedation and weight gain; alpha-1 blockade gives orthostatic hypotension.
- **The cardiac channel.** Cardiac sodium channel blockade, the quinidine-like effect, widens the QRS, prolongs the QTc and can cause lethal arrhythmia. The therapeutic index is narrow with high mortality in overdose.

- **Contraindications.** After myocardial infarction, in conduction abnormality, severe coronary disease, untreated narrow-angle glaucoma and severe retention.

INDIAN PRACTICE NOTE

Serotonin reuptake inhibitors dominate outpatient care. Amitriptyline and imipramine remain widely used in government and primary care clinics on grounds of cost, and amitriptyline is on the essential list at all three tiers. Irreversible monoamine oxidase inhibitors are rarely prescribed or stocked, which leaves moclobemide as the class representative on viva tables.

TRAP Hyponatraemia is asked at almost every table, and the sourced chapter carries the risk profile without the numbers. Answer that risk is highest in older people and in the first month, with sodium checked in the first few weeks and mirtazapine or agomelatine as lower-risk alternatives; the sodium thresholds and fluid restriction figures are absent, so **verify before the exam**.

HOLD THIS

The transporter is blocked in hours and the autoreceptor desensitises in weeks. One sentence answers the latency question and explains the early activation window.

WHAT EARNS THE MARKS

- **Latency explained by the autoreceptor, not by habit**: this is the mechanism question inside the classification question.
- **Each tricyclic effect tied to its receptor**: it turns a side-effect list into pharmacology.
- **The under-25 observation rider**: a safety statement examiners expect unprompted.
- **A choice justified by what the clinic stocks**: cost and availability are legitimate clinical reasoning here.

SEE ALSO 14 MAOIs, tyramine and the hypertensive crisis 17 Discontinuation, serotonin toxicity, class syndromes 39 Tricyclic overdose and sodium bicarbonate

14 · MAOIs, tyramine and the hypertensive crisis

ANTIDEPRESSANTS · TOPIC 14

Your patient on phenelzine has a sudden occipital headache. What has happened and why?

EMERGENCY VIVA TABLE | OSCE DIETARY COUNSELLING STATION

THE SHAPE OF THE ANSWER

1 Enzyme and isoform	Which isoform handles which amine.
2 Mechanism of the crisis	Tyramine survives the gut and displaces stored noradrenaline.
3 Recognise it	Occipital headache, pressure surge, autonomic storm.
4 Act	Stop, parenteral antihypertensive, cardiovascular monitoring.
5 Diet, and its limits	Irreversible agents restrict; moclobemide does not.

Monoamine oxidase deaminates serotonin, noradrenaline and dopamine. The A isoform preferentially handles serotonin and noradrenaline, the B isoform phenylethylamine and benzylamine, and dopamine is handled by both.

- **The retained niche.** Atypical depression with mood reactivity, hyperphagia, hypersomnia, leaden paralysis and severe rejection sensitivity.
- **The cheese reaction.** Gut and hepatic monoamine oxidase A normally oxidises dietary tyramine, restricting its bioavailability. With that enzyme irreversibly inhibited, tyramine enters the circulation intact, is taken into noradrenergic terminals by the transporter, and displaces large vesicular stores into the synapse, producing alpha-1 vasoconstriction with beta-1 cardiac stimulation.

The presentation. Abrupt severe throbbing occipital or temporal headache radiating frontally; palpitations with marked tachycardia, or bradycardia with chest tightness; neck stiffness.

- **The rest of the picture.** Nausea, vomiting and diaphoresis with cold clammy skin or pallor; mydriasis, photophobia and blurring; epistaxis, dyspnoea, confusion, and potentially intracranial haemorrhage or stroke.
- **Management.** Immediate cessation, emergency parenteral antihypertensive therapy and intensive cardiovascular monitoring.

FOOD GROUP	EXCLUDED	PERMITTED
Cheeses	**Aged and artisan: cheddar, Swiss, parmesan, Stilton, gorgonzola, roquefort, feta, camembert, brie, gruyere, edam**	Fresh pasteurised: cottage cheese, ricotta, cream cheese, processed cheese, fresh mozzarella
Meat and fish	Cured, dried, smoked or aged: salami, pepperoni, bologna, bacon, corned beef, smoked fish, pickled herring, caviar	Freshly prepared poultry, fish and meat; leftovers older than 24 hours are avoided
Fermented	Soy sauce, miso, teriyaki, tofu, fermented bean curd, sauerkraut, kimchi, kombucha, kefir	Fresh unfermented soy milk, fresh vegetables
Produce	Broad beans and pods, snow peas, overripe or dried fruit, overripe bananas with peel, overripe avocado	Fresh fruit and vegetables before over-ripening
Yeast and alcohol	Marmite, brewer's yeast, unpasteurised sourdough; tap and home-brewed beer, Chianti, sherry, vermouth	Commercially baked bread; pasteurised bottled beer, commercial spirits, moderate commercial wine with caution

Table: the exclusion list, which continues during treatment and for a few weeks after stopping an irreversible agent.

TWO DIETARY TRAPS

First, claiming a selegiline patch exempts the patient from restriction: only the lowest patch dose spares intestinal monoamine oxidase A through first-pass bypass, and higher doses mandate the full restriction.

Second, prescribing a strict diet on moclobemide: because inhibition is reversible, dietary tyramine displaces the drug and allows its own degradation, so the classical crisis does not occur at standard therapeutic doses.

TRAP The parenteral antihypertensive and its dose, and the patch strengths that mark the selectivity boundary, are not carried by the sourced material. Give the class and the principle, and **verify before the exam**.

HOLD THIS

Irreversible inhibition is what makes the gut dangerous. Reversible inhibition is self-correcting, which is why moclobemide eats cheese and phenelzine cannot.

WHAT EARNS THE MARKS

- **The displacement mechanism said in one sentence**: it explains the diet, the drug interactions and the treatment at once.
- **Occipital headache named as the sentinel symptom**: it is what the patient is taught to report.
- **Reversible inhibition distinguished from irreversible**: it is why moclobemide carries no classical crisis at therapeutic dose.
- **The washout period after stopping**: restriction continues for a few weeks beyond the last dose.

SEE ALSO 13 Classes, mechanisms and the latency question 16 Switching and the washout table 17 Discontinuation, serotonin toxicity, class syndromes

15 · The atypicals, each by what it does not cause

ANTIDEPRESSANTS · TOPIC 15

He has stopped sertraline because of sexual dysfunction and weight gain. What would you offer instead?

LONG CASE, SWITCHING DECISION | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1 **Match drug to symptom**	Sleep and appetite, drive, insomnia alone, circadian.
2 **Give the mechanism**	One receptor sentence for each agent.
3 **Name the discriminator**	What each one does not cause, and what it does.
4 **State the monitoring**	Liver for agomelatine, seizure threshold for bupropion.

DRUG	MECHANISM	NICHE	DISCRIMINATOR
Mirtazapine	Alpha-2 auto and heteroreceptor antagonism with 5-HT2A, 5-HT2C, 5-HT3 and H1 blockade	Depression with severe insomnia, anorexia and weight loss	**Sedation is greater at 7.5 to 15 mg than at 30 to 45 mg, because noradrenergic transmission at higher doses counterbalances H1 blockade**
Bupropion	Dopamine and noradrenaline transporter inhibition with active metabolites	Psychomotor retardation, lethargy, attention deficit symptoms, seasonal pattern, tobacco cessation	**No sexual dysfunction and no weight gain, but a dose-dependent fall in seizure threshold; 150 to 300 mg with an extended-release ceiling of 450 mg/day**

DRUG	MECHANISM	NICHE	DISCRIMINATOR
Trazodone	Potent 5-HT2A antagonism with weak transporter inhibition and alpha-1 and H1 blockade	Antidepressant-induced or depression-associated insomnia at sub-antidepressant doses	**Priapism is recognised; full antidepressant dosing is limited by orthostatic hypotension and daytime somnolence**
Vortioxetine	Transporter inhibition with 5-HT1A agonism, 5-HT1B partial agonism, 5-HT3, 5-HT1D and 5-HT7 antagonism	Depression where processing speed and executive function are prominent	**Nausea is the commonest dose-dependent effect, and a 3-week washout precedes a monoamine oxidase inhibitor**
Agomelatine	MT1 and MT2 agonism with 5-HT2C antagonism	Depression with disrupted sleep architecture and circadian misalignment	**No sexual dysfunction and no weight gain, but baseline and frequent liver function testing for transaminase elevation**

Two further points. The combination of a dual reuptake inhibitor with mirtazapine, known as California rocket fuel, pairs reuptake inhibition with presynaptic disinhibition for synergistic transmission in refractory depression.

- **Bupropion is contraindicated.** Where there is an active or past seizure disorder, a structural brain lesion, recent head trauma or an active eating disorder, because purging and electrolyte shift multiply the seizure risk at any dose.

TRAP The hypnotic and antidepressant dose ranges for trazodone, and the agomelatine liver monitoring interval in Indian practice, are not carried by the sourced material. Give the mechanism and the monitoring requirement, and **verify before the exam**.

HOLD THIS

Mirtazapine for the patient who cannot sleep or eat, bupropion for the one who cannot get going, trazodone for sleep alone, agomelatine for the rhythm. Then name the cost: weight, seizures, priapism, transaminases.

WHAT EARNS THE MARKS

- **The drug chosen for this patient's symptom**: the table rewards a match, not a favourite.
- **The inverse dose effect of mirtazapine**: more sedation at 15 mg than at 45 mg, with the noradrenergic reason.
- **Bupropion's two negatives and one positive**: no sexual dysfunction, no weight gain, a lowered seizure threshold.
- **Agomelatine paired with liver monitoring**: quoting the drug without its testing requirement is an incomplete answer.

SEE ALSO 13 Classes, mechanisms and the latency question 16 Switching and the washout table 17 Discontinuation, serotonin toxicity, class syndromes

16 · Switching and the washout table

ANTIDEPRESSANTS · TOPIC 16

Eight weeks on fluoxetine and she is barely better. Switch or augment, and how would you do it?

LONG CASE MANAGEMENT PLAN | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1 **Define the trial**	Threshold plus clock: 20% by 2 to 4 weeks, 8 to 12 weeks adequate.
2 **Switch or augment**	Non-response switches, partial response augments.
3 **Pick a strategy**	Direct, taper, washout, cross-taper.
4 **Read the washout row**	Fluoxetine 5 weeks, irreversible inhibitor 2 weeks.
5 **Name the interaction risk**	CYP2D6 and CYP1A2 inhibition raises tricyclic levels.

Response is judged against a threshold and a clock. Early response in depression is a 20% or greater improvement within 2 to 4 weeks of therapeutic dosing, and its absence strongly predicts failure to remit.

- **Other thresholds.** In generalised anxiety disorder it is 50% or greater within 6 to 8 weeks, and an adequate trial is 8 to 12 weeks at an established dose. Up to 65% fail to remit and approximately 50% fail to achieve adequate response on the first agent.
- **Switch when.** Improvement is under 20% after an adequate trial, adverse effects are dangerous or poorly tolerated, the patient prefers monotherapy, or impairment is mild enough to allow a taper.
- **Augment when.** There is partial response, that is 25% to 49% improvement, the first drug is well tolerated, two or more trials have run, or the patient will not risk losing partial relief.

Four strategies, and the one the situation dictates.

- **Direct switch.** Stop one drug and start the next the following morning, feasible under a week of exposure or within a class.
- **Taper and immediate switch.** It balances withdrawal against untreated time.
- **Taper and switch after washout.** A drug-free interval is added, and it is mandatory in either direction involving an irreversible inhibitor.
- **Cross-taper.** Reduce the first drug over 1 to 2 weeks or longer, by roughly 25% per week where it has run 6 weeks or more, while the second is titrated up; preferred for outpatients at relapse risk, at the cost of an interaction window.

FROM	TO	STRATEGY
A serotonin reuptake inhibitor	Another in class, or a dual reuptake inhibitor	Direct switch at a comparable dose, or taper and stop then start low; cross-taper cautiously for the dual agent

FROM	TO	STRATEGY
A serotonin reuptake inhibitor	A tricyclic	**Cautious cross-taper with a low bedtime dose. Paroxetine and fluoxetine inhibit CYP2D6 and fluvoxamine inhibits CYP1A2, raising tricyclic levels for weeks. Clomipramine is not combined with a reuptake inhibitor**
Fluoxetine	Any non-inhibitor antidepressant	**Stop, then 4 to 7 drug-free days, because norfluoxetine provides its own taper**
A tricyclic	A serotonin reuptake inhibitor	Reduce the tricyclic by up to 50%, start the new drug at a standard dose, then withdraw the remainder over weeks
Moclobemide	Any reuptake inhibitor or bupropion	**Taper and stop, then a 24-hour washout**
Any reuptake inhibitor or mirtazapine	Moclobemide	**Taper and stop, then a washout of 1 week**
An irreversible inhibitor	Any antidepressant	**Stop completely, then a mandatory 2-week drug-free washout. Cross-tapering is contraindicated**

DRUG BEING STOPPED	INTERVAL BEFORE AN IRREVERSIBLE INHIBITOR
Fluoxetine	**5 weeks, because norfluoxetine has a half-life of 4 to 16 days and persists in tissue for up to 5 weeks**
Vortioxetine	3 weeks
Sertraline, paroxetine, citalopram, escitalopram, tricyclics	**2 weeks**
Mirtazapine, bupropion, venlafaxine, duloxetine	5 to 7 days, 3 to 5 days, and 3 days for the dual agents

HOLD THIS

Two weeks off the inhibitor, five weeks off fluoxetine. One interval is set by enzyme resynthesis, the other by a metabolite half-life, and confusing them is the commonest switching error.

WHAT EARNS THE MARKS

- **A threshold quoted with a time**: 20% by 2 to 4 weeks is what turns judgement into a decision.
- **Switch and augment separated by response category**: non-response and partial response take different routes.
- **Fluoxetine handled through its metabolite**: norfluoxetine self-tapers, and that is why the interval is 5 weeks.
- **Cross-tapering refused around an irreversible inhibitor**: a safety rule, not a preference.

SEE ALSO 14 MAOIs, tyramine and the hypertensive crisis 17 Discontinuation, serotonin toxicity, class syndromes 18 Resistant depression: staging, STAR*D, augmentation

17 · Discontinuation, serotonin toxicity, class syndromes

ANTIDEPRESSANTS · TOPIC 17

Three days after stopping paroxetine he has dizziness and electric shock sensations. Is this relapse?

SHORT CASE, ADVERSE EVENT | EMERGENCY VIVA TABLE

THE SHAPE OF THE ANSWER

1 Name it correctly	Discontinuation, physiological, not dependence.
2 Separate from relapse	Latency, character, response to reinstatement.
3 Predict by kinetics	Short half-life and no active metabolite means high risk.
4 Treat and prevent	Reinstate, then taper slowly, or substitute fluoxetine.
5 Contrast the syndromes	Serotonin toxicity, malignant syndrome, malignant catatonia.

Discontinuation is a physiological response, not dependence. It follows abrupt cessation, rapid taper or dose reduction after continuous treatment, stated as four weeks in one source and at least six weeks in others, with craving and drug-seeking absent.

- **Incidence.** In randomised trials 24 to 28 percent, with severe symptoms in 2.8 percent, against 16 to 17 percent on placebo; observational literature spans 27 to 86 percent; and stopping paroxetine caused symptoms in 35 percent against 14 percent on placebo.
- **Course.** Onset within two to four days, occasionally within hours of a missed dose; symptoms are usually mild and settle over one to two weeks with a mean of five days; protracted courses persist at two months in 7 percent, at one year in 6 percent and beyond three years in 2 percent.

FEATURE	DISCONTINUATION	DEPRESSIVE RELAPSE
Latency	Two to four days	At least two to three weeks
Character	Dizziness, electric shock sensations, nausea, flu-like malaise	Gradual worsening of mood, anhedonia, neurovegetative change
On reinstating the drug	Extinguishes within 24 hours	Does not resolve within 24 hours

MNEMONIC

FINISH

Flu-like symptoms, **I**nsomnia with vivid dreams, **N**ausea, **I**mbalance with dizziness and vertigo, **S**ensory disturbance (paraesthesia, burning, brain zaps), **H**yperarousal with anxiety, irritability and aggression. The sensory item separates this from anything else.

DRUG	HALF-LIFE	ACTIVE METABOLITE	RISK
Paroxetine	21 hours	No	**High, reported up to 50 percent or 66 percent**
Venlafaxine	**3 to 13 hours**	Yes	High
Duloxetine, sertraline, citalopram, escitalopram	11 to 16, 26, 35, and 27 to 32 hours	Yes for the first two	Moderate
Fluoxetine	**84 to 144 hours**	Yes, norfluoxetine	**Low, 9 to 14 percent, because the metabolite self-tapers**
Bupropion, mirtazapine	12 to 30 and 20 to 40 hours	Yes, then no	Low to moderate
Phenelzine, amitriptyline	**1.2 hours, and 9 to 25 hours**	Yes	High, with severe rebound on the inhibitor

Liability runs inversely to half-life and to the presence of an active metabolite, and treatment under six to eight weeks rarely produces the syndrome. Class differences follow mechanism: reuptake inhibitors give dizziness and shock sensations from a sudden synaptic deficit; tricyclics give cramping, sweating and parkinsonian tremor from cholinergic rebound; inhibitors give agitation, delirium, hallucination and myoclonus.

Prevention and management. Taper over six to eight weeks, extending to three months or more after long maintenance. For mild symptoms, psychoeducation.

- **For severe symptoms.** Restart at the original dose, which abolishes them within 24 hours, then taper more slowly; for repeated failure on a short half-life drug, substitute fluoxetine, for example at 20 mg per day.
- **Symptomatic options.** Paracetamol or ibuprofen, ondansetron, a short course of hydroxyzine or a benzodiazepine, and an anticholinergic for tricyclic rebound.
- **One caveat.** Generic formulations are permitted a difference of up to 20 percent, so a brand switch on a short half-life drug can itself trigger the syndrome.

SYNDROME	NEUROMUSCULAR SIGNATURE	ONSET AND GUT
Serotonin toxicity	**Hyperreflexia with spontaneous, inducible and ocular clonus, tremor, legs worse**	**Hours after a serotonergic combination; hyperactive bowel sounds with diarrhoea**
Neuroleptic malignant syndrome	**Hyporeflexia with lead-pipe rigidity and raised creatine kinase**	**Insidious over days after a dopamine antagonist; hypoactive bowel sounds**
Malignant catatonia	Waxy flexibility, posturing, mutism, negativism	Often resolves rapidly after intravenous lorazepam

Serotonin toxicity is diagnosed on the Hunter criteria: with a serotonergic agent present, spontaneous clonus, or inducible clonus with agitation or diaphoresis, or ocular clonus with agitation or diaphoresis, or tremor with hyperreflexia, or hypertonia with fever above a threshold.

- **Triggers.** Cross-class combinations, particularly with an inhibitor, linezolid or methylene blue, and with tramadol, pethidine, dextromethorphan, triptans or St John's wort, or an inadequate washout.
- **Management.** Stop every serotonergic drug, support with fluid, cooling and oxygen, control agitation with intravenous benzodiazepines while avoiding physical restraint because isometric contraction worsens hyperthermia and rhabdomyolysis, and give cyproheptadine as the specific antagonist.
- **Two things that do not work.** Antipyretics, because the fever is muscular. Severe hyperthermia needs intubation with non-depolarising blockade.

TRAP The Hunter temperature cut-off, the criterion count, the benzodiazepine and cyproheptadine doses, and the content of the older Sternbach criteria that examiners ask you to contrast, are not carried by the sourced material. Give the decision rule and the sequence, and **verify before the exam**.

Sexual dysfunction, with the baseline first. Between 35 percent and 50 percent of patients with untreated depression already have sexual dysfunction, so enquire before prescribing.

- **Ranking.** Paroxetine carries the highest risk, with venlafaxine and clomipramine also high; lowest are bupropion, mirtazapine, vilazodone and vortioxetine.
- **The ladder.** Watchful waiting, dose reduction, changing the timing, brief drug holidays for intermediate half-life agents, switching to a low-liability agent, augmenting with bupropion, a phosphodiesterase-5 inhibitor or buspirone, and couple or sex therapy.
- **Persistent post-SSRI dysfunction.** Impairment continuing for months or years after cessation, with genital numbness and muted orgasm: pharmacovigilance identified 89 reports of sexual dysfunction, of which 4 documented persistence, in patients aged 18 to 42 years with symptoms lasting 12 months to 3.5 years, and an international series of 300 enduring cases attributed 218 to reuptake inhibitors, with 62 percent linked to five agents.
- **The discriminator.** Depression-related dysfunction is loss of desire without genital anaesthesia.

Three more class effects, each with its own action.

- **Citalopram and escitalopram prolong the QTc.** Dose-dependently, through potassium channel blockade: baseline electrocardiography above 60 years, with cardiac disease, or alongside other prolonging psychotropics; correct potassium and magnesium; switch to sertraline (50 to 200 mg per day) or bupropion (75 to 450 mg per day).
- **Hyponatraemia.** Through inappropriate antidiuretic hormone secretion, with advanced age, female sex, low weight, warm climate and concurrent diuretic or carbamazepine as risks, presenting insidiously with lethargy, unsteadiness, confusion and falls.
- **Bleeding.** Platelets cannot synthesise serotonin and depend on transporter uptake, so blockade depletes dense-granule serotonin and impairs primary haemostasis, giving bruising, epistaxis, menorrhagia and gastrointestinal haemorrhage, amplified by anti-inflammatory drugs and antiplatelets, which are commonly bought over the counter; add a proton pump inhibitor or switch to bupropion or mirtazapine.
- **Activation syndrome.** Early jitteriness, agitation, restlessness and insomnia within days of starting or increasing a drug, separated from akathisia (a motoric urge with foot stamping), from a bipolar switch (elevated mood, grandiosity, reduced sleep need) and from worsening primary anxiety (no acute tie to the dose).
- **Managing activation.** Start low, reduce the dose, or cover the first one to two weeks with propranolol or a short-acting benzodiazepine.

HOLD THIS

Clonus and hyperreflexia within hours is serotonin toxicity; lead-pipe rigidity and hyporeflexia over days is neuroleptic malignant syndrome. Reflexes and the clock separate them before any blood test.

WHAT EARNS THE MARKS

- **Reinstatement used as the diagnostic test**: symptoms gone inside 24 hours settles discontinuation against relapse.
- **Risk derived from half-life and metabolite**: it predicts the syndrome instead of memorising a drug list.
- **Reflexes offered as the syndrome discriminator**: hyperreflexia with clonus against lead-pipe rigidity with hyporeflexia.
- **Restraint refused in serotonin toxicity**: isometric contraction worsens hyperthermia, and knowing that is a distinction answer.

SEE ALSO 03 Extrapyramidal syndromes and tardive dyskinesia 16 Switching and the washout table 39 Tricyclic overdose and sodium bicarbonate

18 · Resistant depression: staging, STAR*D, augmentation

ANTIDEPRESSANTS · TOPIC 18

She has failed two antidepressants. Is this treatment-resistant depression, and what comes next?

LONG CASE, RESISTANT DEPRESSION | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1 Define, then exclude	Two failed regimens, then rule out pseudo-resistance.
2 Stage it twice	Thase and Rush categorically, Maudsley dimensionally.
3 Quote STAR*D	Remission falls level by level; dropout climbs.
4 Augment on evidence	Four agents beat placebo; name the tolerability cost.
5 Offer the interventional route	Glutamatergic agents and ECT, with their settings.

Define it, exclude the pseudo-version, then stage it. Lifetime prevalence of major depressive disorder is approximately 16%, and between 6% and 55% across settings fail to achieve adequate response. Regulators define resistance as failure of 2 or more regimens of adequate dose and duration with documented adherence in the current episode.

- **Pseudo-resistance is excluded first.** Inadequate duration (at least 6 to 12 weeks, with registry cut-offs of 28 days for outpatient prescriptions and 7 days for inpatient administration), subtherapeutic dosing, poor adherence, and misclassification covering unrecognised bipolar depression, substance use, occult medical illness and comorbid personality disorder.
- **Difficult-to-treat depression.** The broader paradigm, framing the illness as chronic and incorporating chronicity, severity, recurrent admission, disability and comorbidity, with symptom optimisation and functional recovery as goals.

Thase and Rush staging is a categorical model of 5 stages graded by the classes sequentially failed. Stage I is failure of 1 adequate trial from a major class; Stage II is failure of a second from a different class.

- **The later stages.** Stages III, IV and V are conventionally taught as further failure of a tricyclic, of a monoamine oxidase inhibitor, and of a full course of bilateral ECT, which is conventional teaching rather than a sourced fact.
- **Volunteer its limitations.** It presumes later drugs are more efficacious, presumes a fixed sequence ending in ECT, accommodates neither the number of agents used within one episode nor combination strategies, and rests on response alone, omitting duration and baseline severity.

MAUDSLEY DIMENSION	SPECIFICATION	POINTS
Episode duration	Acute up to 12 months; subacute 12 to 24 months; chronic over 24 months	1, 2 or 3
Symptom severity	Subsyndromal to severe with psychosis	**1 to 5**
Treatment failures	0 to 2 agents, 3 to 4, 5 to 6, 7 to 10, more than 10	**1 to 5**
Augmentation and ECT failure	Each adds a point	Plus 1 each
Total	**Score 3 to 15: mild 3 to 6, moderate 6 to 10, severe 11 to 15**	Maximum 15

In a cohort of 105,808 patients with hospital-treated depression, 63.4% scored moderate and 5.1% severe, with a mean of 7.4; the method agrees with a binary definition at the extremes, 0.6% of mild cases against 86.7% of severe cases meeting it, while capturing complexity in the middle.

LEVEL	TREATMENT	REMISSION	OBSERVATION
Level 1	Citalopram monotherapy for 12 to 14 weeks, 2,876 patients	**Approximately 33%**	A further 10% to 15% responded without remitting
Level 2	Switch to sertraline, bupropion or venlafaxine, or add bupropion or buspirone	**Switch approximately 25%, add-on approximately 33%**	Switch agents were comparable; bupropion add-on beat buspirone on tolerability
Level 3	Switch to mirtazapine or nortriptyline, or add lithium or triiodothyronine	**Switch 12% to 20%, add-on approximately 20%**	Thyroid hormone caused fewer troublesome events and fewer dropouts than lithium
Level 4	Tranylcypromine against venlafaxine with mirtazapine	**7% to 10%**	The combination reduced symptoms more; the inhibitor was limited by diet and dropouts

The trial enrolled 4,041 outpatients aged 18 to 75 years across 41 clinics, of whom 2,876 entered the first level. Its legacy is measurement-based care.

Response, at least a 50% reduction, took 6 weeks on average, remission nearly 7 weeks and 5 to 6 visits. Cumulative remission reached approximately 50% across 2 levels and nearly 70% across all 4, dropout rose to 21%, 30% and 42% after the first three levels, and patients needing more steps relapsed more often.

AUGMENTATION	EVIDENCE
Quetiapine, aripiprazole, thyroid hormone, lithium	**Superior to placebo for response, odds ratios 1.56 to 1.92, with no active agent beating another**
Thyroid hormone, risperidone, quetiapine, buspirone, aripiprazole, olanzapine	Superior to placebo for remission, odds ratios 1.79 to 2.94
Bupropion, buspirone for response, methylphenidate, lamotrigine, pindolol	**No significant benefit over placebo**
Tolerability	**Discontinuation for adverse events was significantly higher for quetiapine, olanzapine, aripiprazole and lithium, odds ratios 2.30 to 5.64, across a network of 48 trials and 6,654 participants**
Trial windows	Antipsychotics assessed over 6 to 12 weeks; lithium and thyroid hormone over 3 to 4 weeks

Rapid-acting glutamatergic agents. Ketamine and esketamine are non-competitive N-methyl-D-aspartate antagonists acting on cortical GABAergic interneurons.

- **The mechanism, in sequence.** Inhibiting the interneuron disinhibits pyramidal neurons, releasing a glutamate surge that stimulates AMPA receptors and activates brain-derived neurotrophic factor and mammalian target of rapamycin signalling, producing rapid synaptogenesis.
- **Racemic ketamine.** A sub-anaesthetic infusion, with rapid antidepressant and anti-suicidal effect, and adverse effects of dissociation, psychotomimetic phenomena, dizziness, nausea and transient haemodynamic rise.
- **Monitoring.** A pre-treatment electrocardiogram with continuous blood pressure, oximetry and cardiac monitoring, and effects dissipate within days so maintenance is needed.
- **Intranasal esketamine.** Given under supervision in an accredited facility alongside a newly started oral antidepressant, with observation until blood pressure stabilises and dissociation resolves.

TRAP Target doses and serum levels for the augmenting agents, and the ketamine dose in mg/kg, infusion duration, observation period and maintenance interval, are all absent from the sourced material. Give the setting, the window and the monitoring, and **verify before the exam**.

LONG CASE, AND THE QUESTION LADDER

A woman in her forties, synthetic vignette, has had a depressive episode for over a year, has failed two adequate antidepressant trials and one augmentation, and is losing weight.

1. Does she meet the regulatory definition of resistance, and what must you exclude first?
2. Stage her on Thase and Rush and then on the Maudsley method, and say what the second adds.
3. Which dimensions change if the episode had run three years instead?
4. Switch or augment, and on what threshold does that turn?
5. Which augmenting agents have meta-analytic support, and which cost most in discontinuation?
6. What features move you to ECT without further stepped trials, and what must be in place in law first?

HOLD THIS

Half remit in two levels and seven to ten in a hundred remit at the fourth. That is the argument for staging properly and for an interventional option rather than a fifth drug.

WHAT EARNS THE MARKS

- **Pseudo-resistance excluded before the label is applied**: dose, duration, adherence, diagnosis.
- **Both staging methods offered, with what the dimensional one adds**: duration and severity, not just failed drugs.
- **Remission rates quoted level by level**: 33%, then 25%, then 12% to 20%, then 7% to 10%.
- **Augmentation ranked with its discontinuation cost**: efficacy odds ratios beside odds ratios for stopping.

SEE ALSO 16 Switching and the washout table 34 ECT: indications, workup and anaesthesia 37 Neuromodulation: rTMS, iTBS, tDCS, DBS, VNS, MST

Anxiolytics, stimulants and cognition

19 · Benzodiazepines: kinetics and equivalence

ANXIOLYTICS · TOPIC 19

Which benzodiazepine would you choose in a patient with liver disease, and why?

PSYCHOPHARMACOLOGY VIVA TABLE | SHORT CASE, COMORBID LIVER DISEASE

THE SHAPE OF THE ANSWER

1 Mechanism in one line	Allosteric modulation, frequency of channel opening.
2 Three kinetic principles	Lipophilicity, distribution against elimination, metabolism.
3 Name the LOT drugs	Direct glucuronidation, no active metabolite.
4 Use equivalence honestly	An estimate for tapering, not a conversion equation.

Positive allosteric modulation at the GABA A receptor, binding the alpha and gamma subunit interface, facilitating GABA and increasing the frequency of chloride channel opening, so the membrane hyperpolarises. **Three kinetic principles then decide everything clinical.**

- **Lipophilicity.** It sets onset, not time to peak plasma level.
- **Distribution against elimination half-life.** After a single dose of a lipophilic drug, offset comes from rapid distribution into fat, while with repeated dosing tissue stores saturate and both accumulation and offset follow the elimination half-life and any active metabolites.
- **Route of biotransformation.** Phase I oxidation via CYP3A4 and CYP2C19 then glucuronidation, with oxidation of diazepam, chlordiazepoxide and flurazepam yielding long-lived active metabolites, while lorazepam, oxazepam and temazepam go straight to inactive glucuronides.
- **Bioavailability differs too.** Oral diazepam and lorazepam exceed 90%, diazepam ranging 53% to 97%, while oral midazolam averages about 40%, range 35% to 75%.

DRUG	EQUIVALENT ORAL DOSE (MG)	PEAK (HOURS)	HALF-LIFE (HOURS)	CHARACTER
Alprazolam	0.5 to 1	1 to 2	12	Short to intermediate, high potency
Clonazepam	0.25 to 0.5	1 to 4	34	Long acting, high potency
Diazepam	5 to 10	1 to 2	**100**	Long acting with active metabolites
Lorazepam	1 to 2	1 to 4	15	**Intermediate, direct glucuronidation**
Chlordiazepoxide	10 to 25	1 to 4	100	Long acting with active metabolites
Oxazepam and temazepam	15 to 30, and 10 to 20	1 to 4, and 2 to 3	8, and 11	**Direct glucuronidation, no active metabolite**
Triazolam	0.25 to 0.5	1 to 2	2	Ultra-short acting hypnotic
Nitrazepam and flurazepam	2.5 to 10, and 15 to 30	0.5 to 2, and 0.5 to 1	30, and 100	Hypnotics, the second with active metabolites

Equivalence is an estimate, not an equation: published values vary by a factor of 10 or more, most derive from consensus and alcohol withdrawal protocols, and none adjusts for age, organ function or repeated dosing. Parenterally, 2 mg of intravenous midazolam is roughly equipotent to 1 mg of intravenous lorazepam.

TRAP Using an equivalence chart to calculate a starting dose in a benzodiazepine-naive patient. The charts convert an established dose during a taper, and quoting one as an exact biological conversion invites the follow-up you cannot answer.

HOLD THIS

LOT: lorazepam, oxazepam, temazepam. Direct glucuronidation, no active metabolite, safer in liver disease and old age. Everything else oxidises first and accumulates.

WHAT EARNS THE MARKS

- **Onset attributed to lipophilicity**: it explains why intravenous diazepam acts before its level peaks.
- **Single-dose offset separated from steady-state offset**: the distinction candidates most often miss.
- **LOT named with its reason**: glucuronidation is preserved where oxidation is impaired.
- **Equivalence presented as an estimate**: charts vary by a factor of 10 and adjust for nothing.

SEE ALSO 20 Withdrawal, the diazepam taper and flumazenil 31 Renal, hepatic, cardiac, epilepsy, HIV 38 Rapid tranquillisation and acute agitation

20 · Withdrawal, the diazepam taper and flumazenil

ANXIOLYTICS · TOPIC 20

He has taken alprazolam nightly for four years and wants to stop. How would you take him off it?

LONG CASE, DEPENDENCE MANAGEMENT | OSCE STATION ON TAPERING

THE SHAPE OF THE ANSWER

1 State the licence	Two to four weeks, and what happens beyond it.
2 Describe withdrawal	Onset, peak, duration, and the severe end.
3 Convert to diazepam	Long half-life smooths inter-dose spikes.
4 Taper and support	Smaller steps as the dose falls, with therapy alongside.
5 Place flumazenil	Bolus in toxicology, infusion in detoxification.

These drugs are licensed for short-term relief, with treatment recommended not to exceed 2 to 4 weeks. Tolerance to the anxiolytic and hypnotic effect develops within weeks, through decreased allosteric potentiation, receptor uncoupling and altered subunit expression.

- **Dependence.** It develops readily beyond 4 weeks, at which point the drug suppresses withdrawal rather than treating the disorder.
- **Acute withdrawal.** It may not appear until 2 days after cessation, peaks in the first week and commonly persists up to 6 weeks or longer, with rebound anxiety, panic, depersonalisation, perceptual distortion, sweating, insomnia, headache, coarse tremor, palpitations, muscle spasm and sensory hypersensitivity, and at the severe end seizures, delirium and hallucination.
- **Protracted withdrawal.** It affects approximately 10% to 15% of those detoxifying and runs a fluctuating wave-like course over months to years. Severity is rated on the Clinical Institute Withdrawal Assessment Scale for benzodiazepines.

The taper. Assess readiness, with minimal concurrent crises. Convert to an equivalent daily dose of diazepam, whose 100 hour half-life and active metabolites smooth fluctuation, remove inter-dose spikes and allow fine titration.

- **The reduction pattern.** Stabilise, then reduce in small steps every 1 to 2 weeks, making each reduction smaller and the intervals longer as the dose falls.
- **How long it takes.** Typically several months, and up to a year in long-standing or high-dose dependence, where high dose means 30 mg daily diazepam equivalents or more, with baselines reported up to 280 mg and use spanning 1 to 23 years.
- **Alongside the taper.** Concurrent cognitive behavioural therapy significantly improves outcome, an antidepressant is added if depression emerges, and propranolol covers tremor and palpitations.

FLUMAZENIL USE	REGIMEN	CONSEQUENCE
Emergency reversal	200 mcg intravenously over 15 seconds, then 100 mcg over 15 seconds every 60 seconds if needed, to a maximum of 1 mg in 24 hours	**Reverses coma and respiratory depression, but in a tolerant or dependent patient, or a mixed overdose with a pro-convulsant, it precipitates severe withdrawal, panic and status epilepticus**
Low-dose infusion for detoxification	Continuous subcutaneous 4 mg/24 h for approximately 8 days; alternatives 1 mg intravenously over 4 hours twice daily with oxazepam tapering for 8 days, or 1 mg/24 h subcutaneously with clonazepam tapering for 7 days	**Does not raise state anxiety, avoids precipitating panic, and significantly reduces trait anxiety at 1 month**
Outcome	Randomised crossover in 26 participants at baseline equivalents of 5 to 280 mg	Self-reported abstinence 65.4% at 1 month, 50.0% at 2 months, 46.2% at 3 months; with record review 73.1%, 65.4% and 61.5%; historical cohorts 52.9% at 6 months

Flumazenil is a competitive antagonist at the benzodiazepine site with weak partial agonist properties, hypothesised to reset uncoupled receptors. One caveat for the review visit: because diazepam has a 100 hour half-life, a urine screen can stay positive at the 1 month follow-up in a fully abstinent patient.

RULE **Bolus flumazenil is a toxicology decision; infusion flumazenil is a detoxification decision.** Same molecule, opposite risk, decided by the rate of delivery.

HOLD THIS

Convert to diazepam, reduce every one to two weeks, and expect months. The long half-life is the therapeutic tool, and the taper slows as the dose falls.

WHAT EARNS THE MARKS

- **Conversion to diazepam justified by kinetics**: the 100 hour half-life is the therapeutic tool.
- **A realistic timescale offered**: months, up to a year, rather than a fortnight.
- **Psychological treatment prescribed with the taper**: it changes the outcome and is often left out.
- **Flumazenil split by route and rate**: a bolus can precipitate status epilepticus, an infusion does not.

SEE ALSO 19 Benzodiazepines: kinetics and equivalence 21 Z-drugs, buspirone, pregabalin, hydroxyzine, melatonin 40 Lithium, benzodiazepine and paracetamol overdose

21 · Z-drugs, buspirone, pregabalin, hydroxyzine, melatonin

ANXIOLYTICS · TOPIC 21

What would you give for anxiety in a patient with a history of substance dependence?

LONG CASE, COMORBID ADDICTION | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1 **Sort by receptor**	GABA A agents against the non-GABA options.
2 **Name the liability**	Which carry dependence, which do not.
3 **Match to the patient**	Buspirone, hydroxyzine or melatonin by target symptom.
4 **Prescribe within the rules**	Schedule, register, driving advice, review interval.

AGENT	MECHANISM	PLACE	LIABILITY
Zolpidem	Imidazopyridine, selective at the GABA A complex, metabolised mainly by CYP3A4	Short-term insomnia	**Tolerance, dependence, daytime sedation and falls comparable to benzodiazepines beyond 2 to 4 weeks; interacts with chlorpromazine and ritonavir**
Zopiclone	Cyclopyrrolone with a chiral centre and stereoselective kinetics, cleared by demethylation, oxidation and decarboxylation	Short-term insomnia	**Same dependence profile; interacts with erythromycin, trimipramine and carbamazepine**
Buspirone	Azapirone partial agonist at presynaptic and postsynaptic 5-HT1A receptors, no GABA A binding, extensive first-pass hydroxylation and dealkylation	Generalised anxiety over sustained daily dosing	**Negligible abuse liability, and no cross-tolerance, so it cannot prevent or treat benzodiazepine withdrawal**
Pregabalin	Binds the alpha-2-delta subunit of presynaptic calcium channels, reducing excitatory release	Generalised anxiety and neuropathic pain	**Recognised dependence and withdrawal liability, so discontinuation is gradual**
Hydroxyzine	First-generation piperazine H1 antagonist crossing the blood-brain barrier	Rapid sedation and anxiety relief without a controlled prescription	**No GABA A dependence, at the cost of anticholinergic sedation, dry mouth and cognitive slowing**
Prolonged-release melatonin	MT1 and MT2 agonism in the suprachiasmatic nucleus	Primary insomnia, particularly in older adults	**No dependence, tolerance, hangover or rebound insomnia**

Buspirone deserves its own sentence, because of how often it is misused in an answer. Selective anxiolysis with little sedative, hypnotic, anticonvulsant or muscle relaxant effect, minimal psychomotor impairment, minimal kinetic interaction, and a delayed onset requiring sustained daily dosing over several weeks.

TRAP Prescribing buspirone to cover a benzodiazepine taper: it acts at 5-HT1A and not at GABA A, so there is no cross-tolerance and no protection. The daily doses of buspirone, pregabalin, hydroxyzine and melatonin, and the melatonin age cut-off, are not carried by the sourced material, so **verify before the exam**.

INDIAN PRACTICE NOTE

Sedative-hypnotics are restricted to courses of no more than 2 to 4 weeks, with pre-existing substance use assessed, consent documented about falls and road traffic risk, and a definite review interval set.

Benzodiazepines and the Z-drugs sit on the restricted prescription schedule of the Drugs and Cosmetics Rules, needing a dedicated pharmacy register, and several of the same molecules are separately scheduled under the narcotics statute. Advise explicitly about impaired driving and additive depression with alcohol.

HOLD THIS

Two to four weeks is the licence, not a suggestion. Z-drugs behave like benzodiazepines beyond it; buspirone, hydroxyzine and melatonin do not, and that is why they are worth knowing.

WHAT EARNS THE MARKS

- **A non-GABA option chosen for this patient**: the question is about dependence liability, not about potency.
- **Buspirone's delayed onset stated**: it rules the drug out for acute distress and in for sustained anxiety.
- **No cross-tolerance with benzodiazepines**: buspirone cannot cover a taper, and saying so pre-empts the follow-up.
- **The prescription schedule and the register**: regulatory detail is examinable in an Indian viva.

SEE ALSO 20 Withdrawal, the diazepam taper and flumazenil 23 Alcohol: naltrexone, acamprosate, disulfiram 33 Essential psychotropics in India

22 · ADHD and cognition drugs

STIMULANTS AND COGNITION · TOPIC 22

A boy of nine with ADHD. Which drug would you start in an Indian clinic, and what do you check first?

LONG CASE, CHILD PSYCHIATRY | OSCE STATION ON BASELINE WORKUP

THE SHAPE OF THE ANSWER

1 Classify the agents	Stimulant, noradrenergic, alpha-2, wakefulness, cholinergic.
2 Baseline evaluation	Cardiac history, psychiatric history, growth, liver enzymes.
3 Dose by weight	Atomoxetine per kilogram, with the ceiling.
4 Name the warnings	Suicidality, liver injury, growth, priapism, interactions.
5 Say why here	The regulatory answer, not the pharmacological one.

Classify first. Central stimulants (methylphenidate, amphetamines) through transporter inhibition; selective noradrenaline reuptake inhibition (atomoxetine); alpha-2 agonism

(clonidine, guanfacine); wakefulness promotion (modafinil); acetylcholinesterase inhibition (donepezil, rivastigmine, galantamine); and uncompetitive N-methyl-D-aspartate antagonism (memantine).

- **Cardiovascular baseline.** History of exertional chest pain, syncope, palpitations or structural defect with a family history of premature sudden death, plus blood pressure and pulse, and an electrocardiogram where cardiac risk is identified.
- **The rest of the baseline.** Psychiatric history of bipolar disorder, mania, psychosis or suicidal ideation; height and weight on growth charts; and liver enzymes when atomoxetine is started.

POPULATION	START	TARGET	MAXIMUM
Children under 70 kg, from 6 years	0.5 mg/kg once daily, increasing after a minimum of 3 days	Approximately 1.2 mg/kg daily	**1.4 mg/kg daily or 100 mg daily, whichever is less**
Children 70 kg or more, and adults	40 mg once daily, increasing after a minimum of 3 days	80 mg once daily or divided	100 mg daily after a further 2 to 4 weeks
Poor CYP2D6 metabolisers, or on a potent inhibitor	**Escalate only if symptoms have not improved after 4 weeks**	Same targets, reached over extended 4 week intervals	Same ceiling
Hepatic impairment	**Reduce initial and target doses by 50% at Child-Pugh B and by 75% at Child-Pugh C**	Halved or quartered	Halved or quartered

Atomoxetine: bioavailability 63% to 94%, peak at 1 to 2 hours, volume of distribution 0.85 L/kg, protein binding 98%, half-life 5.2 hours, eliminated 80% in urine and 17% in faeces, metabolised by CYP2D6 to an equipotent metabolite, with therapeutic onset over 2 to 4 weeks.

In poor metabolisers the area under the curve is about 10 times higher and peak concentration 5 times higher, and adverse effects shift with it: dry mouth 20% against 35%, decreased appetite 16% against 23%, erectile dysfunction 8% against 21%, hyperhidrosis 4% against 15%. No renal adjustment is needed, and tapering is not mandatory.

Warnings. A boxed warning for suicidal ideation in children, with reported events in children aged 6 to 12 years during the first month.

- **Liver injury.** Idiosyncratic severe injury typically presents within 120 days with jaundice, pruritus, dark urine or right upper quadrant tenderness, and calls for discontinuation without rechallenge.
- **Cardiovascular and growth.** Blood pressure and heart rate rise, and the drug is contraindicated where that would cause deterioration. Growth lags normative curves through the first 9 to 12 months, with weight rebounding and height stabilising at approximately 3 years.
- **Emergency and interactions.** Priapism beyond 4 hours is an emergency. Co-administration with or within 14 days of a monoamine oxidase inhibitor is contraindicated; potent CYP2D6 inhibitors need the extended 4 week interval; thioridazine and dronedarone are contraindicated on QTc grounds; and combining with methylphenidate risks an acute hypertensive episode.

TRAP The starting dose, increment, frequency and maximum of immediate-release and extended-release methylphenidate, in children and adults, is the figure an examiner is most likely to want on this topic, and it is not carried by the sourced material. Answer mechanism, controlled status and monitoring, and **verify before the exam** against the product label rather than improvising a milligram.

FEATURE	CHOLINESTERASE INHIBITORS	MEMANTINE
Mechanism and stage	Reversible acetylcholinesterase inhibition; mild to moderate Alzheimer dementia	Uncompetitive N-methyl-D-aspartate antagonism; moderate to severe dementia
Gut	**Cholinergic hypermotility: nausea, vomiting, diarrhoea, and as serious effects severe abdominal pain and very dark stools from gut bleeding**	**Hypomotility with constipation**
Cardiovascular	**Unsteadiness or fainting raising fall risk; bradycardia and heart block are the classic concern**	**Raised blood pressure**
Starting dose carried by the sources	Donepezil 5 mg daily; rivastigmine 1.5 mg twice daily orally or a 4.6 mg/24 h patch	**Not carried by the sources**

Tolerability decides continuation: hold the lower dose for at least 1 month before escalating, give with or after food, move the dose to the evening for dizziness and to the morning for insomnia, and maintain fluids during diarrhoea.

With rivastigmine patches, remove the old patch before applying the new one, because failing to do so produces cumulative overdose, and rotate the site.

INDIAN PRACTICE NOTE

Methylphenidate is regulated under the narcotics statute and the restricted schedule of the Drugs and Cosmetics Rules, needing special licensing, dedicated registers, preservation of records and dispensing from authorised pharmacies.

Those constraints are why atomoxetine, a non-controlled drug on the ordinary schedule, is frequently used as initial pharmacotherapy in Indian clinics: **the answer examiners want here is regulatory, not pharmacological.**

Clonidine is used off-label where hyperactivity, severe insomnia, tics or clozapine-induced sialorrhoea dominate, with sedation, hypotension and rebound hypertension as the concerns; modafinil treats narcolepsy and excessive sleepiness and can aggravate anxiety.

HOLD THIS

Stimulants work on the first day; atomoxetine takes two to four weeks. And cholinesterase inhibitors cause diarrhoea while memantine causes constipation.

WHAT EARNS THE MARKS

- **Cardiac screening offered before the prescription**: family history of premature sudden death is the item examiners listen for.
- **Atomoxetine chosen on regulatory grounds**: it is the honest answer about what a clinic here can dispense.
- **The poor-metaboliser adjustment**: escalate only after 4 weeks, because exposure is roughly tenfold.
- **Diarrhoea against constipation for the dementia drugs**: opposite gut effects, and a one-line discriminator.

SEE ALSO 32 Children, adolescents and the elderly 28 Interactions: CYP450, smoking, grapefruit, binding 33 Essential psychotropics in India

Addiction pharmacology

23 · Alcohol: naltrexone, acamprosate, disulfiram

ADDICTION · TOPIC 23

Which anti-craving drug would you prescribe after detoxification, and how do you justify the choice?

LONG CASE, ALCOHOL DEPENDENCE | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1 Place pharmacotherapy	Alongside psychosocial treatment, by severity.
2 Mechanism for each	Opioid reward, glutamate normalisation, aldehyde block.
3 Quote one effect size	Relative risk or odds ratio, with its outcome.
4 Decide by organ	Liver points to acamprosate, kidney to naltrexone.
5 Set the conditions	Abstinence interval, supervision, monitoring.

Pharmacotherapy sits alongside psychosocial intervention, offered in moderate to severe disorder or in mild disorder on request. Global single-year prevalence exceeds 100 million people, and 2.6 million deaths, that is 4.7% of global mortality, are attributed annually to alcohol.

DRUG	MECHANISM AND DOSE	EFFICACY	CAUTIONS AND MONITORING
Naltrexone, oral	Mu-opioid antagonism blunting dopaminergic reward; 50 mg daily, with trial efficacy up to 100 mg	**Relative risk 0.93 (0.87 to 0.99) for return to any drinking and 0.81 (0.72 to 0.90) for heavy drinking; weighted mean differences -5.1 in drinking days and -4.3 in heavy drinking days**	**Avoided on any opioid agonist or analgesic, because blockade precipitates withdrawal; avoided in acute hepatitis or failure; liver enzymes at baseline, within weeks and every 6 months**

DRUG	MECHANISM AND DOSE	EFFICACY	CAUTIONS AND MONITORING
Naltrexone, long-acting	380 mg intramuscularly into the gluteal muscle every 4 weeks	Reduces drinking days per month by a weighted mean of -2 and heavy drinking days by -1.2; started before discharge it gave 3-month adherence of 41% against 27% orally	**Nausea 33%, fatigue 20%, appetite loss 13%, and injection-site reactions in up to 65%**
Acamprosate	Metabotropic glutamate-5 modulation normalising the post-withdrawal hyperglutamatergic state; 666 mg three times daily, or 333 mg twice daily below 60 kg	**Odds ratio 1.86 (1.49 to 2.33) for continuous abstinence at 12 months, relative risk 0.88 (0.83 to 0.93) for return to drinking, weighted mean difference -8.3 in drinking days**	**Renally excreted unchanged: 333 mg three times daily at a clearance of 30 to 50 mL/min, contraindicated at 30 mL/min or less, and preferred in advanced cirrhosis**
Disulfiram	Irreversible aldehyde dehydrogenase inhibition competing with nicotinamide adenine dinucleotide; 250 to 500 mg daily for 1 to 2 weeks then 250 mg daily, range 125 to 500 mg	**Supervised administration gives relative risk 1.71 (1.39 to 2.10) for abstinence and 0.19 (0.10 to 0.35) for heavy drinking**	**Needs 48 hours of verified abstinence before the first dose, and sensitivity persists up to 14 days after stopping; inhibits CYP2E1, raising warfarin, phenytoin and theophylline**

Targeted as-needed naltrexone, in younger drinkers defined by 4 or more heavy drinking days across 4 weeks, cut the likelihood of intoxication by nearly 23%.

In the COMBINE trial fivefold transaminase elevations occurred in 11 of 614 participants, resolving in 9 who stopped drinking. Disulfiram has 80% oral bioavailability after a 350 mg dose, and approximately 20% remains in the body for 1 to 2 weeks.

DISULFIRAM METABOLITE	ACTION	CONSEQUENCE WITHOUT ANY ALCOHOL
Parent drug	Blocks aldehyde dehydrogenase; inhibits CYP2E1	**A 5 to 10-fold rise in acetaldehyde on ethanol ingestion**
Diethyldithiocarbamate	Copper chelation with dopamine beta-hydroxylase inhibition	**Noradrenaline depletion with central dopamine accumulation: hypomania, psychosis, parkinsonism**
Carbon disulfide	Interferes with pyridoxal-5-phosphate, inhibiting GABA synthesis	**Benzodiazepine-resistant seizures and status epilepticus, delirium, hepatotoxicity**

RULE **Naltrexone and acamprosate change the urge; disulfiram changes the consequence.** Disulfiram does not alter craving, which is why it needs supervision and insight, and why it is wrong for a patient who cannot understand the warning.

HOLD THIS

Acamprosate is renal, naltrexone is hepatic, disulfiram is neither safe in liver disease nor useful without supervision. That line decides agent choice in most vignettes.

WHAT EARNS THE MARKS

- **A mechanism attached to each drug**: reward, glutamate, consequence, in that order.
- **One quantified benefit**: an odds ratio or relative risk shows the choice rests on trial evidence.
- **Organ function driving the choice**: acamprosate renal, naltrexone hepatic, and the thresholds.
- **The 48 hour abstinence rule before disulfiram**: with sensitivity persisting up to 14 days after stopping.

SEE ALSO 24 The disulfiram reaction, and choice by comorbidity 25 Opioids and tobacco 31 Renal, hepatic, cardiac, epilepsy, HIV

24 · The disulfiram reaction, and choice by comorbidity

ADDICTION · TOPIC 24

He drank while on disulfiram and is now flushed and hypotensive. How do you manage him?

EMERGENCY VIVA TABLE | SHORT CASE, DRUG REACTION

THE SHAPE OF THE ANSWER

1 Recognise the crisis	Low ethanol threshold, onset in 10 to 30 minutes.
2 Grade the severity	Flushing and headache, through collapse and arrhythmia.
3 Resuscitate in order	Position, oxygen, fluid, then a direct alpha agonist.
4 Exclude infarction	Electrocardiogram with serial biomarkers.
5 Choose differently next time	Agent by comorbidity, liver, kidney, seizures, cognition.

An acetaldehyde crisis with a startlingly low threshold. Symptoms can be triggered by blood ethanol as low as 5 to 10 mg/dL, begin within 10 to 30 minutes and persist for hours up to a day.

- **Early picture.** Facial flushing, throbbing headache, diaphoresis, dyspnoea, tachycardia, palpitations, nausea and vomiting.
- **Moderate to severe.** Orthostatic hypotension, confusion, vertigo and chest pain as noradrenaline depletion amplifies the vasodilation, and at the severe end circulatory collapse, ST depression, arrhythmia, infarction, seizures and death.
- **Look-alike exposures.** Similar reactions follow metronidazole, cephalosporins, sulfonamides, nitrofurantoin, chloramphenicol, griseofulvin, carbamate pesticides, industrial degreasing solvents, the inky cap mushroom and chloral hydrate.

The emergency sequence. Head-down positioning, oxygen and continuous cardiac and pressure monitoring; an immediate twelve-lead electrocardiogram with serial biomarkers to exclude infarction in anyone with chest pain or hypotension.

- **Then pressure support.** Aggressive isotonic crystalloid, and a direct alpha-adrenergic agonist such as noradrenaline if fluid fails, because indirect sympathomimetics may not work with depleted presynaptic stores.
- **Symptomatic and refractory.** Intravenous diphenhydramine for flushing and a parenteral antiemetic; and in refractory shock or ischaemic change, fomepizole as a single intravenous dose of 7.5 mg/kg,

which inhibits alcohol dehydrogenase and halts acetaldehyde generation.

COMORBIDITY	FIRST CHOICE	AVOIDED
Decompensated cirrhosis	**Acamprosate, with topiramate or naltrexone only with extreme caution**	Disulfiram
Compensated cirrhosis	Naltrexone or acamprosate	Nothing strictly barred
Seizure disorder	**Topiramate, for its intrinsic anticonvulsant action**	Disulfiram, because vomiting and electrolyte shift lower the threshold
Clearance 30 to 50 mL/min	Naltrexone or topiramate, with acamprosate at 333 mg three times daily	Standard-dose acamprosate
Clearance 30 mL/min or less	Naltrexone or disulfiram	**Acamprosate, which is renally eliminated**
Comorbid opioid use or therapy	Naltrexone only after a verified opioid-free interval	**Naltrexone during active agonist therapy**
Cognitive impairment	Acamprosate or naltrexone, or baclofen	**Topiramate, which impairs cognition and word retrieval; disulfiram, where the warning cannot be understood**
Pregnancy	Psychosocial therapy, with acamprosate or naltrexone if heavy drinking persists past 4 to 6 weeks	Disulfiram; stop naltrexone several weeks before delivery to preserve analgesia options

Sustaining pharmacotherapy beyond 3 months in decompensated cirrhosis reduces mortality, 31 against 34 percent.

Second-line agents, and when to move. Switch, combine across mechanisms, or move to second line when a first-line drug fails after 4 to 6 months, and continue for at least 1 year where goals are met before a gradual taper.

- **Topiramate.** Blocks sodium channels, potentiates GABA A and antagonises AMPA and kainate receptors: start 25 mg daily and titrate over 8 weeks to a maximum of 300 mg, accepting paraesthesia, word-finding difficulty, cognitive slowing, anorexia and dizziness.
- **Baclofen and gabapentin.** Baclofen, a GABA B agonist cleared unchanged by the kidney, is second line in advanced liver disease. Gabapentin, at 900 to 3600 mg daily, reduces heavy drinking days with a Hedges g of -0.64 and works best where protracted withdrawal, anxiety or insomnia dominate, with misuse liability.
- **Investigational.** Once-weekly semaglutide 2.4 mg reduced heavy drinking days over 26 weeks, -41% against -26%; intravenous ketamine shows preliminary benefit; ondansetron works in early-onset disorder, that is onset before age 25 years; nalmefene is approved in the European Union for targeted use; psilocybin-assisted psychotherapy reduced heavy drinking days to 9.7% against 23.6%.
- **One negative finding.** Serotonin reuptake inhibitors do not improve drinking outcomes and may worsen them.

HOLD THIS

Ten to thirty minutes, flushing and a throbbing headache, then hypotension from noradrenaline depletion. Fluid first, a direct alpha agonist second, fomepizole 7.5 mg/kg when refractory.

WHAT EARNS THE MARKS

- **The ethanol threshold quoted**: 5 to 10 mg/dL explains why a cough syrup can set it off.
- **A direct alpha agonist chosen deliberately**: presynaptic stores are depleted, so an indirect one may fail.
- **Infarction excluded, not assumed absent**: chest pain here needs an electrocardiogram and biomarkers.
- **A comorbidity-driven alternative offered**: it converts an emergency answer into a management plan.

SEE ALSO 23 Alcohol: naltrexone, acamprosate, disulfiram 31 Renal, hepatic, cardiac, epilepsy, HIV 41 New and pipeline drugs

25 · Opioids and tobacco

ADDICTION · TOPIC 25

How would you start buprenorphine in a man using heroin daily, and what could go wrong?

LONG CASE, OPIOID DEPENDENCE | OSCE STATION ON INDUCTION

THE SHAPE OF THE ANSWER

1 State the principle	Long-acting agonist replaces a short-acting one.
2 Explain the receptor	High affinity, partial agonism, and the ceiling.
3 Time the induction	Wait for objective withdrawal, rated on a scale.
4 Compare methadone	Accumulation during induction, and the QTc.
5 Dispense within the law	Observed therapy, register, narcotics schedule.

Agonist maintenance replaces a short-acting illicit opioid with a long-acting one to suppress withdrawal, abolish craving and remove euphoria.

- **Buprenorphine.** A high-affinity partial mu agonist and kappa antagonist, given sublingually because oral ingestion is extensively metabolised on first pass, and its partial agonism produces a ceiling on respiratory depression.
- **The same affinity is the hazard.** Given while mu receptors are occupied by a full agonist it displaces that agonist and precipitates severe withdrawal, so induction requires objective early to moderate withdrawal rated on the Clinical Opiate Withdrawal Scale.
- **Why naloxone is in the tablet.** Combination deters injection, because naloxone is not absorbed sublingually but precipitates withdrawal if dissolved and injected.
- **Methadone.** A long-acting full mu agonist and non-competitive N-methyl-D-aspartate antagonist given as an oral solution; its long and variable terminal half-life means tissue accumulation during induction, which is the dangerous phase, and it carries dose-dependent QTc prolongation, so baseline and periodic electrocardiography is recommended.
- **Naloxone.** A pure competitive antagonist at mu, kappa and delta receptors with onset within minutes and a plasma half-life often shorter than the offending opioid, so the patient is watched for re-narcotisation.

TRAP Three examiner-critical numbers here are absent from the sourced material: the withdrawal-scale score at which induction can safely begin, the buprenorphine maintenance range, and the methadone initial, maintenance and half-life figures. Give the induction principle, the ceiling effect and the monitoring, and **verify before the exam** against a national substitution guideline.

AGENT	LISTED FORM	TIER AND NOTE
Buprenorphine	Sublingual tablet 0.4 mg	**All three tiers, so a primary health centre officer can now treat opioid dependence**
Buprenorphine with naloxone	Sublingual 0.4 mg with 0.1 mg, and 2 mg with 0.5 mg	**All three tiers; the naloxone component deters injection and enables take-home dosing**
Nicotine	Oral dosage forms 2 mg and 4 mg	All three tiers; combining a patch with a short-acting oral form beats either alone
Naloxone	Injection 0.4 mg/mL	Listed outside the psychiatric section, under specific antidotes
Bupropion and varenicline	Not listed in India	**Both appear on the global model list, which is one of the omissions worth naming**

INDIAN PRACTICE NOTE

Substitution centres established under the national AIDS control and drug de-addiction programmes dispense directly observed therapy daily; sublingual buprenorphine, alone or with naloxone, is the primary agent in public sector centres, oral methadone is dispensed at accredited tertiary centres, and take-home doses are restricted.

Buprenorphine is controlled twice over: the restricted prescription schedule governs the pharmacy register, while the narcotics statute schedules the molecule.

Listing and primary-tier approval do not displace that layer, and presenting the tier change as a pure access reform without naming the second statute loses the mark. The extra licence a substitution centre must hold is not carried by the sourced material.

Tobacco, completing the answer. Replacement therapy delivers controlled nicotine without combustion carcinogens, carbon monoxide or tar, using a basal transdermal patch with a short-acting oral form for breakthrough craving, and is used cautiously immediately after myocardial infarction, in severe arrhythmia and in unstable angina.

- **Bupropion.** Inhibits dopamine and noradrenaline reuptake and antagonises nicotinic receptors non-competitively, easing withdrawal dysphoria and blunting post-cessation weight gain.
- **Bupropion is contraindicated.** With an active seizure disorder or predisposing lesion, a history of anorexia or bulimia nervosa, abrupt cessation of alcohol, benzodiazepines or antiepileptics, or a monoamine oxidase inhibitor.
- **Varenicline.** An alpha-4 beta-2 nicotinic partial agonist that relieves withdrawal through moderate mesolimbic dopamine release while blocking nicotine during a lapse, causing nausea, sleep

disturbance, vivid dreams and headache, with monitoring for agitation, depressed mood and suicidal ideation.

HOLD THIS

High affinity plus partial agonism explains both the safety ceiling and the precipitated withdrawal. One receptor property, two clinical consequences, and the induction rule follows from the second.

WHAT EARNS THE MARKS

- **Induction timed to objective withdrawal**: it is the single step that prevents a precipitated withdrawal.
- **Ceiling effect and displacement derived from one property**: high affinity with partial agonism.
- **Methadone's induction phase named as the dangerous one**: accumulation, not the maintenance dose, is what kills.
- **Re-narcotisation anticipated after naloxone**: the antagonist can outlast neither a depot nor a long-acting agonist.

SEE ALSO 23 Alcohol: naltrexone, acamprosate, disulfiram 33 Essential psychotropics in India 38 Rapid tranquillisation and acute agitation

Adjuncts, special populations, interactions and basics

26 · Kinetic basics the examiner opens with

BASICS AND ADJUNCTS · TOPIC 26

Define half-life for me, and tell me when this drug reaches steady state.

OPENING VIVA QUESTION, PSYCHOPHARMACOLOGY | THEORY TO BEDSIDE LINK

THE SHAPE OF THE ANSWER

1 Separate the two words	Kinetics is body on drug, dynamics is drug on body.
2 Volume of distribution	Apparent, not anatomical, and what shifts it.
3 Write the relationship	Half-life as a ratio of volume to clearance.
4 Steady state and loading	Three to five half-lives; loading changes the calendar.
5 Link to a receptor	Each blockade bought with its own adverse effect.

Pharmacokinetics is what the body does to the drug; pharmacodynamics is what the drug does to the body.

- **Volume of distribution is apparent, not anatomical.** Dose divided by plasma concentration, so a value above 1 L/kg means most of the drug has left plasma for fat, membranes and intracellular sites. It rises with fluid retention, ascites and in children, and falls with dehydration and in the elderly.
- **Three relationships to be able to write down.** Clearance as volume of distribution multiplied by the elimination rate constant; the rate constant as 0.693 divided by the half-life; and therefore the half-life as 0.693 multiplied by volume of distribution and divided by clearance. Clearance itself is the volume cleared per unit time.
- **Why the last one is examinable.** It shows half-life is a derived hybrid parameter, not a physiological constant.

HALF-LIVES ELAPSED	APPROACH TO STEADY STATE	WASHOUT AFTER STOPPING
1, 2, 3	50%, 75%, 87.5%	50%, 75%, 87.5% eliminated
4	93.75%	Approximately 94% eliminated
5	**Approximately 97%**	**Practically complete**

Steady state arrives in 3 to 5 half-lives, or 4 to 5 by another account, independent of dose or frequency: dose sets the plateau, half-life sets the calendar.

Average steady-state concentration is bioavailability multiplied by dose, divided by clearance multiplied by the dosing interval, equivalently the area under the curve divided by the interval. Fluctuation follows the ratio of interval to half-life: a drug with a 4-hour half-life given at a 12-hour interval loses 87.5% between doses.

A loading dose, volume of distribution multiplied by target concentration divided by bioavailability, reaches target at once; in one model 60 mg loaded against 30 mg daily reached a target of 10 ng/mL within 1 to 2 days rather than after a 10-day wait.

TRAP A loading dose produces target concentrations, not true kinetic steady state, so an accurate maintenance dose must follow or levels drift over 3 to 5 half-lives. Two further items the sourced chapter does not carry: zero-order or saturation elimination with its drug examples, and the whole set of therapeutic drug monitoring figures, so **verify before the exam**.

Receptor pharmacology, compressed. D2 antagonism buys antipsychotic effect and costs movement and prolactin; D2 partial agonism buffers high and low tone and costs akathisia; 5-HT2A antagonism spares movement; H1 blockade sedates and adds weight; alpha-1 blockade drops pressure; M1 blockade dries and confuses.

- **The dopamine hypothesis, in three moves.** From subcortical overactivity, to a regional split of mesolimbic D2 excess with prefrontal D1 deficiency, to striatal dysregulation as a final common pathway, with raised presynaptic synthesis capacity and sensitised release localised to the dorsal associative striatum.
- **Its four shortcomings.** The therapeutic lag against immediate occupancy, treatment resistance responding to low-affinity clozapine, the wider psychotomimetic spectrum of N-methyl-D-aspartate antagonists, and equivocal post-mortem findings.
- **The glutamate account closes the loop.** Receptor hypofunction on prefrontal parvalbumin interneurons disinhibits pyramidal output, and hyperactive hippocampal projections to the accumbens drive excessive dopamine burst firing.

HOLD THIS

Half-life is not a constant, it is a ratio. Volume of distribution over clearance, times 0.693. Say that and the dehydration, loading-dose and steady-state questions answer themselves.

WHAT EARNS THE MARKS

- **Volume of distribution called apparent**: it is the word that shows the concept is understood, not recited.
- **Half-life derived rather than quoted**: volume over clearance answers dehydration and ascites questions at once.
- **Three to five half-lives, independent of dose**: dose sets the plateau, half-life sets the calendar.
- **A loading dose distinguished from steady state**: target concentration now, with maintenance to follow.

SEE ALSO 08 Lithium: kinetics, levels and the sampling rule 19 Benzodiazepines: kinetics and equivalence 28 Interactions: CYP450, smoking, grapefruit, binding

27 · Anticholinergics, propranolol and the burden scale

BASICS AND ADJUNCTS · TOPIC 27

He has been on trihexyphenidyl with haloperidol for two years. Would you continue it?

LONG CASE PRESCRIPTION REVIEW | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1 **Mechanism and dose**	Muscarinic block restores striatal balance.
2 **Refuse routine prophylaxis**	Audit rates, and the principles that replace it.
3 **Name the misuse liability**	Dopaminergic facilitation at higher doses.
4 **Score the burden**	Three or more is high risk, with the cognitive cost.
5 **Offer alternatives**	Propranolol for akathisia, amantadine without muscarinic load.

Trihexyphenidyl is a non-selective muscarinic antagonist with pronounced M1 action. D2 blockade in the nigrostriatal pathway creates a relative cholinergic excess, and blocking striatal muscarinic receptors restores the balance; at higher doses the drug also gains dopaminergic affinity, which is the basis of its abuse liability.

- **Onset and duration.** Onset within 1 hour, duration 6 to 12 hours, and clinical response does not track plasma concentration.
- **Dosing by indication.** Drug-induced parkinsonism 1 mg/day to 2 mg/day rising to 2 to 20 mg/day in divided doses; refractory tremor 6 mg to 15 mg per day; childhood-onset dystonia 6 mg/day to over 60 mg/day, where 67% improved and 68% still benefited after 2.4 years at a mean of 40 mg daily.

Prophylaxis, the Indian question. Routine universal co-prescription with first-generation antipsychotics is common in audits and advised against: one audit found 82.6% co-prescription, an Indian tertiary outpatient survey over a 12-month period found 92% alongside risperidone, and a comparable survey elsewhere found 22%.

- **Reduce the antipsychotic first.** Reducing the dose after stabilisation often resolves parkinsonism without any anticholinergic.
- **Do not stack muscarinic load.** Drugs with strong intrinsic muscarinic antagonism, that is chlorpromazine, olanzapine and clozapine, should not receive an added one.
- **Cover after a depot is time-limited.** It should not exceed 2 weeks, because peak vulnerability falls within the first 1 week and diminishes markedly after 10 days.
- **Where dystonia is documented.** Supply a limited as-required quantity, for example 10 doses per month.

Abuse and withdrawal. Trihexyphenidyl carries the highest misuse liability among prescribed anticholinergics, through muscarinic blockade with secondary dopaminergic facilitation, with reported prevalence from 1.1% in community surveys to 34% in psychiatric cohorts.

- **The screening study.** In 64 outpatients screened on a 10-item instrument with a cut-off score of 3, 25.00% met criteria for abuse, 12.5% in the harmful band and 12.5% dependent, with male sex ($p = 0.032$) and past substance use ($p = 0.014$) as significant risks.
- **Review and taper.** Review any adjunct at least every 3 months with a planned taper: abrupt cessation produced overt withdrawal in 46.70% of an outpatient cohort, with rigidity 14.10%, somnolence 12.50%, sialorrhoea 6.20%, tremor and bradykinesia 4.70% each, and rebound insomnia 3.10%.

BURDEN TIER	REPRESENTATIVE DRUGS	RISK
Score 1, possible	Aripiprazole, paliperidone, risperidone, haloperidol, bupropion, alprazolam, diazepam, venlafaxine, cetirizine, ranitidine	Additive contribution to the total
Score 2, definite	Amantadine, carbamazepine, oxcarbazepine, loxapine, pimozide, cyproheptadine, pethidine	Counts with score 3 toward the definite total
Score 3, definite	**Trihexyphenidyl, benztropine, atropine, chlorpromazine, clozapine, olanzapine, quetiapine, thioridazine, amitriptyline, clomipramine, imipramine, nortriptyline, paroxetine, promethazine, hydroxyzine, diphenhydramine, oxybutynin, orphenadrine**	**Each definite drug raises 6-year cognitive impairment risk by 46%**

A cumulative score of 3 or more defines high risk for cognitive impairment, falls and mortality; each point corresponds to a fall of 0.33 points in Mini-Mental State Examination score over 2 years and a 26% increase in mortality risk. Vulnerability is greatest from 60 years of age.

Toxicity runs system by system: delirium and hallucination centrally, closed-angle glaucoma ocularly, xerostomia with parotitis, ileus and impaction, acute retention, tachycardia and arrhythmia, and anhidrosis with fatal hyperthermia. Reversal of severe central toxicity is intravenous physostigmine, with the dose and its cardiac cautions not carried by the sources.

Propranolol and the alternatives. Centrally active beta blockade, chiefly propranolol, is first line for acute akathisia, while anticholinergics are ineffective unless parkinsonian signs coexist; propranolol is also used in essential tremor alongside metoprolol and nadolol.

- **Benztropine and promethazine.** Benztropine adds antihistaminic action; promethazine is a phenothiazine antihistamine with potent muscarinic antagonism.
- **Biperiden and orphenadrine.** Biperiden is a selective M1 antagonist available parenterally for dystonic crisis with lower abuse liability; orphenadrine has a narrow therapeutic index with high fatality in overdose, which matters in a suicidal patient.
- **Amantadine.** It relieves extrapyramidal symptoms without muscarinic blockade, avoiding constipation, blurred vision and anhidrosis. The bedside differentiation of the four extrapyramidal syndromes is given in full earlier in this note.

TRAP Anticholinergic delirium read as an exacerbation of schizophrenia. Visual hallucination, fluctuating orientation and purposeless plucking in a patient on an antipsychotic with an anticholinergic is antimuscarinic toxicity, and the discriminators are peripheral: xerostomia, mydriasis, flushed dry skin, tachycardia and retention. Escalating the antipsychotic here can produce fatal hyperthermia or coma.

INDIAN PRACTICE NOTE

In a hot climate the anhidrosis matters more than the dry mouth: trihexyphenidyl with physical labour or exertion can precipitate fatal hyperpyrexia among manual labourers and agitated inpatients, and the picture is readily mistaken for neuroleptic malignant syndrome even though it is peripheral failure of heat dissipation.

Trihexyphenidyl 2 mg tablets are on the essential list, under the antiparkinsonism section rather than the psychiatric one.

HOLD THIS

Review every three months and taper; do not run it indefinitely. A definite-burden drug costs 46% more cognitive impairment over six years, and nearly half of patients show withdrawal phenomena on abrupt cessation.

WHAT EARNS THE MARKS

- **Routine prophylaxis declined with a reason**: lowering the antipsychotic dose often removes the need.
- **A review interval attached to the adjunct**: three months with a planned taper, not indefinite continuation.
- **Anticholinergic burden scored, not described**: a cumulative 3 marks high risk of impairment, falls and death.
- **Anhidrosis raised for a hot climate**: heat dissipation failure mimics the malignant syndrome and kills manual workers.

SEE ALSO 03 Extrapyramidal syndromes and tardive dyskinesia 04 Prolactin, metabolic risk, QTc, anticholinergic load 32 Children, adolescents and the elderly

28 · Interactions: CYP450, smoking, grapefruit, binding

BASICS AND ADJUNCTS · TOPIC 28

Your clozapine patient is admitted to a smoke-free ward. What happens to his level, and what do you do?

LONG CASE, INTERACTION QUESTION | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1 Name the mechanism	Hydrocarbons induce CYP1A2; nicotine does not.
2 Quantify the change	Dose differences for clozapine and olanzapine.
3 Give the ward protocol	Pre-emptive reduction across the first week.
4 Read the enzyme table	Substrate, inhibitor, inducer, by isoenzyme.
5 Correct the binding myth	Displacement rarely matters; inhibition does.

It is the smoke, not the nicotine. Polycyclic aromatic hydrocarbons activate the hepatic aryl hydrocarbon receptor, inducing CYP1A2 predominantly and also CYP1A1, CYP2A6, CYP2B6 and CYP2E1.

- **Why replacement therapy does not help here.** Nicotine does not induce, and replacement delivers nicotine without the hydrocarbons, so patients who switch to a patch lose induction at the same rate as unassisted quitters.

- **Passive exposure counts.** Children exposed to parental smoking of at least 20 cigarettes daily show an estimated 50% increase in theophylline clearance.
- **Context.** Smoking prevalence is 2-fold to 3-fold higher in schizophrenia, with 58% to 88% active smokers.

DRUG	PRIMARY META-ANALYSIS	REGULATORY BULLETIN
Clozapine	**A 50% lower dose in non-smokers than smokers, from 4 studies in 196 patients; clearance is 1.45-fold higher in smokers, and the concentration-to-dose ratio lower by a weighted mean of 1.11**	Levels in smokers are approximately 80% of non-smokers, with a reduction of about 36% within 1 week of quitting
Olanzapine	**A 30% lower dose, from 7 studies in 1094 patients; clearance 55% higher in smokers, ratio lower by 0.75**	Cites a 5-fold lower concentration and recommends 36% over the first 1 week
Caffeine, warfarin, theophylline	Caffeine clearance rises by more than 50%; theophylline clearance by 58% or more with half-life falling 63%	Halve caffeine on quitting; reduce warfarin by 14% to 23% with monitoring of the international normalised ratio

Where a primary meta-analysis and a secondary bulletin disagree, name both and say which takes precedence.

Clozapine kinetics for reference: 27% to 50% reaches the circulation unchanged, half-life 9 to 17 hours, steady state after 7 to 10 days, response at 250 to 400 ng/mL with a cut-off of 350 ng/mL (sensitivity 72%, specificity 70%), seizure risk above 500 to 600 mg/day.

Olanzapine: bioavailability approximately 60%, half-life 33 hours, response at 20 to 50 ng/mL, with CYP1A2 accounting for 50% to 60% of metabolism.

The ward protocol when a smoker on either drug is admitted to a smoke-free unit: establish baseline consumption, anticipate loss of induction over 1 to 2 weeks, and pre-emptively reduce clozapine by 30% to 50% and olanzapine by 30% to 36% across the first week.

- **Two adjuncts to the protocol.** Use replacement therapy for cravings while remembering it does not prevent the rise, and halve caffeine intake so caffeinism is not mistaken for nicotine withdrawal.
- **What a rising level looks like.** Rising clozapine gives clonic seizures, drowsiness, extreme sedation, hypersalivation, confusion and hallucination; rising olanzapine gives sedation, anticholinergic effects and orthostasis.

ISOENZYME	SUBSTRATES	INHIBITORS	INDUCERS
CYP1A2	Clozapine, olanzapine, duloxetine, caffeine, theophylline	**Fluvoxamine, ciprofloxacin, oestrogen-containing contraceptives**	**Tobacco smoke, carbamazepine, phenobarbital**
CYP2D6	Risperidone, haloperidol, aripiprazole, atomoxetine, venlafaxine, tricyclics	**Paroxetine, fluoxetine, bupropion, quinidine**	**Not clinically inducible; set by genetic polymorphism**
CYP3A4	Clozapine, quetiapine, ziprasidone, lurasidone, aripiprazole, alprazolam, diazepam	Ketoconazole, clarithromycin, ritonavir, and moderately grapefruit juice and verapamil	Carbamazepine, phenytoin, rifampicin, and moderately oxcarbazepine and St John's wort

Grapefruit. Furanocoumarins irreversibly inhibit intestinal CYP3A4 and mucosal P-glycoprotein with negligible hepatic effect at dietary quantities, so first-pass extraction falls and oral bioavailability and peak concentration rise for quetiapine, lurasidone, ziprasidone, oral midazolam, triazolam, alprazolam and buspirone, giving sedation, orthostasis and psychomotor impairment.

- **Protein binding, corrected.** Classical teaching overstates displacement: displacing a drug transiently raises the unbound fraction, which immediately expands the volume of distribution and increases clearance, so steady-state free concentration is virtually unchanged unless the displacing agent also inhibits clearance.
- **The worked distinction.** Valproate inhibiting lamotrigine glucuronidation is metabolic inhibition, not displacement, and that distinction is the answer.
- **Pharmacodynamic interactions.** Watch additive serotonin toxicity, additive QTc prolongation, and stacked sedation, with reports of fatal cardiorespiratory arrest after combined parenteral olanzapine and a benzodiazepine.

TRAP Restarting clozapine after it was held for toxicity by halving the dose and nothing else. First confirm the neutrophil count and differential, confirm current registration on the monitoring system that governs rechallenge, and check how long the drug was withheld, because the length of the interruption decides whether re-titration from a low dose is required. Those requirements are named in the sources without numbers, so **verify before the exam**.

HOLD THIS

A nicotine patch does not induce CYP1A2. Stopping cigarettes raises clozapine whether or not replacement is given, so the dose comes down pre-emptively and the trough is checked.

WHAT EARNS THE MARKS

- **Smoke separated from nicotine**: it decides whether a patch protects the level, and it does not.
- **A pre-emptive reduction with a percentage**: 30% to 50% for clozapine across the first week.
- **Caffeine halved alongside**: the confound that gets read as nicotine withdrawal.
- **Displacement dismissed in favour of inhibition**: it is the interaction question most candidates answer from an old textbook.

SEE ALSO 05 Clozapine: workup and ancestry-adjusted titration 12 Carbamazepine, oxcarbazepine, lamotrigine

26 Kinetic basics the examiner opens with

29 · Pregnancy: risk drug by drug

SPECIAL POPULATIONS · TOPIC 29

She is ten weeks pregnant on sertraline and asks whether to stop. What is your advice?

LONG CASE, PERINATAL PSYCHIATRY | OSCE COUNSELLING STATION

THE SHAPE OF THE ANSWER

1 **Frame it as a comparison**	Fetal exposure against untreated maternal illness.
2 **Give the baseline**	2% to 3% malformation rate in any pregnancy.
3 **Go drug by drug**	Valproate out, lithium watched, reuptake inhibitors reassuring.
4 **Plan the neonate**	Poor adaptation, pulse oximetry, what to observe.
5 **Refuse abrupt cessation**	Relapse risk, and postpartum recurrence figures.

The decision is a comparison, not an avoidance. Weigh fetal exposure against untreated maternal illness, which is associated with impaired attachment, compromised nutrition, poor antenatal attendance and worse child development.

- **The baseline to quote first.** Most psychotropics do not raise the baseline malformation rate once confounders are controlled, and that baseline is 2% to 3%, approximately 1 in 40 pregnancies.
- **Principles of prescribing.** Preconception counselling is the ideal start, and monotherapy at the lowest effective dose is preferred because polypharmacy worsens neonatal adaptation.
- **Why not to stop abruptly.** Abrupt cessation on confirmation of pregnancy carries a high relapse risk: stopping lithium before pregnancy gives almost 3 times the recurrence rate in the first postpartum month.

DRUG OR CLASS	RISK	ACTION
Sodium valproate	**The most teratogenic psychotropic: malformations, neural tube defects, craniofacial and cardiac anomalies, dose-dependent, with long-term neurodevelopmental deficit**	**Not prescribed in pregnancy, planned pregnancy or breastfeeding, and restricted in reproductive potential under the prevention programme**
Lithium	A small defined increase in cardiovascular malformation after first-trimester exposure	Fetal cardiac echocardiography and detailed anomaly scanning; monitoring schedule as in the lithium topic
Serotonin reuptake inhibitors	**No structural teratogenicity above baseline; poor neonatal adaptation in approximately one-third after third-trimester exposure; pulmonary hypertension of the newborn beyond 20 weeks, relative risk up and absolute incidence low**	**Routine fetal or neonatal echocardiography is not supported; routine pulse oximetry before discharge is the screen**

DRUG OR CLASS	RISK	ACTION
Dual reuptake inhibitors	Poor adaptation in one-third, with a theoretical pulmonary hypertension risk	Monitor the newborn; in lactation watch drowsiness, feeding and weight gain
Tricyclics	Infant serum levels non-detectable or under 28 ng/ml with no documented effects	**Doxepin is the exception, contraindicated in lactation after neonatal respiratory depression resolved 24 hours after feeding stopped**
Antipsychotics	**Phenothiazines impair glucose tolerance; second-generation agents carry high metabolic risk with gestational diabetes; neonatal withdrawal and extrapyramidal signs near term**	**Glucose tolerance testing, consultant-led obstetric care and planned delivery in an acute unit; observe the neonate for hypertonia, hypotonia, somnolence and respiratory distress**
Lamotrigine	Low malformation rate; the hazard is kinetic, as oestrogen-driven glucuronidation raises clearance and levels fall	**Titrate up in gestation and reduce promptly after delivery; folic acid 5 mg daily; check the neonate for rash**
Carbamazepine	**Craniofacial defects, fingernail hypoplasia, malformations and neural tube defects; fetal enzyme induction depletes vitamin K-dependent factors**	**Folic acid 5 mg daily in the first trimester and vitamin K at birth; watch for vomiting, diarrhoea, restlessness and seizures**
Benzodiazepines near term	Floppy infant syndrome: hypotonia, hyporeflexia, low Apgar scores, hypothermia	Distinguish from antipsychotic extrapyramidal signs, which give hypertonia, rigidity and tremor

Poor adaptation appears shortly after delivery with jitteriness, tremor, hypotonia, irritability, feeding difficulty, hypoglycaemia and respiratory distress, resolving within days to 2 weeks on skin-to-skin contact, swaddling, a quiet nursery and frequent feeds. Pulmonary hypertension follows exposure after 20 weeks with severe distress, hypoxaemia and cyanosis, needing neonatal resuscitation.

TRAP Attributing lethargy, poor feeding or hypothermia in a neonate to maternal medication before excluding sepsis, hypoglycaemia and intracranial pathology. The paediatric workup comes first and the drug history is the last item in the differential.

HOLD THIS

Background malformation risk is 2% to 3%, the number every other figure is compared against. Say it before you say anything about a drug.

WHAT EARNS THE MARKS

- **Untreated illness costed as a risk**: the comparison, not avoidance, is the whole answer.
- **The background rate given before any drug figure**: without it no relative risk means anything.
- **Pulse oximetry named as the newborn screen**: routine echocardiography is not supported.
- **Sepsis excluded before blaming the drug**: the paediatric workup comes first in a sick neonate.

SEE ALSO 10 Lithium in pregnancy and the anti-suicidal claim 11 Valproate: toxicity and the childbearing rules 30 Lactation: what crosses, what is advised against

30 · Lactation: what crosses, what is advised against

SPECIAL POPULATIONS · TOPIC 30

She is breastfeeding and needs an antipsychotic. Which one, and what do you watch in the baby?

LONG CASE, POSTNATAL PSYCHIATRY | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1 **Milk physiology**	Hind milk, lipophilicity, timing around a feed.
2 **Three immature barriers**	Liver, kidney, blood-brain barrier.
3 **Sort the agents**	First line, cautious, advised against.
4 **Monitor clinically**	Feeding, sleep, tone, milestones, rather than an assay.

Milk physiology decides exposure; the neonatal liver and kidney decide consequence. Production averages 600 to 1,000 ml daily at a pH of 6.35 to 7.65, mature milk arriving approximately 2 weeks after delivery, and hind milk carries more lipid, so lipophilic drugs concentrate there. Dosing immediately after a feed minimises transfer.

- **Immature liver.** Hepatic cytochrome activity is roughly 50% of adult levels with uneven glucuronidation and reduced clearance for at least the first 2 weeks.
- **Immature kidney.** Filtration is only 30% to 40% of adult capacity with tubular secretion 20% to 30%, reaching adult values between the second and fifth months.
- **Immature barrier.** An immature blood-brain barrier allows concentrations in cerebrospinal fluid 10 to 30 times adult serum, with limited fat stores raising free central levels further.

CATEGORY	AGENTS	RATIONALE
Supported, first line	**Sertraline, paroxetine, olanzapine, sulpiride**	Low milk excretion, minimal infant serum levels, reassuring databases, with routine paediatric observation
Supported with caution	Fluoxetine, citalopram, lamotrigine, short-acting benzodiazepines	**Moderate transfer or long half-lives: monitor sedation, feeding, apnoea and rash, and withhold milk if a rash appears on lamotrigine until assessed**
Advised against	**Clozapine, lithium, carbamazepine, sodium valproate, doxepin**	**Agranulocytosis; high relative infant dose and toxicity; hepatotoxicity with reported cholestatic hepatitis; haematological and hepatic toxicity, with thrombocytopenic purpura and severe anaemia reported in an infant of three months; respiratory depression**

Agent detail: sertraline levels non-detectable or under 5 ng/ml with metabolite under 10 ng/ml and no effects across 49 infants, peaking between 7 and 11 hours after the maternal dose, so timing feeds around that window helps.

Paroxetine is non-detectable in 24 of 27 infants with no effects across 37; fluoxetine no effects in 180 of 190 infants, with isolated colic, crying and vomiting; citalopram measurable at 2.3 to 12.7 ng/ml.

Aripiprazole is compatible but can lower maternal prolactin and supply. Antipsychotic monotherapy is preserved where possible, because combination therapy was associated with delayed developmental scores at 12 to 18 months.

- **RULE** **An undetectable infant serum level is not reassurance on its own.** Receptor sensitivity and barrier permeability are heightened in early infancy, so clinical monitoring of feeding, sleep, tone and milestones is the standard rather than an assay.

HOLD THIS

Sertraline and paroxetine for mood, olanzapine for psychosis; clozapine, lithium, carbamazepine, valproate and doxepin advised against. Then give the neonatal reason: half the adult liver, a third of the adult kidney, a leaky barrier.

WHAT EARNS THE MARKS

- **Three neonatal barriers named with numbers**: half the liver, a third of the kidney, a leaky central barrier.
- **An agent from each category**: first line, cautious, advised against, rather than a single safe name.
- **Clinical monitoring preferred to an infant assay**: an undetectable level is not reassurance by itself.
- **Feed timing offered as a practical measure**: dosing straight after a feed reduces transfer.

SEE ALSO 29 Pregnancy: risk drug by drug 10 Lithium in pregnancy and the anti-suicidal claim 32 Children, adolescents and the elderly

31 · Renal, hepatic, cardiac, epilepsy, HIV

SPECIAL POPULATIONS · TOPIC 31

He is on maintenance haemodialysis and needs a mood stabiliser. What do you prescribe and how do you monitor it?

LONG CASE, MEDICAL COMORBIDITY | PSYCHOPHARMACOLOGY VIVA TABLE

THE SHAPE OF THE ANSWER

1	Name the four domains	Absorption, distribution, metabolism, excretion in uraemia.
2	Lead with protein binding	Free fraction expands on a normal total level.
3	Dose and sample correctly	Lithium after the run, sampled before the next.
4	Grade the liver	Child-Pugh class before any hepatic adjustment.
5	Cardiac, epilepsy, HIV	One discriminating drug choice in each.

Renal failure alters all four kinetic domains, and protein binding is the examinable one. Albumin falls and retained organic acids displace drug, so for agents bound above 90%, such as valproic acid and diazepam, the free active fraction expands even when the total level looks therapeutic.

- **The other three domains.** Absorption shifts with gastric alkalinisation and phosphate-binder chelation; distribution expands with oedema and contracts with uraemic wasting; oxidation, glucuronidation and sulfation are relatively preserved while reduction and hydrolysis slow, and intermittent dialysis leaves polar active metabolites to accumulate between runs.
- **The counterintuitive part.** Because most psychotropics are lipophilic with large volumes and hepatic clearance, the majority need no drastic reduction, and underdosing is a frequent therapeutic failure even though texts cap initial dosing at about two-thirds of the standard maximum.

DRUG	RENAL ADJUSTMENT	NOTE
Sertraline, fluoxetine, citalopram	Little change; sertraline 50 to 200 mg/day, fluoxetine 20 mg/day, citalopram 10 to 60 mg/day	**Not removed by dialysis; sertraline also reduces recurrent dialysis-induced hypotension**
Paroxetine	**Start at 10 mg/day, a 50% reduction, range 10 to 30 mg/day**	Metabolites inhibit CYP2D6, so exposure rises
Venlafaxine, mirtazapine, bupropion	37.5 to 112.5 mg/day; 7.5 to 22.5 mg/day with clearance down about 50%; bupropion 100 mg/day up to every 8 hours	**Bupropion metabolites accumulate and lower the seizure threshold; venlafaxine needs blood pressure monitoring**
Lithium	**Half-life 40 hours against 14 to 28 in health; on maintenance haemodialysis a single oral dose of 200 to 600 mg, commonly 600 mg or 300 mg for augmentation, immediately after each run**	**Draw the level immediately before the next session, because a post-dialysis sample is falsely low during tissue rebound**
Valproate	15 to 60 mg/kg/day with modest dialytic extraction	**Bound 80% to 90% normally, so measure free valproate rather than total**
Risperidone	**Halve the start to 0.5 to 1.5 mg twice daily; clearance of the active moiety falls by 60% and metabolite half-life rises to 25 hours against 19**	The active moiety is substantially renally eliminated
Haloperidol, olanzapine, quetiapine, aripiprazole	Haloperidol 1 to 2 mg every 8 to 12 hours; olanzapine 5 to 20 mg/day; quetiapine 150 to 750 mg/day; aripiprazole 10 to 30 mg/day	**Under 1% of haloperidol or aripiprazole appears unchanged in urine, so no primary renal adjustment**
Clozapine, ziprasidone, thioridazine	Clozapine 12.5 to 450 mg/day guided by levels, kept below 400 mcg/L	**Ziprasidone and thioridazine are avoided, because dialysis-associated hypokalaemia and hypomagnesaemia potentiate QTc prolongation**
Midazolam, lorazepam, zolpidem, buspirone	Zolpidem halved; buspirone reduced by 25% to 50%	**Midazolam accumulates an active glucuronide with a half-life of 50.4 to 76.8 hours against 1 to 12; lorazepam has no active metabolite yet the parent runs 32 to 70 hours against 9 to 16**

Hepatic impairment, graded first. Reduced hepatocyte mass, downregulated cytochromes, low albumin and portosystemic shunting mean first-pass metabolism is bypassed and peak bioavailability rises. Grade with the Child-Pugh classification, class A, B and C for mild, moderate and severe, and name the class before any adjustment.

- **Agents to avoid.** Nefazodone is contraindicated for unpredictable fulminant failure; duloxetine is avoided in chronic liver disease; valproate is contraindicated in severe dysfunction, acute hepatitis or a mitochondrial disorder; and carbamazepine is hepatotoxic and autoinduces.
- **The mood stabiliser that fits.** Lithium, with no hepatic clearance pathway, is the non-hepatotoxic option in cirrhosis provided renal function and fluid balance are stable.
- **Anxiolytic choice.** Avoid the oxidised long-acting agents, diazepam, chlordiazepoxide and flurazepam, whose active metabolites precipitate encephalopathy, and use the glucuronidated group.

TRAP The drug-by-drug mapping of which adjustment applies at which Child-Pugh grade, and the quetiapine and lurasidone hepatic starting doses, are not carried by the sourced material. Quote the class as the framework and the adjustment only where the prescribing text supports it, and **verify before the exam**.

Cardiac disease. Tricyclics carry quinidine-like sodium channel blockade, slowing intraventricular conduction, widening the QRS and prolonging the QTc, and are contraindicated in bundle branch block, recent infarction and heart failure.

- **The cardiac-safe choices.** Sertraline has a safety record in acute coronary syndromes and does not impair ejection fraction; venlafaxine raises blood pressure dose-dependently; and alpha-1 blockade by chlorpromazine, clozapine, quetiapine, risperidone and tricyclics produces postural drops that can provoke ischaemia.
- **Epilepsy.** Approximately 1 out of 3 patients have a comorbid mood disorder, rising to 1 out of 2 with pharmacoresistant seizures; tertiary tricyclics and maprotiline are pro-convulsant, second-generation agents are first line and do not worsen seizure control, bupropion is dose-dependently pro-convulsant, and clozapine has the highest antipsychotic liability with plasma above 400 mcg/L as the sourced marker.
- **HIV, the interaction map.** Protease inhibitors sharply raise quetiapine, aripiprazole and benzodiazepine levels, with midazolam and triazolam contraindicated alongside a boosted regimen; efavirenz induces CYP3A4 and causes vivid dreams, dizziness, depression and psychosis early; nevirapine lowers methadone, antipsychotic and valproate levels.
- **HIV, the prescribing choice.** Integrase inhibitors have minimal interaction and are preferred, though polyvalent cations including oral lithium can chelate them; and heightened basal ganglia sensitivity means aripiprazole or low-dose risperidone rather than a high-potency typical agent.

HOLD THIS

Renal failure changes the free fraction; hepatic failure changes the first pass. Measure free valproate in uraemia, sample lithium before the dialysis run, and use the glucuronidated benzodiazepines in cirrhosis.

WHAT EARNS THE MARKS

- **Free fraction raised before any dose is quoted**: it explains toxicity at a therapeutic total level.
- **Lithium sampling timed to the dialysis cycle**: a post-run sample is falsely low during tissue rebound.
- **Child-Pugh class named before adjusting**: grading first is what separates a plan from a guess.
- **Underdosing named as a failure mode**: renal caution is often overcorrected in practice.

SEE ALSO 08 Lithium: kinetics, levels and the sampling rule 19 Benzodiazepines: kinetics and equivalence 28 Interactions: CYP450, smoking, grapefruit, binding

32 · Children, adolescents and the elderly

SPECIAL POPULATIONS · TOPIC 32

How does your prescribing change in a child, and in a woman of eighty?

VIVA TABLE ON SPECIAL POPULATIONS | LONG CASE, GERIATRIC PRESCRIBING

THE SHAPE OF THE ANSWER

1	**Non-drug measures first**	Assessment, psychoeducation, behavioural management.
2	**Quote the paediatric warning**	Suicidality under 24 years, and which agents are approved.
3	**Monitor the metabolic risk**	Children are more susceptible than adults.
4	**Apply the Beers rules**	Named pairs to avoid, and the dementia restriction.
5	**State the legal route**	Name the provision rather than a section number.

At both ends of life the pharmacology is the same and the dosing rule is the opposite of adult practice. In children, specialised assessment, parental psychoeducation and non-pharmacological intervention come first, and preschool disruptive behaviour gets behavioural management before any drug.

- **The boxed warning.** Antidepressants carry a black box warning for increased suicidal ideation and behaviour in children, adolescents and young adults under 24 years of age, issued after a review of adolescent depression trials.
- **Approved agents.** Fluoxetine has the strongest evidence and approval in paediatric depression, with escitalopram also approved. Approved antipsychotic indications are schizophrenia, bipolar mania and irritability in autistic disorder, and risperidone in adolescents starts at 0.5 mg/day with a book-sourced range of 0.5 to 2.5 mg daily.
- **Metabolic vulnerability.** Children are more susceptible than adults to rapid weight gain, hypertriglyceridaemia, insulin resistance and hyperprolactinaemia with gynaecomastia and pubertal delay, so body mass index, fasting lipids, glucose and prolactin are monitored.

BEERS DOMAIN	THE RULE	WHY
Scope	Adults aged 65 years and older across acute, ambulatory and institutional settings, excluding hospice and palliative care	**Approximately 50% of community-dwelling older adults take 5 or more medications**
Opioid with benzodiazepine, or with a gabapentinoid	**Avoid both combinations, except when transitioning between agents**	Fatal overdose, respiratory depression, profound central depression
Antidepressants with a fall or fracture history	**Dual reuptake inhibitors, serotonin reuptake inhibitors and tricyclics are all potentially inappropriate**	Unsteadiness, ataxia, orthostatic syncope, recurrent falls; where unavoidable, start low and go slow
Anticholinergic burden	Avoid tertiary tricyclics, low-potency first-generation antipsychotics, chlorpromazine, hydroxyzine, diphenhydramine	Worsened cognition, retention, constipation, delirium

BEERS DOMAIN	THE RULE	WHY
Histamine-2 receptor antagonists	**Removed from the dementia list for weak evidence, retained as inappropriate in delirium or high delirium risk**	An update that tests whether the candidate has read the current version
Tramadol	Use with caution and monitor sodium at initiation and titration	High rates of hyponatraemia and inappropriate antidiuretic hormone secretion
Antipsychotics in dementia	**Restricted to severe aggression, psychosis or imminent harm resistant to non-pharmacological measures**	A black box warning for increased mortality and cerebrovascular events
Other updates	Dabigatran and rivaroxaban carry bleeding warnings at 75 years and older; the aspirin caution for primary prevention now begins at 70 years rather than 80; chronic seizures were removed	Reduced renal clearance, reduced hepatic blood flow, less body water, more relative fat, heightened central sensitivity

TRAP Three paediatric items the sourced material does not carry: the frequency and duration of early monitoring contacts after starting an antidepressant, the adolescent paliperidone starting dose, and the numbered sections of the Mental Healthcare Act 2017 governing treatment of minors and supported admission of adults. Name the provisions, not the numbers, and **verify before the exam**; quoting the adult supported-admission section for a paediatric question is a specific avoidable error.

HOLD THIS

Start low, go slow, and count the anticholinergics. The three commonest avoidable harms in the elderly are a fall, a delirium and a bleed, and each has a named drug pair behind it.

WHAT EARNS THE MARKS

- **Behavioural treatment offered before the prescription**: in preschool children it is the first-line answer.
- **Fluoxetine named as the evidenced paediatric agent**: with escitalopram as the second approved option.
- **A named Beers pair refused**: an opioid with a benzodiazepine, or with a gabapentinoid.
- **Antipsychotics in dementia restricted, with the reason**: a mortality and stroke warning, so the indication is narrow.

SEE ALSO 22 ADHD and cognition drugs 27 Anticholinergics, propranolol and the burden scale 31 Renal, hepatic, cardiac, epilepsy, HIV

33 · Essential psychotropics in India

SPECIAL POPULATIONS · TOPIC 33

Which psychiatric drugs are on the national essential medicines list, and what is missing from it?

VIVA TABLE ON HEALTH POLICY | LONG CASE, ACCESS AND AFFORDABILITY

THE SHAPE OF THE ANSWER

1 **Say what the list is**	A policy instrument with tiers, not a formulary.
2 **Count the entries**	Sixteen entities across five subsections.
3 **Quote the tier restrictions**	Clozapine, lithium, haloperidol, and why they matter.
4 **Name the omissions**	The alcohol gap is the one examiners want.
5 **Add the two statutes**	Restricted schedule register, and narcotics scheduling.

The essential medicines list is examined because it is a policy instrument, not a formulary. The fifth edition appeared in September 2022, superseding the fourth of 2015, with earlier editions in 1996, 2003 and 2011.

- **Size.** It carries 384 medicines across 27 therapeutic sections, against 376 in 2015 and 348 in 2011, with 34 drugs dropped and 26 added.
- **Inclusion criteria.** Regulatory approval, proven efficacy, safety, cost-effectiveness, alignment with national programmes and relevance to the Indian disease burden, and single agents covering several indications are preferred, which is why fluoxetine is listed twice.
- **The tier tag.** Every formulation is tagged: primary for health centres, secondary for community and district hospitals, tertiary for medical colleges and apex institutes.

SUBSECTION	MEDICINE AND STRENGTHS	TIER
Psychotic disorders	Clozapine, tablet 25 mg, 50 mg, 100 mg	**Tertiary only**
Psychotic disorders	Fluphenazine injection 25 mg/mL; risperidone tablet 1 mg, 2 mg, 4 mg, liquid 1 mg/mL, long-acting injection 25 mg and 37.5 mg	All three tiers
Psychotic disorders	Haloperidol tablet 2 mg, 5 mg, 10 mg, 20 mg, liquid 2 mg/5 mL, injection 5 mg/mL	**Secondary and tertiary**
Depressive disorders	Amitriptyline 10 mg, 25 mg, 50 mg, 75 mg; escitalopram 5 mg, 10 mg, 20 mg; fluoxetine 10 mg, 20 mg, 40 mg, 60 mg	All three tiers
Bipolar disorders	Lithium tablet 300 mg	**Secondary and tertiary**
Bipolar disorders	Sodium valproate 200 mg, 300 mg, 500 mg with modified release 300 mg and 500 mg; carbamazepine 100 mg, 200 mg, 400 mg with modified release and paediatric liquid 100 mg/5 mL	**All three tiers, carbamazepine newly integrated here from the neurology section**
Anxiety and sleep	Clonazepam 0.25 mg, 0.5 mg, 1 mg; zolpidem 5 mg, 10 mg	All three tiers
Obsessive-compulsive disorder and panic	Clomipramine 10 mg, 25 mg, 75 mg; fluoxetine as above	**All three tiers for clomipramine, but secondary and tertiary for fluoxetine in this indication**

SUBSECTION	MEDICINE AND STRENGTHS	TIER
Psychoactive substance abuse	Buprenorphine 0.4 mg; buprenorphine with naloxone 0.4 mg with 0.1 mg and 2 mg with 0.5 mg; nicotine 2 mg and 4 mg	All three tiers, and a new subsection

Sixteen therapeutic entities across five subsections, with no psychiatric drug deleted between editions.

Outside the psychiatric section sit diazepam (liquid 2 mg/5 mL, injection 5 mg/mL, suppository 5 mg), lorazepam (tablet 1 mg and 2 mg, injection 2 mg/mL and 4 mg/mL), midazolam, trihexyphenidyl 2 mg, donepezil 5 mg and 10 mg, propranolol 10 mg, 20 mg and 40 mg, naloxone injection 0.4 mg/mL and thiamine 100 mg.

That is where candidates look in the wrong place.

The omissions to name. There is no alcohol pharmacotherapy in the psychiatric section at all, neither anticraving agents nor a deterrent, and no oral formulation for outpatient detoxification.

- **Three further gaps.** Bupropion and varenicline are absent although both appear on the global model list; memantine is absent although donepezil is listed; and only two oral second-generation antipsychotics appear.
- **The tier inconsistencies matter.** Haloperidol is restricted to secondary and tertiary despite being cheap and critical for agitation, clozapine is restricted to tertiary which blocks district programme psychiatrists, and fluoxetine is available at the primary tier for depression but not for obsessive-compulsive disorder.

FEATURE	ORDINARY PRESCRIPTION SCHEDULE	RESTRICTED PRESCRIPTION SCHEDULE
Coverage	Prescription-only drugs against a registered practitioner's prescription	**Habit-forming drugs, targeted antibiotics, later-generation cephalosporins**
Record	The regular retail register	**A dedicated bound register with patient and prescriber details, registration number, drug, batch, date and quantity**
Retention	Not carried by the sourced material	**A minimum of 3 years, open to inspection**
Psychotropics	Antipsychotics, tricyclics, reuptake inhibitors, lithium, valproate, carbamazepine, trihexyphenidyl	**All benzodiazepines, the Z-drugs, buprenorphine, tramadol**

THE SECOND STATUTE, AND THE GENERIC QUESTION

The narcotics coordination list of drugs also falling under the Narcotic Drugs and Psychotropic Substances Act names buprenorphine at schedule serial number 169, and covers alprazolam, chlordiazepoxide, clobazam, clonazepam, diazepam, lorazepam, nitrazepam, midazolam, zolpidem, tramadol, pentazocine, codeine, methylphenidate, the amphetamines and the barbiturates. **Quote the serial number for buprenorphine only**, because the source prints its tables twice.

On pricing, listing is the statutory trigger for price control and the national authority fixes a ceiling price above which a retail pharmacy cannot sell, while the arithmetic behind that ceiling and the annual cap for non-scheduled formulations are not carried by the sources.

On generics, the market runs on branded generics with a national programme dispensing unbranded quality generics at lower prices.

For lithium and carbamazepine, whose concentration-toxicity curves are steep, switching brands without level monitoring is not recommended, and modified-release valproate and carbamazepine need dissolution equivalence, so a stabilised patient is told to keep brand consistency or is monitored closely if the supplier changes.

HOLD THIS

Sixteen psychiatric medicines, five subsections, three tier restrictions: clozapine tertiary, lithium secondary and tertiary, haloperidol secondary and tertiary. Then name the alcohol gap, which is the omission examiners want.

WHAT EARNS THE MARKS

- **The list described as policy with tiers**: it explains why a district psychiatrist cannot start clozapine.
- **An omission named without prompting**: no alcohol pharmacotherapy in the psychiatric section.
- **Both regulatory layers named**: the restricted prescription schedule and the narcotics statute.
- **Brand consistency advised for narrow-index drugs**: lithium and carbamazepine, with levels if the supplier changes.

SEE ALSO 05 Clozapine: workup and ancestry-adjusted titration 23 Alcohol: naltrexone, acamprosate, disulfiram 25 Opioids and tobacco

ECT and neuromodulation

34 · ECT: indications, workup and anaesthesia

ECT AND NEUROMODULATION · TOPIC 34

Why would you give this patient ECT, and what would you do before the first treatment?

LONG CASE, ECT DECISION | OSCE STATION ON CONSENT AND WORKUP

THE SHAPE OF THE ANSWER

1 **Lead with speed**	Indications where a rapid response is the argument.
2 **Contraindications**	One absolute, the rest optimised rather than excluded.

3 Review the drugs	Benzodiazepines and lithium come off first.
4 Anaesthetic sequence	Preoxygenation, induction agent, relaxant, airway.
5 Protect the heart	Vagal phase then sympathetic surge, and its cover.

Indications first, and lead with the ones where speed is the argument. Severe major depressive episodes, particularly with psychotic features, acute suicidal intent or marked psychomotor retardation; catatonia of any cause including malignant catatonia; acute mania resistant to pharmacotherapy.

- **The rest of the list.** Treatment-resistant depression across multiple failed classes; neuroleptic malignant syndrome refractory to medical management; and any situation where rapid response is needed to prevent collapse from dehydration or severe agitation.
- **Course length.** An acute index course is 6 to 12 treatments over 2 to 4 weeks.
- **Contraindications.** Intracranial hypertension is the absolute one. Relative ones are a space-occupying lesion with normal pressure, an intracranial aneurysm, recent myocardial infarction, unstable angina, congestive failure, severe glaucoma, retinal detachment, recent fractures, thrombophlebitis and pregnancy, each managed by optimisation rather than exclusion.

STEP	WHAT IS DONE
Medication review	**Taper or stop benzodiazepines, which raise the threshold and shorten the seizure; stop lithium, which risks neurotoxicity, severe delirium and prolonged cognitive impairment; monitor tricyclics, monoamine oxidase inhibitors and reuptake inhibitors cautiously**
Premedication	Avoid routine sedative premedication, which prolongs emergence; give glycopyrrolate or atropine to reduce secretions and prevent vagal bradycardia or asystole; ondansetron where there is a history of post-treatment vomiting
Oxygenation	**Preoxygenate with pure oxygen by mask for 5 minutes, then positive-pressure bag-mask ventilation from apnoea until spontaneous breathing returns**
Induction	**Methohexital 1 mg/kg to 1.5 mg/kg intravenously as the standard, for minimal anticonvulsant effect; propofol 1 mg/kg for faster recovery and antiemesis at the cost of a shorter seizure; alternatives thiopental, etomidate (stable but painful and emetogenic) or ketamine**
Relaxation	Succinylcholine 1 mg/kg to abolish the contractions that could fracture bone or vertebrae; cisatracurium, or rocuronium reversed with sugammadex, where it is contraindicated
Haemodynamics	**Initial vagal bradycardia then sympathetic tachycardia and hypertension for 5 to 10 minutes; pre-stimulus labetalol 10 to 50 mg or esmolol 40 to 80 mg intravenously, or magnesium sulfate, in ischaemic heart disease; ketorolac for headache and myalgia afterwards**

TRAP The pre-procedure fasting durations for solids and for clear fluids are asked at almost every table and are not carried by the sourced material. State that fasting is to minimise aspiration under general anaesthesia and **verify before the exam** against your unit's anaesthetic protocol.

HOLD THIS

Intracranial hypertension is the only absolute contraindication; pregnancy and cardiac disease are relative. Candidates lose the mark by promoting a relative contraindication to an absolute one.

WHAT EARNS THE MARKS

- **Catatonia named early**: it is the indication where ECT is the treatment, not the last resort.
- **One absolute contraindication only**: promoting a relative one costs the mark.
- **Benzodiazepines and lithium addressed before the first session**: threshold and neurotoxicity respectively.
- **The haemodynamic sequence anticipated**: bradycardia first, then a sympathetic surge needing cover in ischaemic disease.

SEE ALSO 35 Placement, stimulus dosing and seizure adequacy 36 ECT adverse effects, maintenance and the statute 18 Resistant depression: staging, STAR*D, augmentation

35 · Placement, stimulus dosing and seizure adequacy

ECT AND NEUROMODULATION · TOPIC 35

Which electrode placement would you choose, what dose, and how do you know the seizure was adequate?

ECT VIVA TABLE | OSCE STATION ON THE ECT RECORD

THE SHAPE OF THE ANSWER

1 **Choose the placement**	Cognitive cost against speed of response.
2 **Dose above threshold**	Unilateral 125% to 150%, bilateral 50%.
3 **Titrate, then restimulate**	Raise by half if the seizure is under 25 seconds.
4 **Judge quality, not length**	Amplitude, regularity, latency to slowing, suppression.
5 **Track the rising threshold**	Escalate across the course or retitrate at the sixth.

FEATURE	RIGHT UNILATERAL (D'ELIA)	BILATERAL (BITEMPORAL)
Position	Right frontotemporal with the second electrode at the parietal vertex	Symmetrical frontotemporal placement
Cognitive profile	**Significantly less retrograde and anterograde amnesia**	**Greater postictal disorientation and memory disruption**
Speed of response	Slower if the dose is barely suprathreshold	More rapid
Dose above threshold	**125% to 150% above the seizure threshold**	**50% above the seizure threshold**

Modern devices deliver bidirectional rectangular pulses, which transfer charge more efficiently than sine-wave machines.

- **Empirical threshold titration.** Done at the first session to find the minimum charge producing a generalised seizure, with the starting charge set by sex and placement, because threshold is lower in women and with unilateral electrodes.
- **If the stimulus fails.** Where the seizure does not exceed 25 seconds on the electroencephalogram, intensity is raised by 50% and the patient restimulated in the same session; in the study cohort a seizure over 25 seconds was elicited in every case by the fourth stimulation.
- **The motor seizure.** A tonic phase of 10 to 15 seconds then a clonic phase of 32 to 50 seconds, monitored with the cuff technique on an isolated limb.

Adequacy is quality, not duration. A duration above 25 seconds is only a baseline criterion.

- **Amplitude markers.** Therapeutic responders show greater immediate poststimulus amplitude, measured across the largest 3 successive peaks in the first 10 seconds, and greater midictal amplitude across 3 contiguous slow waves.
- **Regularity, latency and suppression.** Higher midictal regularity, scored on a 7-point ordinal scale from 0 to 6; a shorter latency to slowing, that is the point at which the dominant frequency drops below 5 Hz; and abrupt, profound postictal suppression, measured over the first 6 artefact-free seconds.

The anticonvulsant effect, mediated partly through enhanced GABAergic transmission, raises the threshold across the course: by 47% to 50% between treatments 5 and 7, and by 42% to 99% by completion, with a mean rise of 40% over the first 6 treatments and at least a 50% rise in 25 of 47 patients. Older age predicts a rise.

- **Why it causes non-response.** With unilateral treatment at a fixed absolute dose, an uncompensated rise lowers relative intensity: response at the sixth treatment was 63% in patients without a threshold rise against 26% in those with one, and none of the 4 patients with a 125% rise responded without restimulation.
- **Prediction and action.** A model using ictal electroencephalogram components at the fifth treatment predicts threshold elevation with 82% accuracy, 88% sensitivity and 78% specificity. Escalate intensity across the course, or retitrate formally at the sixth treatment.

Fig 7.1 The dosing ladder to redraw from memory: threshold titrated at the first session, then bilateral treatment at half again above threshold and unilateral treatment at one and a quarter to one and a half times above it, with the threshold itself climbing across the course so that a fixed absolute charge falls progressively further below the therapeutic window.

TRAP Focusing on motor or electroencephalographic duration alone. A long, low-voltage seizure can mean inadequate relative dosing. The electrode distance in centimetres above the meatus-to-canthus line, and the cuff inflation pressure, are not carried by the sources, so **verify before the exam**.

HOLD THIS

Unilateral needs 125% to 150% above threshold, bilateral only 50%, and the threshold rises about 40% across the course. Fixed charge plus rising threshold equals treatment failure.

WHAT EARNS THE MARKS

- **Dose expressed relative to threshold**: an absolute charge without a threshold means nothing.
- **Restimulation offered in the same session**: a failed stimulus is corrected then, not next time.
- **Quality markers named**: amplitude, regularity, latency to slowing, postictal suppression.
- **The rising threshold built into the plan**: fixed charge plus rising threshold is treatment failure.

SEE ALSO 34 ECT: indications, workup and anaesthesia 36 ECT adverse effects, maintenance and the statute 37 Neuromodulation: rTMS, iTBS, tDCS, DBS, VNS, MST

36 · ECT adverse effects, maintenance and the statute

ECT AND NEUROMODULATION · TOPIC 36

The family asks whether ECT will damage his memory. What do you tell them, and what does the law require?

OSCE CONSENT STATION | LONG CASE, LEGAL ASPECTS

THE SHAPE OF THE ANSWER

1 **Separate the amnesias**	Postictal, anterograde, retrograde, and their courses.
2 **Answer the damage question**	No evidence of structural or cellular injury.

3 List the systemic effects	Headache, myalgia, haemodynamic shift, awareness.
4 Distinguish continuation from maintenance	Same episode against a new one.
5 Give the three legal provisions	Modified only, minors by board, not an emergency treatment.

Cognitive effects are the principal concern and the commonest question. Transient postictal disorientation resolves within minutes to hours. Anterograde amnesia reduces retention of new information during and just after the course and typically improves over subsequent weeks.

- **Retrograde amnesia.** It impairs recall of events weeks to months before treatment, and persistent deficits extending months to years are rare. A systematic review found no evidence that ECT causes structural or cellular brain damage.
- **Systemic effects.** Headache, nausea and myalgia from fasciculation, haemodynamic shift or temporalis stimulation, treated with ketorolac or paracetamol and ondansetron.
- **Cardiac and anaesthetic.** Transient vagal bradyarrhythmia followed by tachycardia and hypertension for 5 to 10 minutes, managed with pre-stimulus esmolol 40 to 80 mg or labetalol 10 to 50 mg; and rarely awareness during paralysis, needing adjustment of anaesthetic depth and reassurance.

Continuation and maintenance, separated by purpose. Continuation ECT is given in the months immediately after remission from an index course, to prevent relapse of the same episode; maintenance ECT continues beyond that, to prevent recurrence of new episodes.

- **Who gets them.** Severe, recurrent, treatment-resistant affective or psychotic illness that responded to index treatment but relapses despite adequate maintenance pharmacotherapy, with intervals tapered from weekly to fortnightly and then longer as stability allows.

TRAP The cutoff in months separating continuation from maintenance ECT, and the exact taper schedule in weeks, are not carried by the sourced material. Give the distinction by purpose, that is relapse of the same episode against recurrence of a new one, and **verify before the exam**.

THE STATUTE

Under the Mental Healthcare Act 2017 three provisions govern practice. Direct, that is unmodified, ECT is prohibited, so treatment is given with an intravenous anaesthetic and a muscle relaxant under an anaesthesiologist.

ECT in minors is restricted, requiring informed consent of the nominated representative together with prior approval of the Mental Health Review Board.

And ECT is excluded from the treatments that may be given as an emergency measure without statutory consent, so valid, free and informed written consent is obtained from a patient with capacity, or from the nominated representative in accordance with any advance directive. Unmodified ECT in India is not merely poor practice, it is unlawful.

TRAP Examiners ask for the numbered sections carrying the ban on unmodified ECT, the restriction in minors and the emergency exclusion, and for the age threshold defining a minor. None of that numbering is carried by the sourced chapters. State the provisions, say the numbers are read off the statute, and **verify before the exam**.

HOLD THIS

Retrograde amnesia for the weeks before, anterograde for the weeks during, and no structural damage. Then say the law: modified only, board approval for minors, and not an emergency treatment.

WHAT EARNS THE MARKS

- **Three memory effects distinguished by direction and course**: it is what the family is actually asking.
- **The structural damage question answered directly**: a systematic review found no such evidence.
- **Continuation and maintenance separated by purpose**: same episode against new episode.
- **The three statutory provisions named**: modified only, board approval for minors, and no emergency use.

SEE ALSO 34 ECT: indications, workup and anaesthesia 35 Placement, stimulus dosing and seizure adequacy 37 Neuromodulation: rTMS, iTBS, tDCS, DBS, VNS, MST

37 · Neuromodulation: rTMS, iTBS, tDCS, DBS, VNS, MST

ECT AND NEUROMODULATION · TOPIC 37

What is the difference between magnetic stimulation and direct current stimulation, and who would you offer each to?

NEUROMODULATION VIVA TABLE | LONG CASE, RESISTANT DEPRESSION

THE SHAPE OF THE ANSWER

1 Classify the field	Energy type against invasiveness, three groups.
2 Give the biophysics	Suprathreshold induction against subthreshold shift.
3 Target and protocol	Left dorsolateral prefrontal cortex, and how it is located.
4 Quote the evidence	Approval, theta burst non-inferiority, deep stimulation response.
5 Say what is available here	Outpatient magnetic stimulation, and the invasive exception.

Classify by energy and by invasiveness.

- **Magnetic and non-invasive.** Repetitive transcranial magnetic stimulation, intermittent theta burst stimulation and magnetic seizure therapy, all using Faraday induction, where time-varying magnetic pulses pass unattenuated through the skull and generate intracranial electric fields.
- **Electrical and non-invasive.** Transcranial direct current stimulation, delivering weak subthreshold constant current that shifts resting membrane potential without triggering action potentials.
- **Electrical and invasive.** Deep brain stimulation and vagus nerve stimulation, with implanted generators delivering pulses to subcortical nuclei, white matter tracts or cranial nerve afferents.
- **Why a cortical coil reaches deep circuits.** Superficial cortical stimulation propagates along white matter tracts to modify subcortical, striatal and limbic circuits.

MODALITY	TARGET AND PROTOCOL	EVIDENCE
Repetitive magnetic stimulation	**Left dorsolateral prefrontal cortex, Brodmann areas 9 and 46, localised by the 5 cm method, by the F3 position on the international system, or by neuronavigation; stimulation at the resting motor threshold, 10 Hz, 3 seconds on with a 21 second intertrain interval, 50 trains and 1,500 pulses over 20 minutes, 5 sessions weekly for 2 weeks in biomarker trials**	**Approved for refractory major depression in 2008; 72.73% were early improvers against 27.27% non-improvers on a 20% threshold**
Intermittent theta burst	The same target, delivering equivalent pulse counts in a shorter session, which enables accelerated protocols	**Multi-society consensus concluded it is non-inferior to standard stimulation for a major depressive episode**
Direct current stimulation	Scalp montage, anodal over the left and cathodal over the right prefrontal cortex for depression, or temporoparietal cathodal for auditory hallucination; anodal current depolarises and cathodal hyperpolarises subthreshold	Parameter data and effect sizes are not carried by the sources
Deep brain stimulation	Subgenual anterior cingulate cortex, nucleus accumbens and ventral striatum, ventral capsule, bed nucleus of the stria terminalis, or the superolateral medial forebrain bundle	**An average response rate of 60% in treatment-resistant depression, with tractography of intersecting white matter tracts now mattering more than grey matter coordinates**
Vagus nerve stimulation	Generator in the left infraclavicular chest wall with leads on the left cervical vagus; afferents reach the nucleus tractus solitarius and alter locus coeruleus and raphe firing	Adjunctive in chronic refractory depression; parameters and response rates are not carried by the sources
Magnetic seizure therapy	Induces a generalised seizure under anaesthesia and blockade, with focal intracranial current concentrated in superficial frontal cortex rather than the widespread spread of ECT	Comparable antidepressant effect with faster cognitive recovery and less retrograde amnesia; parameters not carried by the sources

Biomarkers, for the distinction answer. Baseline regional activity and local synchronisation at the stimulated target do not predict response.

- **Connectivity does predict it.** Improvement correlates negatively with baseline resting-state connectivity between the stimulated left prefrontal cortex and the left nucleus accumbens, and early improvers show stronger negative connectivity there, with higher accumbens connectivity to ventromedial prefrontal and subgenual cingulate cortex.

- **One caveat.** Prefrontal to subgenual connectivity alone does not separate improvers where anxiety is prominent.

TRAP The pre-treatment safety screen, the contraindication list and the adverse effects of magnetic stimulation, including seizure-risk figures, are not carried by the sourced material, because the consensus document is held only as a truncated record. Answer that screening is for implanted devices and seizure risk in principle, and **verify before the exam** against the full consensus and a device safety guideline.

INDIAN PRACTICE NOTE

Non-invasive, non-convulsive modalities do not induce seizures and are given without anaesthesia, so they fall outside the statutory restrictions that govern electroconvulsive therapy under the Mental Healthcare Act 2017, and magnetic stimulation is available in outpatient settings.

Invasive stimulation is psychiatric neurosurgery, requiring informed consent, institutional ethics approval and review by the Mental Health Review Board.

Availability: magnetic stimulation and theta burst are established at apex national neuropsychiatric centres, government medical colleges and major private tertiary units; direct current stimulation sits mostly in academic and research units; deep brain stimulation for psychiatric indications is investigational in a few advanced neurosurgical centres; and vagus nerve stimulation and magnetic seizure therapy are not in routine public service.

HOLD THIS

Magnetic stimulation is suprathreshold and triggers action potentials; direct current stimulation is subthreshold and only shifts the resting potential. That one distinction carries the biophysics question.

WHAT EARNS THE MARKS

- **Suprathreshold and subthreshold used precisely**: it is the biophysical distinction the question turns on.
- **The target localised three ways**: the 5 cm method, the F3 position, or neuronavigation.
- **Theta burst offered as non-inferior**: it is what makes accelerated protocols clinically relevant.
- **The legal line drawn at the seizure**: non-convulsive modalities sit outside the ECT provisions.

SEE ALSO 18 Resistant depression: staging, STAR*D, augmentation 34 ECT: indications, workup and anaesthesia 36 ECT adverse effects, maintenance and the statute

Emergencies and new drugs

38 · Rapid tranquillisation and acute agitation

EMERGENCIES · TOPIC 38

A young man is violent in casualty and refuses oral medication. What do you give, and what do you monitor?

EMERGENCY VIVA TABLE | OSCE STATION ON RESTRAINT AND CONSENT

THE SHAPE OF THE ANSWER

1 State the goal	Calming so examination is possible, not sleep.
2 De-escalate first	Medication is the restrictive last resort.
3 Choose by population	Adult, elderly or frail, adolescent, with the ceilings.
4 Monitor to a schedule	Every 15 minutes for an hour, then 30 for three.
5 Meet the statute	Emergency treatment, restraint duties, no seclusion.

The goal is calming, not remission. Rapid tranquillisation is emergency medication, by injection or orally when accepted, to calm or modify behaviour that poses immediate risk of harm, so that examination becomes possible.

- **The target endpoint.** Light sedation, and not sleep, unconsciousness or loss of the capacity to respond to speech, because the antipsychotic therapeutic effect takes 1 to 3 weeks and is not what is being attempted.
- **Where it sits.** A restrictive last resort after verbal de-escalation, psychological technique and environmental change have failed, and distinct from as-required prescribing, which treats early mild agitation to avert a crisis.

POPULATION	ORAL	INTRAMUSCULAR	LIMITS AND WARNINGS
Adults	Lorazepam 1 to 2 mg; haloperidol 2.5 to 5 mg with lorazepam 1 to 2 mg; risperidone 2 mg with lorazepam 2 mg	**Lorazepam 1 to 2 mg; haloperidol 2.5 to 5 mg with promethazine 50 mg; aripiprazole 9.75 mg**	**Repeat oral after 45 minutes; intramuscular lorazepam or haloperidol with promethazine after 30 minutes; intramuscular aripiprazole after 2 hours; maximum lorazepam 6 mg orally and 10 mg intramuscularly in 24 hours, haloperidol 20 mg, promethazine 100 mg, risperidone 4 mg, aripiprazole 3 injections and 30 mg. Add procyclidine 5 mg in a naive patient on haloperidol**
Elderly or frail	Lorazepam 0.5 to 1 mg; haloperidol 0.5 to 1 mg	Promethazine 25 mg; aripiprazole 2.5 mg initially	**Avoid haloperidol in Lewy body dementia and Parkinson disease; promethazine adds anticholinergic burden and may worsen delirium**

POPULATION	ORAL	INTRAMUSCULAR	LIMITS AND WARNINGS
Adolescents	Lorazepam 0.5 to 1 mg; haloperidol 1 to 2.5 mg	Haloperidol 1 to 2.5 mg	**Repeat after 60 minutes; maximum lorazepam 4 mg, haloperidol 10 mg orally and 5 mg intramuscularly; high extrapyramidal sensitivity, so consider procyclidine 2.5 mg, and watch for paradoxical disinhibition**

Intramuscular diazepam is avoided for painful, erratic absorption, and intramuscular chlorpromazine for hypotension, collapse and tissue necrosis. Zuclopenthixol acetate 50 to 150 mg is reserved for patients with a known history of repeated short-acting injections with transient response, and is contraindicated for initial tranquillisation, in naive patients and in anyone actively struggling.

Giving a benzodiazepine to a patient on clozapine carries a high risk of fatal respiratory depression and cardiovascular collapse. In traumatic brain injury, oral promethazine or quetiapine, or intramuscular aripiprazole or promethazine, are preferred.

Monitoring is the answer that separates candidates. This is a medical emergency needing continuous visual observation by staff trained in immediate life support, with a doctor available within 30 minutes.

- **The schedule.** Chart observations every 15 minutes for the first 1 hour, then every 30 minutes for the next 3 hours, completing a 4-hour window: respiratory rate, level of consciousness on the alert, voice, pain and unresponsive scale, pulse, blood pressure, oximetry and temperature, and at minimum respiratory rate with that scale if resistance prevents the rest.
- **Summon the physician immediately if.** Systolic pressure falls below 80 mmHg or the radial pulse becomes impalpable; the pulse falls below 60 beats per minute or becomes irregular; the temperature is elevated, in which case antipsychotics are withheld for neuroleptic malignant syndrome risk; or respiratory depression, stridor or airway compromise develops.
- **Two further duties.** Obtain a twelve-lead tracing at the earliest opportunity where a prolonging antipsychotic was given and the baseline was abnormal or no tracing exists from the preceding 3 months. Avoid seclusion afterwards because of unobserved airway compromise, and if it is unavoidable keep continuous within-sight monitoring.

Cocktail against chaser. Two service-level approaches, and the examiner may ask which you use.

- **The cocktail approach.** Used by approximately 55 percent of emergency service directors, it gives a high-potency first-generation antipsychotic with a benzodiazepine in separate syringes: haloperidol 5 to 10 mg with lorazepam 1 to 2 mg in healthy adults, or 1 to 2 mg with 0.5 to 1 mg in the elderly, repeatable in 30 to 60 minutes, achieving synergistic sedation within 30 minutes at the cost of dystonia and akathisia needing benztropine 1 mg or procyclidine 5 mg.
- **The chaser approach.** Used by approximately 15 percent, it calms first with lorazepam alone, 1 to 2 mg repeated every 30 to 60 minutes to a ceiling of 4 to 6 mg over 1.5 hours, then educates the patient and invites them to choose an oral atypical agent, which builds alliance and adherence at the cost of nursing time and reassessment.

THE STATUTE

Emergency treatment without informed consent is permitted under the Mental Healthcare Act 2017 for a limited statutory period, to prevent imminent death, severe irreversible harm or injury to self or others.

Physical restraint is restricted to a temporary last resort to prevent imminent harm, requires authorisation by a psychiatrist with continuous monitoring and detailed documentation, and cannot be punitive or a substitute for staffing.

The Act also prohibits seclusion, and carries two downstream duties examiners probe: notifying the nominated representative after each instance of restraint, and periodic reporting of restraint episodes to the Mental Health Review Board. The numbered provisions, the maximum emergency period and the prescribed reporting periods are not carried by the sourced material and must be read off the Act.

HOLD THIS

Calming, not sleep; then fifteen minutes for an hour and thirty minutes for three more. The monitoring schedule is what shows you have done this rather than read about it.

WHAT EARNS THE MARKS

- **Light sedation named as the endpoint**: aiming for sleep is the commonest wrong answer here.
- **The 15 and 30 minute observation schedule**: it is the part that shows ward experience.
- **A benzodiazepine refused in a clozapine patient**: the combination risks fatal respiratory depression.
- **Restraint documented, notified and reported**: three statutory duties, not one.

SEE ALSO 03 Extrapyramidal syndromes and tardive dyskinesia 19 Benzodiazepines: kinetics and equivalence 39 Tricyclic overdose and sodium bicarbonate

39 · Tricyclic overdose and sodium bicarbonate

EMERGENCIES · TOPIC 39

She has taken forty amitriptyline tablets and the QRS is 120 milliseconds. What do you do?

EMERGENCY VIVA TABLE | SHORT CASE, SELF-POISONING

THE SHAPE OF THE ANSWER

1	**Four receptor actions**	Sodium channel, alpha-1, muscarinic, potassium channel.
2	**Grade by dose and clock**	Bands in mg/kg, and the high-risk window.
3	**Read the tracing**	100 ms predicts seizure, 160 ms predicts arrhythmia.
4	**Give bicarbonate properly**	Boluses to three endpoints, before intubation.
5	**Name what is forbidden**	Physostigmine, flumazenil, class 1 antidysrhythmics.

Four mechanisms, one antidote. Tricyclics block fast cardiac sodium channels, alpha-1 adrenoceptors, peripheral and central muscarinic receptors, and potassium channels.

- **Dose bands.** Under 5 mg/kg gives minimal symptoms; 5 to 10 mg/kg gives drowsiness, sinus tachycardia and mild anticholinergic signs; above 10 mg/kg is potentially life-threatening with coma,

hypotension, seizures and ventricular dysrhythmia, the anticholinergic features often masked by coma; and above 30 mg/kg gives pH-dependent cardiotoxicity with coma beyond 24 hours.

- **The clock.** Severe toxicity usually declares within 2 hours, the highest risk of seizure and arrhythmia falls in the first 6 to 8 hours, continuous cardiac monitoring runs for a minimum of 6 hours, and toxicity lasts 24 to 48 hours, or 4 to 5 days with modified-release preparations.

ELECTROCARDIOGRAPHIC FEATURE	MEANING
QRS above 100 ms	**Strongly predictive of seizures, and the threshold approximately 88 percent of poison centre directors use to start bicarbonate**
QRS above 160 ms	**Highly predictive of ventricular tachycardia and fibrillation**
Lead aVR	Prominent terminal R wave with an increased R to S ratio above 0.7
QTc	Prolonged, from potassium channel blockade and QRS widening

The protocol, airway first. Below a Glasgow score of 12, intubate, and give sodium bicarbonate 1 to 2 mmol/kg intravenously before intubation, because the transient hypoventilation and acidosis of laryngoscopy acutely worsen sodium channel blockade and are a recognised cause of peri-intubation arrest.

- **How bicarbonate works, and its endpoints.** Sodium loading overcomes competitive blockade and alkalinisation unbinds the drug from neutral channels. Give rapid boluses of 1 to 2 mmol/kg every 1 to 2 minutes until the QRS narrows, to three endpoints: QRS below 140 ms, arterial pH between 7.5 and 7.55, and serum sodium not exceeding 155 mmol/L, with gases every 30 minutes and specialist advice beyond a cumulative 6 mmol/kg.
- **Hypotension.** A 20 ml/kg crystalloid bolus and bicarbonate 2 mmol/kg, then noradrenaline or adrenaline at 0.05 to 0.5 mcg/kg/min through a dedicated line.
- **Seizures.** Intravenous lorazepam 0.1 mg/kg to a maximum of 4 mg, diazepam 0.15 mg/kg to 10 mg or midazolam 0.2 mg/kg to 10 mg, with concurrent bicarbonate because the lactic acidosis of a seizure worsens cardiotoxicity.
- **Decontamination.** Activated charcoal is only for ingestion above 10 mg/kg presenting within 2 hours, and only after the airway is secured with a cuffed tube.

What is contraindicated. Physostigmine is absolutely contraindicated: it precipitates complete atrioventricular block, severe hypotension and asystole. Flumazenil is absolutely contraindicated: it precipitates refractory fatal status epilepticus.

- **Also to be avoided.** Class 1A and 1C antidysrhythmics aggravate the blockade; amiodarone and beta blockers worsen conduction failure and myocardial depression; ipecac risks aspiration; and cardioversion is unlikely to succeed until the biochemistry is corrected.
- **Salvage options.** Lidocaine 1 to 1.5 mg/kg by slow push then 20 to 50 mcg/kg/min once pH exceeds 7.5, lipid emulsion at 1.5 ml/kg over 1 minute as an intravascular sink for the lipophilic drug, hypertonic saline to reach the sodium target where alkalaemia blocks more bicarbonate, and veno-arterial extracorporeal support in refractory cardiogenic shock.
- **What does not help.** Haemodialysis and haemoperfusion are ineffective, because the drug is lipid soluble, widely distributed and highly protein bound.

RULE **Boluses, not an infusion, and bicarbonate before the laryngoscope.** A slow infusion without preceding boluses does not create the transient sodium peak and alkalinisation that unbind the drug from the channel.

HOLD THIS

QRS above 100 ms predicts seizures, above 160 ms predicts arrhythmia, and the titration target is below 140 ms with a pH of 7.5 to 7.55. Three numbers carry this whole station.

WHAT EARNS THE MARKS

- **Bicarbonate given before the laryngoscope**: peri-intubation arrest is the avoidable death here.
- **Boluses rather than an infusion**: the transient sodium peak is the point of the treatment.
- **Three titration endpoints quoted**: QRS, pH and serum sodium together.
- **Physostigmine and flumazenil refused by name**: both are lethal in this poisoning.

SEE ALSO 13 Classes, mechanisms and the latency question 40 Lithium, benzodiazepine and paracetamol overdose 38 Rapid tranquillisation and acute agitation

40 · Lithium, benzodiazepine and paracetamol overdose

EMERGENCIES · TOPIC 40

An obtunded patient has taken an unknown overdose. What do you send, and what do you refuse to give?

EMERGENCY VIVA TABLE | SHORT CASE, UNKNOWN INGESTION

THE SHAPE OF THE ANSWER

1	**Three different logics**	Clearance, support, and a time-critical antidote.
2	**Lithium: restore clearance**	Saline, then dialysis on severity, with rebound.
3	**Benzodiazepine: support**	Airway and oxygen, and flumazenil withheld.
4	**Paracetamol: test everyone**	The commonest missed co-ingestant, nomogram-guided.
5	**Separate the toxidromes**	Anticholinergic against the malignant syndrome.

Three overdoses, three different logics: clearance, support, and a time-critical antidote.

- **Lithium.** Toxicity depends on chronicity and renal function, with coarse tremor, vomiting, diarrhoea, ataxia, dysarthria, hyperreflexia, myoclonus, seizures and encephalopathy; the intoxication ladder and management sequence are given in full in the lithium topic of this note.
- **Clearing the ion.** Volume expansion with isotonic saline restores filtration and promotes excretion; charcoal does not bind the ion; whole bowel irrigation may be considered for a large sustained-release ingestion presenting early; and haemodialysis is the definitive elimination modality, with post-dialysis rebound from slow intracellular redistribution meaning repeat sessions may be needed.

TRAP The serum concentrations that define the dialysis threshold in acute against chronic lithium toxicity are not carried by the sourced material, which frames the indication by severity. Answer on severity and on renal function, and **verify before the exam** against a toxicology reference.

Benzodiazepine. Isolated oral ingestion usually produces mild central depression with somnolence, ataxia and slurred speech and stable vital signs; co-ingestion with alcohol, an opioid

or clozapine precipitates respiratory depression, hypotension and fatal coma.

- **The cornerstone is supportive.** Airway care, supplemental oxygen and mechanical ventilation.
- **Flumazenil, and when not.** A competitive antagonist at the benzodiazepine site, it is not given routinely in an undifferentiated overdose, and is strictly contraindicated with suspected tricyclic co-ingestion, a known seizure disorder or physiological dependence, because it reliably precipitates intractable status epilepticus and autonomic collapse.
- **Where it is used for reversal.** The sourced regimen is 200 mcg intravenously over 15 seconds, then 100 mcg over 15 seconds every 60 seconds as required, to a maximum of 1 mg in 24 hours.

Paracetamol. Quantitative serum testing is mandatory in every intentional self-poisoning, because the early stages are asymptomatic or give only non-specific nausea, and this is the single most commonly missed co-ingestant in psychiatric overdose.

- **The mechanism.** Hepatic CYP2E1 converts the drug to a toxic electrophile, and once glutathione stores are depleted that metabolite binds hepatocytes and causes centrilobular necrosis.
- **The antidote.** N-acetylcysteine replenishes glutathione, acts as an alternative substrate and improves microcirculatory flow, and treatment is governed by a plotted nomogram against the post-ingestion level, or started in any delayed presentation with raised hepatic enzymes.

TRAP The three-bag acetylcysteine regimen, its total dose and infusion times, and the nomogram treatment line at four hours, are not carried by the sourced material. Say that treatment is nomogram-guided with acetylcysteine and that testing is mandatory in self-poisoning, and **verify before the exam** against your unit's poisoning protocol.

COMMON TRAP

Giving flumazenil as a diagnostic reversal trial in an obtunded patient of unknown ingestion. If a tricyclic was co-ingested, or the patient is benzodiazepine-dependent, removing GABA-mediated seizure suppression precipitates fatal status epilepticus.

Separately, the anticholinergic toxidrome and neuroleptic malignant syndrome both give hyperthermia and tachycardia: anticholinergic toxicity gives warm dry skin, absent bowel sounds, dilated unreactive pupils and retention, while the syndrome gives lead-pipe rigidity, profuse diaphoresis and a raised creatine kinase.

HOLD THIS

Send a paracetamol level on every intentional overdose, and do not reach for flumazenil. One test is free and decisive; one drug is neither.

WHAT EARNS THE MARKS

- **A paracetamol level sent unprompted**: it is the missed co-ingestant that changes the outcome.
- **Charcoal refused for lithium**: it does not bind the ion, and saying so shows the pharmacology is understood.
- **Post-dialysis rebound anticipated**: the level is rechecked because the ion leaves tissue slowly.
- **Two toxidromes separated at the bedside**: dry skin and silent abdomen against rigidity and sweating.

SEE ALSO 09 Lithium: organ toxicity and intoxication 20 Withdrawal, the diazepam taper and flumazenil 39 Tricyclic overdose and sodium bicarbonate

41 · New and pipeline drugs

NEW DRUGS · TOPIC 41

What is genuinely new in psychopharmacology, and can any of it be prescribed in India?

VIVA TABLE ON RECENT ADVANCES | LONG CASE, REFRACTORY ILLNESS

THE SHAPE OF THE ANSWER

1 **Lead with the new mechanism**	Muscarinic agonism with a peripheral shield.
2 **Name the neurosteroids**	Infusion against oral, with their boxed warnings.
3 **Place the newer partial agonists**	Cariprazine, brexpiprazole, lumateperone.
4 **Tardive dyskinesia agents**	Transporter inhibition, and what they worsen.
5 **Answer on availability**	What is manufactured here, and what is unlawful.

DRUG	MECHANISM	EVIDENCE AND DOSING	THE DISCRIMINATOR
Xanomeline with trospium	**Central M1 and M4 muscarinic agonism, paired with a quaternary peripheral antagonist that does not cross the blood-brain barrier**	Approved for schizophrenia in adults in September 2024; target 100 mg/20 mg twice daily, maximum 125 mg/30 mg, restricted to 100 mg/20 mg in older adults or with a strong CYP2D6 inhibitor; taken on an empty stomach, at least 1 hour before or 2 hours after food	**It does not bind dopamine D2 receptors at all, so there is no extrapyramidal or prolactin liability; peak symptom reduction in the pivotal trials was -20.6 against -12.2 for placebo, a difference of -8.4, and 75% maintained a 30% reduction at 52 weeks**
Brexanolone	Synthetic allopregnanolone, a positive allosteric modulator at synaptic and extrasynaptic GABA A receptors	A single continuous 60-hour intravenous infusion in hospital, maintained at 60 or 90 ug/kg/hour between an upward and downward taper	**Boxed warning for excessive sedation and sudden loss of consciousness, needing continuous oximetry with sedation checks every 2 hours under a restricted programme**
Zuranolone	The orally bioavailable analogue of the same class	50 mg once daily in the evening for 14 consecutive days, with a fat-containing meal of at least 400 calories	**Boxed warning for driving impairment, with no driving for at least 12 hours after each dose, and contraception during treatment and for 1 week after; neither agent is approved for non-postpartum depression**

DRUG	MECHANISM	EVIDENCE AND DOSING	THE DISCRIMINATOR
Cariprazine	D3-preferring D3 and D2 partial agonist with 5-HT1A partial agonism	1.5 mg daily in schizophrenia, titrated in 1.5 mg steps to a maximum of 6.0 mg	**Approximately 10-fold higher affinity for D3 than D2, and an active metabolite with a half-life of 1 to 3 weeks, so akathisia can appear weeks after a dose change**
Brexpiprazole	Serotonin-dopamine activity modulator with higher 5-HT1A than D2 affinity	**The starting dose, intermediate step, target range and ceiling are not carried by the sources**	Lower intrinsic D2 partial agonist activity than aripiprazole, hence significantly less treatment-emergent akathisia
Lumateperone	Potent 5-HT2A antagonism with postsynaptic D2 antagonism, presynaptic partial agonism and serotonin transporter inhibition	42 mg once daily with food	**Near-placebo rates of weight, glucose and lipid change, minimal motor liability and no meaningful prolactin or QTc effect**
Valbenazine and deutetrabenazine	Reversible vesicular monoamine transporter 2 inhibition, depleting presynaptic dopamine available for release	Once daily for the prodrug; twice daily with food for the deuterated agent, whose carbon-deuterium bonds slow metabolism	**For tardive dyskinesia and not for acute extrapyramidal symptoms, which they worsen; they relieve dyskinesia without postsynaptic blockade, where anticholinergics do the reverse**

The pipeline, one line each.

- **Psilocybin.** A prodrug for psilocin, an agonist at 5-HT2A and 5-HT1A receptors that releases cortical glutamate, promotes plasticity and destabilises rigid default-mode connectivity, and a deuterated analogue is in phase 3 trials at fixed doses of 8 mg and 16 mg alongside baseline antidepressants.
- **MDMA.** It reverses monoamine transporters with oxytocin release, dampening amygdala reactivity while strengthening prefrontal control to open a window for trauma memory reconsolidation, and remains under regulatory deliberation over blinding, psychological safety and cardiovascular tolerability.
- **Navacaprant.** A selective kappa-opioid antagonist, targeting the stress-activated dynorphin system that suppresses ventral striatal dopamine and produces anhedonia.
- **Glucagon-like peptide-1 agonists.** They matter in psychiatry for three reasons: reversing antipsychotic-induced metabolic syndrome, reducing cue-induced craving through accumbens and ventral tegmental receptors, and a neuroprotective signal under investigation.

INDIAN REALITY

Cariprazine and brexpiprazole are reported as approved and domestically manufactured at moderate cost, and deutetrabenazine as marketed by Indian manufacturers, with valbenazine accessible through specialty suppliers or import authorisation.

Xanomeline with trospium, zuranolone and brexanolone are not recorded as holding broad domestic manufacturing licences, so access is by individual compassionate import at high personal cost. Psilocybin and MDMA are controlled substances under the narcotics statute, and possession, prescription or administration outside an approved institutional trial is unlawful.

Practically, a 60-hour infusion with continuous oximetry and telemetry is not a routine Indian option, so postpartum depression is managed with structured psychotherapy, maternal support and conventional reuptake inhibitors such as sertraline or escitalopram, which cost almost nothing and are compatible with breastfeeding.

Every availability statement in this box is unverified in the sources and is to be re-checked against the current regulatory database.

HOLD THIS

The first genuinely new mechanism is muscarinic, not dopaminergic. Central M1 and M4 agonism with a peripheral shield, no D2 binding, and therefore no extrapyramidal or prolactin cost.

WHAT EARNS THE MARKS

- **Xanomeline named as non-dopaminergic**: no D2 binding is the reason there is no movement or prolactin cost.
- **The boxed warnings quoted with the neurosteroids**: sedation monitoring for one, driving restriction for the other.
- **Transporter inhibitors placed correctly**: for tardive dyskinesia, and they worsen acute extrapyramidal symptoms.
- **Availability answered honestly**: what is made here, what needs import, and what is unlawful outside a trial.

SEE ALSO 03 Extrapyramidal syndromes and tardive dyskinesia 18 Resistant depression: staging, STAR*D, augmentation 33 Essential psychotropics in India

Quick review

The discriminators in one table	
ANTIPSYCHOTIC OCCUPANCY	Response near 65% striatal D2 occupancy, extrapyramidal risk from 78% to 80%. Mesolimbic benefit, cortical cognition cost, nigrostriatal movement cost, tuberoinfundibular prolactin cost.
DOSE ARITHMETIC	Percentage of the licensed maximum, summed across drugs including as-required doses. Chlorpromazine equivalence is retired; potency is not efficacy.
MOVEMENT SYNDROMES	Dystonia hours, parkinsonism days, akathisia months, tardive dyskinesia years. Anticholinergics help the first two, not akathisia, and worsen the last.
CLOZAPINE	Start 12.5 mg, target 175 to 300 mg in Indian ancestry, trough 350 to 600 ng/mL. Myocarditis in weeks two to three: protein first, troponin after, stop at protein above 100 mg/L.
INJECTABLES	Test dose for oil-based depots, three weeks oral cover for risperidone microspheres, deltoid loading for paliperidone, three hours of observation for olanzapine pamoate.
LITHIUM	Twelve-hour trough in a red-top tube; 0.8 to 1.2 for mania, 0.8 to 1.0 maintenance, 0.4 to 0.8 in the elderly. Sodium depletion raises the level. Taper, do not stop.
VALPROATE AND LAMOTRIGINE	Valproate: about one in ten malformations, three to four in ten neurodevelopmental disorders, folic acid does not fix it. Lamotrigine: halve with valproate, double with inducers, rash in weeks two to eight.
ANTIDEPRESSANT SWITCHING	Two weeks off an irreversible inhibitor for enzyme resynthesis, five weeks off fluoxetine for norfluoxetine. Reversible inhibition needs no cheese diet.
TWO TOXIDROMES	Serotonin toxicity: clonus and hyperreflexia within hours, hyperactive gut. Neuroleptic malignant syndrome: lead-pipe rigidity and hyporeflexia over days, raised creatine kinase.
RESISTANCE AND ECT	Two adequate failed trials define resistance; exclude pseudo-resistance first. ECT before a second antipsychotic in clozapine resistance. Unilateral needs 125% to 150% above threshold, bilateral 50%, and threshold climbs about 40%.
INTERACTIONS	Smoke induces CYP1A2, nicotine does not: cut clozapine by up to 50% and olanzapine by about 30% on cessation. Grapefruit inhibits gut CYP3A4. Protein displacement rarely matters alone.
EMERGENCIES	Tranquillisation aims at calming, with observations every 15 minutes for an hour. Tricyclic overdose: QRS above 100 ms, bicarbonate boluses to a QRS below 140 ms and pH 7.5 to 7.55. Send a paracetamol level every time.

HOLD THIS, LAST

Every one of these tables is a mechanism with a number attached. Give the mechanism first, then the number, then the monitoring interval, and say plainly when a figure has to be checked against a source rather than guessed.

PART VIII

VIII

Viva Tables III: Assessments, Scales & Specialty Psychiatry

Psychometrics and every rating scale by domain, then the law as the examiner asks it, community psychiatry and the sub-specialties.

01 Psychometrics and tests

02 Rating scales by domain

03 Forensic psychiatry and the law

04 Community psychiatry

05 Child, women, geriatric, addiction, liaison, psychotherapy, culture, sex and sleep

Viva Tables III: Assessments, Scales & Specialty Psychiatry

Assessments, scales and the specialty tables

The assessment table is the most predictable station in the practical.

An apparatus, a rating form or a single scale name is put in front of you, and the questions come in one order: what is this, what does it measure, how is it administered and scored, and what does the number mean. **Answer in that order and stop.**

Psychometrics comes first here because every scale question is a psychometrics question wearing a scale's name.

- **Concept.** A cut-off trades sensitivity against specificity.
- **Concept.** A positive result is worth only what the base rate allows.
- **Key idea.** A score is severity, not diagnosis.

Where the library could not source a value, this note prints the question instead of the number: a confidently wrong cut-off costs more than an honest gap.

Psychometrics and tests

01 · Reliability and validity: what a scale may claim

PSYCHOMETRICS · TOPIC 01

This scale calls itself reliable and valid. What would you need to see?

VIVA TABLE: PSYCHOMETRICS, AND BEHIND EVERY SCALE SPOTTER

THE SHAPE OF THE ANSWER

1 **Define the pair**	Reliability is consistency, validity is accuracy about the construct
2 **Four reliabilities**	Test-retest, alternate forms, internal consistency, inter-rater, each with its index
3 **The thresholds**	Alpha bands by purpose, and r of 0.80 for stability
4 **Validity evidence**	Five categories of evidence under one construct, content judged by panel
5 **The ceiling**	Reliability caps validity at its square root

Reliability is consistency; validity is accuracy. An observed score is a true score plus random error, and reliability is the share of observed variance that is true variance.

- **Concept.** An unreliable scale cannot be valid, because erratic measurement cannot track a stable construct.
- **Trap.** A reliable scale may measure nothing of interest: finger length is a stable measure of finger length and says nothing about self-esteem.

RELIABILITY TYPE	ERROR SOURCE	METHOD	INDEX AND THRESHOLD
Test-retest	Temporal instability	Same test, same cohort, 1 week or 2 to 4 weeks apart	**Pearson r of 0.80 or above; state measures may drift, trait measures should not**
Alternate forms	Content sampling	Two equivalent versions, same group	**Pearson r of 0.80 or above**
Internal consistency	Item heterogeneity	One administration: split-half, Kuder-Richardson 20, Cronbach alpha	**Alpha 0.70 exploratory, 0.80 good, 0.90 for a decision about an individual**
Inter-rater	Observer subjectivity	Independent raters, same interview	**Cohen kappa for categories, intraclass correlation for continuous scores**

RULE **Reliability is necessary but not sufficient for validity, and caps it at its square root.** A reliability of 0.81 permits a maximum validity coefficient of 0.90.

Reliability becomes clinical through the standard error of measurement, the standard deviation multiplied by the square root of one minus reliability. A 95 percent confidence interval for a true score is the observed score plus or minus 1.96 standard errors, so poorer reliability widens the band around the number you are about to act on.

VALIDITY, IN THE MODERN FRAME

One construct, five categories of evidence: test content, response processes, internal structure, relations to other variables (concurrent, predictive, convergent, discriminant), and the consequences of testing. Content validity is judged by an expert panel of 3 or more with item agreement of 80 percent or above. Face validity is informal only: instruments of more than 567 statements work despite items that look irrelevant.

TRAP Offering a high alpha as proof the scale measures the disorder. Redundant paraphrased items inflate alpha while narrowing content, and the source treats an alpha of 0.95 as a signal of redundancy. Raw percentage agreement between raters is equally misleading, since it does not correct for chance.

VERIFY BEFORE THE EXAM The numeric interpretation bands for Cohen kappa are not in the library sources, so none are printed. Learn them from a standard text; kappa falls paradoxically low when base rates are extreme.

HOLD THIS

Reliability caps validity at its square root, and convergence with a similar scale is not criterion validity. Criterion validity needs an independent standard or a future outcome.

WHAT EARNS THE MARKS

- **Naming the index with the method**: kappa for categories, intraclass correlation for continuous scores
- **Quoting the alpha bands by purpose**: 0.70 exploratory, 0.80 good, 0.90 for a decision about one person
- **Saying reliability is necessary and not sufficient**: erratic measurement cannot track a stable construct
- **Separating convergence from criterion validity**: criterion needs an independent standard or a future outcome

SEE ALSO 02 Accuracy and cut-offs 04 Screening and interviews 09 Spotters

02 · Accuracy: sensitivity, specificity, predictive values and the cut-off

PSYCHOMETRICS · TOPIC 02

This screen cuts off at 8. What happens to that cut-off in a community camp?

VIVA TABLE: PSYCHOMETRICS; THE FOLLOW-UP TO ANY CUT-OFF QUESTION

THE SHAPE OF THE ANSWER

1 The instrument pair	Sensitivity rules out on a negative, specificity rules in on a positive
2 The population pair	Predictive values move with prevalence, test properties do not
3 Work one example	One arithmetic run at a stated prevalence, then the false-positive share
4 Likelihood ratios	They carry the test between populations without recalculation
5 Pick the direction	Screening tolerates false positives, confirmatory testing does not

- **Sensitivity.** The proportion of true cases called positive, so a negative result on a sensitive test rules out: the SNOUT rule.
- **Specificity.** The proportion of non-cases called negative, so a positive result on a specific test rules in: the SPIN rule.
- **Key idea. Both belong to the instrument** and do not move with prevalence.

Predictive values do move, which is the real question when the stem shifts from a tertiary clinic to a community camp.

PREVALENCE	POSITIVE PREDICTIVE VALUE	NEGATIVE PREDICTIVE VALUE	AT THE TABLE
1 percent	8 percent	Above 99 percent	**A community positive is usually false**
10 percent	50 percent	99 percent	**A coin toss on the positive side**
20 percent	69 percent	97 percent	**Usable in a selected clinic**
50 percent	90 percent	90 percent	**The balanced case textbooks assume**

Work one example and it stays. In 1 million people at 15 percent prevalence, a test with 95 percent sensitivity and 95 percent specificity gives 142,500 true and 42,500 false positives: accuracy is 95 percent, yet 23 percent of positives are false and the positive predictive value is 77 percent.

Where disorder prevalence is 1 percent to 2 percent, it falls below 10 percent. Likelihood ratios escape this: the positive ratio is sensitivity divided by one minus specificity, and above 10 it rules in; the negative ratio is one minus sensitivity divided by specificity, and below 1.0 it lowers probability.

RULE **Lowering a cut-off buys sensitivity with specificity and raising it does the reverse.** Pick the direction from clinical cost: screening tolerates false positives, confirmatory diagnosis and high-risk treatment do not.

Fig 1 Plate to draw: the two-by-two table with true and false positives and negatives, sensitivity and specificity read down the disease columns, predictive values read across the test rows, and an arrow marking that only the row quantities move when prevalence changes.

INDIAN PRACTICE

Standardisation means fixed administration, objective scoring and reference norms. Western norms cannot be lifted here uncritically: language, schooling, literacy and the cultural shaping of symptoms degrade construct and criterion validity, so a scale needs translation, back-translation, validation and stratification against local norms. Say this before quoting any imported cut-off.

TRAP Saying sensitivity and specificity fall when you move from a tertiary centre to a primary health centre. They do not; the predictive values move. This is the most reliable discriminator in the station.

VERIFY BEFORE THE EXAM The area-under-curve anchor values for an ideal and a chance test, and the Youden rule for an optimal cut-off, are not in the library sources, so none are printed. Describe the curve as sensitivity against one minus specificity across every cut-off, and read the anchors from a standard text.

HOLD THIS

Sensitivity and specificity belong to the test, predictive values to the population, and likelihood ratios travel between both.

WHAT EARNS THE MARKS

- **Holding sensitivity and specificity still**: only the predictive values move when the setting changes
- **Working one arithmetic example**: it shows you can derive the predictive value rather than recall it
- **Naming the standardisation caveat**: translation, back-translation, validation and local norms before an imported cut-off
- **Choosing the cut-off from clinical cost**: the direction of the trade is the examinable judgment

SEE ALSO 01 Reliability and validity 03 Risk scales 13 Substance use scales

Choose the denominator.

Read down for sensitivity and specificity. Read across for predictive values.

	REFERENCE STANDARD	
	Condition present	**Condition absent**
TEST **Positive**	a True positive	b False positive
Negative	c False negative	d True negative

Sensitivity

a / (a + c)

Of those with the condition

Specificity

d / (b + d)

Of those without the condition

Positive predictive value

a / (a + b)

Of those who test positive

Negative predictive value

d / (c + d)

Of those who test negative

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. The two-by-two table with sensitivity, specificity and predictive-value denominators. Predictive values condition on the test result; sensitivity and specificity condition on reference status. Read the reference-standard columns for sensitivity and specificity, then the test-result rows for predictive values. The letters stand for counts; no numerical study results are implied.

Read down the reference-standard columns, then across the test-result rows.

Schematic prepared by Luna for Weave Viva, based on chapter P5D-01. Original edition artwork; publication licence to follow the edition.

03 · Risk scales and the base-rate ceiling

PSYCHOMETRICS · TOPIC 03

Which risk scale will you use to decide whether this patient can go home?

LONG CASE SAFETY QUESTIONS, AND THE VIVA TABLE ON ASSESSMENT

THE SHAPE OF THE ANSWER

1 **Refuse the premise**	No scale predicts individual suicide, and say why
2 **Three generations**	Clinical intuition, then psychometric scales, then statistical models
3 **The arithmetic**	A rare outcome caps the positive predictive value of any test
4 **What the instruments do hold**	Structured enquiry: the ideation ladder and the behaviour subscale
5 **The clinical answer**	Needs assessment, means restriction, structured intervention, brief contact follow-up

Prediction of suicide has passed through three generations. None of them delivered usable individual prediction, and the examiner is testing whether you know why.

- **First.** Unaided clinical intuition.
- **Second.** Psychometric scales and biological tests.
- **Third.** Scales from multivariable statistical models.

POOLED GROUP	EVALUATIONS	POSITIVE PREDICTIVE VALUE	THE READING
All scales, fatal suicide	51	5.5 percent (95 percent CI 3.9 to 7.9)	**About 19 of 20 high-risk labels are wrong**
All scales, non-fatal self-harm	62	26.3 percent (95 percent CI 21.8 to 31.3)	**Better only because the outcome is commoner**
Evaluations at low risk of bias	16	16.1 percent (95 percent CI 11.3 to 22.3)	**Equals the 16.3 percent background repetition rate, so it adds nothing**
Psychological scales, fatal suicide	35	3.7 percent	**Worse than the all-scale figure**
Third-generation statistical scales	19	38.7 percent for the composite outcome	**Modelling did not break the ceiling**
Biological tests, fatal suicide	16	14.5 percent, and 21.1 percent for cerebrospinal 5-HIAA across 6	**A biological marker is no exception**

Table: pooled values from the 2017 systematic review of 70 studies and 128 scale evaluations. By setting, 32.5 percent in general hospital self-harm presentations and 26.8 percent in psychiatric inpatients.

The reason is arithmetic. Pokorny showed in 1983 that in an inpatient cohort with a suicide rate of 500 per 100,000 per year, a prevalence of 0.5 percent, an idealised test with 99 percent

sensitivity and 99 percent specificity gives a positive predictive value of 33 percent, so 66 percent of high-risk labels are false.

With realistic figures, 50 percent sensitivity and 90 percent specificity, it collapses to 2 percent. The clinical utility index, sensitivity multiplied by positive predictive value, grades excellent at 0.81 or above and poor below 0.49: all scales reach 0.24, psychological scales 0.27, third-generation scales 0.33.

WHAT THE INSTRUMENTS DO CONTAIN

The Columbia suicide severity rating scale is worth describing as structured enquiry, not prediction. Its ideation ladder climbs through wish to be dead, non-specific active thoughts, ideation with method but no plan or intent, ideation with some intent and no plan, and ideation with specific plan and intent, then rates frequency, duration, controllability, deterrents and reasons.

Its behaviour subscale separates actual, interrupted and aborted attempts from preparatory acts. That separation is the examinable content.

RULE **Risk instruments are aides-memoire for systematic enquiry, not actuarial tests.** The relevant national guideline directs clinicians not to use scales to predict suicide or repetition, and substitutes needs assessment: treat the modifiable, restrict access to lethal means, offer a structured psychological intervention of 3 to 12 sessions, and use brief contact follow-up. Since 84 percent of people presenting with self-harm do not repeat within 12 months, non-stigmatising outpatient care is the default.

TRAP Discharging on a low-risk label, because most eventual deaths occur in the low and moderate tiers that hold most patients; and offering the HCR-20 as a suicide tool, when it is a structured professional judgment instrument for interpersonal violence built on historical, clinical and risk-management domains.

VERIFY BEFORE THE EXAM Not printed for want of sources: the item count and cut-off of the Beck hopelessness scale, the item count of the Beck scale for suicide ideation, the item list and admission bands of the SAD PERSONS scale, the item and subscale counts of the HCR-20, and the statutory section numbers in the Mental Healthcare Act 2017 and the penal code provision it displaced. Safe to say: hopelessness self-report reached a pooled positive predictive value of 29.1 percent against a background of 16.3 percent, a positive likelihood ratio of 2.1; and SAD PERSONS, from 1983, performs no better than chance.

INDIAN PRACTICE

The Mental Healthcare Act 2017 decriminalised attempted suicide through a statutory presumption of severe stress and placed a duty on the appropriate Government to provide care, treatment and rehabilitation. Supported admission still turns on severe mental illness, high support needs and imminent risk on two independent assessments, not on a score.

The national suicide prevention strategy adds pesticide regulation, integration into primary health centres through the District Mental Health Programme and postvention, and Tele-MANAS is the national tele mental health service for crisis triage.

HOLD THIS

A rare outcome caps the positive predictive value of any test, however accurate. Pooled value for suicide is 5.5 percent, so the answer is needs assessment and means restriction, not a risk tier.

WHAT EARNS THE MARKS

- **Naming the base-rate ceiling**: a pooled positive predictive value of 5.5 percent for fatal suicide
- **Quoting the guideline position**: scales are not used to predict repetition or to set disposal
- **Substituting needs assessment**: treat the modifiable, restrict means, offer a structured intervention
- **Refusing the low-risk discharge**: most eventual deaths sit in the low and moderate tiers

SEE ALSO 02 Accuracy and cut-offs 11 Mood scales 20 Treatment rules

04 · Screening versus diagnosis: the structured interviews

PSYCHOMETRICS · TOPIC 04

What separates a screening instrument from a diagnostic interview?

VIVA TABLE: INSTRUMENTS; THE STRUCTURED INTERVIEW IS A STANDING SPOTTER

THE SHAPE OF THE ANSWER

1 **Two stages**	Case-finding first, verification second, with opposite statistical priorities
2 **The cut-off follows**	A screening threshold is set for sensitivity, not for agreement
3 **Format sets the rater**	Semi-structured needs a clinician, fully structured allows a lay interviewer
4 **The family**	Name each interview and give its one discriminator
5 **Indian feasibility**	Long instruments belong to research and epidemiology, not a crowded clinic

Assessment is a 2-stage process with opposite statistical priorities. Because psychiatry has no confirmatory biomarker, Stage 2 is a clinical or semi-structured interview.

- **Stage 1, screening.** Aims at case-finding, prioritises sensitivity, and its error is a missed diagnosis.
- **Stage 2, diagnostic evaluation.** Aims at verification, prioritises specificity, and its error is misdiagnosis.

That asymmetry sets how a cut-off is derived: prioritise sensitivity, rather than maximise agreement through the Youden index, because a patient who screens negative does not reach Stage 2. In a review of 68 reports on the 3 most researched bipolar screening scales, only 11 studies, 16.2 percent, prioritised sensitivity.

Format then decides who may administer. Semi-structured instruments supply questions but require the rater to probe, elicit examples and judge threshold, so they need a clinician. Fully structured instruments script every probe with closed questions and rigid branching, so trained lay interviewers can use them.

INSTRUMENT	STRUCTURE AND RATER	PARAMETERS	THE DISCRIMINATOR
SCID-5 clinician version	Semi-structured, clinically trained; adults aged 18 years and above	Commonest clinical disorders, omitting some lifetime assessments and the eating disorders; 30 to 120 minutes	**Published 2013 for DSM-5, superseding the 1994 edition**
SCID-5 research and trials versions	Semi-structured, clinician	Research core 45 to 120 minutes, enhanced 45 to 180; trials version 30 to 75 minutes	**Research version carries subtypes, severity and course specifiers; the trials version verifies eligibility only**
SCID-5-PD and AMPD	Semi-structured, clinician	Personality module covers the 10 categorical disorders in 30 to 120 minutes with a self-report screen; the alternative model uses 3 modules for functioning, 5 trait domains with 25 facets, and 6 disorders	**Categorical and dimensional models sit in separate instruments**
QuickSCID-5	Fully structured, closed questions, trainees may administer	30 minutes or less	**Screens psychotic symptoms and is not intended to diagnose a psychotic disorder**
MINI	Fully structured, lay or clinical rater	Sheehan and Lecrubier 1997; 17 conditions plus suicidality; 1 or 2 gate questions end a module; mean 21 minutes	**Brief by design: current disorders only, no severity or medical rule-out probes**
CIDI	Fully structured, trained lay interviewer	World Health Organization instrument for epidemiology; 92 minutes for the sections equivalent to the MINI	**Fully scripted, which is what permits lay administration**

INSTRUMENT	STRUCTURE AND RATER	PARAMETERS	THE DISCRIMINATOR
SCAN and PSE	Semi-structured, clinician	The present state examination rates present-state phenomena; the schedules expand it under World Health Organization auspices and yield ICD and DSM diagnoses	**Clinician rating of observed and reported phenomena, not self-report**
Kiddie-SADS	Semi-structured, clinician	Paediatric adaptation of the adult affective disorders and schizophrenia schedule; child and parent both interviewed	**The clinician reconciles informants into summary ratings; the paediatric MINI is the fully structured alternative**

Table: the interview family. Item counts and timings for the present state examination, the clinical assessment schedules and the paediatric schedule are not in the library sources.

RULE **A screening cut-off is set for sensitivity, not for agreement.** Maximising concordance looks rigorous and manufactures false negatives that leave the pathway.

TRAP Saying anyone can administer the SCID-5, or citing the brief interview as a 15-minute tool: that figure is secondary, and primary validation of 346 patients established a mean of 21 minutes against 92 for the comparable sections of the epidemiological interview, with weaker concordance for generalised anxiety disorder (kappa 0.36), agoraphobia (sensitivity 0.59) and bulimia (kappa 0.53).

INDIAN PRACTICE

Instruments taking 45 to 120 minutes are not feasible in a high-volume government outpatient department, and their place here is research, trial intake and community epidemiology under the District Mental Health Programme, where the Hindi translation of the brief interview and short screens do the work.

In the practical examination the long and short cases demand an unstructured clinical interview: leaning on an instrument during a case presentation is penalised, while the instruments themselves are examined at the table.

HOLD THIS

Screening maximises sensitivity, diagnosis maximises specificity, and a positive screen buys only the right to a Stage 2 interview.

WHAT EARNS THE MARKS

- **Getting the asymmetry right**: a missed case in stage one, a misdiagnosis in stage two
- **Naming the rater rule**: scripted probes and rigid branching are what permit lay administration
- **Carrying one timing pair**: a mean of 21 minutes against 92 for the comparable sections
- **Knowing what the practical wants**: an unstructured clinical interview in the long and short cases

SEE ALSO 01 Reliability and validity 06 Personality assessment 09 Spotters

05 · Intelligence testing, and certifying intellectual disability

PSYCHOMETRICS · TOPIC 05

This child scores 64. Will you certify intellectual disability?

VIVA TABLE: PSYCHOLOGICAL TESTS, AND THE CERTIFICATION STATION

THE SHAPE OF THE ANSWER

1	**Quotient arithmetic**	Deviation quotient, mean of 100, standard deviation of 15
2	**The instruments**	The adult scale, the Indian adaptation, and the age-scale alternative
3	**Age scale against point scale**	Mental age from one, comparable index scores from the other
4	**Three criteria together**	Quotient, concurrent adaptive deficits, onset in the developmental period
5	**Indian certification**	Cognitive test with an adaptive scale, read by a registered psychologist

Wechsler defined intelligence in 1939 as the global capacity to act purposefully, think rationally and deal effectively with the environment. Ratio quotients expressed mental age over chronological age and fail in adults, because cognitive development plateaus in late adolescence while chronological age rises.

Deviation quotients use a mean of 100 with a standard deviation of 15: about two thirds of adults fall between 85 and 115, about 95 percent between 70 and 130, and more than 99 percent between 55 and 145.

INSTRUMENT	AGES AND TIME	STRUCTURE	THE DISCRIMINATOR
WAIS-IV	Ages 16 to 90 years; 60 to 90 minutes	Released 2008; 10 core and 5 supplemental subtests across verbal comprehension, perceptual reasoning, working memory and processing speed; measures from 40 to 160	**A point scale, so subtest scatter across the four indices is the clinical product**
General ability index	Derived, not administered	The 6 core verbal comprehension and perceptual reasoning subtests, excluding working memory and processing speed	**Use it when motor slowing, head injury or severe anxiety depresses speed and drags the full scale below true reasoning**
MISIC	Ages 6 to 16 years; 60 to 90 minutes	Malin's Indian adaptation of the children's scale from Nagpur, removing cultural bias in verbal items; 11 subtests, 6 verbal and 5 performance, giving verbal, performance and full-scale quotients	**Indian guidelines mandate this or the third edition of the children's scale for specific learning disability certification**
Binet-Kamat test	Roughly ages 3 to 22 years; 45 to 60 minutes	Kamat's adaptation of the Stanford-Binet, published 1934 and revised 1967, standardised in Kannada and Marathi	**An age scale: a basal age where every task passes, a ceiling age where all fail, and credits between them build mental age**

RULE **Age scale against point scale is the question behind most intelligence vivas.** Age scales group items by the age at which most children pass and yield a mental age; point scales group items by content, award points and yield comparable index scores.

Intellectual disability needs three findings together.

- **The quotient.** A full scale quotient below 70, more than 2 standard deviations below the mean of 100.
- **Adaptive deficits.** Concurrent, across conceptual, social or practical domains.
- **Onset.** During the developmental period.

BAND	DESCRIPTOR	PERCENTILE
130 and above	Very superior	98 to 99.9
120 to 129	Superior	91 to 97
110 to 119	High average	75 to 90
90 to 109	Average	25 to 73
80 to 89	Low average	9 to 23
70 to 79	Borderline	2 to 8
69 and below	Intellectual disability range	0.01 to 2

CERTIFICATION IN INDIA

Statutory evaluation pairs a cognitive test, the Binet-Kamat or Malin's scale, with an adaptive scale. The Vineland social maturity scale rates social competence across communication, daily living, self-direction and socialisation by caregiver interview.

Certifying intellectual disability needs a documented score below 70 together with adaptive deficits, and the cognitive test is administered and interpreted by a psychologist registered with the Rehabilitation Council of India.

TRAP Quoting onset before age 22 as India's statutory limit. That figure belongs to United States social security criteria. State onset during the developmental period, and name the jurisdiction before quoting any age.

VERIFY BEFORE THE EXAM Not printed for want of sources: the subtyping bands into mild, moderate, severe and profound, which the library flags as historical strata outside its sources; the age range and index structure of the children's scale itself; and the item counts and age bands of the three Raven's progressive matrices forms, which sit as unverified notebook entries. Raven's is safe to name as a non-verbal test of fluid reasoning in coloured, standard and advanced forms, yielding percentile bands rather than quotient points.

HOLD THIS

A quotient below 70 is one of three criteria. Adaptive deficits and developmental onset carry equal weight, and in India the adaptive half is documented on the Vineland scale.

WHAT EARNS THE MARKS

- **Refusing to certify on a number alone**: adaptive deficits and developmental onset carry equal weight
- **Naming the adaptive instrument**: the Vineland scale documents the adaptive half in India
- **Distinguishing age from point scales**: this is the question behind most intelligence vivas
- **Declining the onset age of 22 years**: that figure belongs to another jurisdiction

SEE ALSO 01 Reliability and validity 09 Spotters 16 Function and disability

06 · Personality assessment: inventories and the personality interviews

PSYCHOMETRICS · TOPIC 06

You are handed an MMPI-2 profile. In what order do you read it?

VIVA TABLE: PSYCHOLOGICAL TESTS, WITH THE PERSONALITY INTERVIEW ALONGSIDE

THE SHAPE OF THE ANSWER

1 The instrument	Item count, reading level, and the uniform T-score metric
2 Validity indicators first	Non-content indices, then over-reporting, then under-reporting
3 Clinical scales and code types	Ten scales, and four code types worth carrying
4 Trait inventories	The factor inventories, and what a facet profile adds
5 Interview over inventory	Personality disorder is diagnosed by interview, with a developmental anchor

The MMPI-2 came from Hathaway and McKinley in 1943 and was restandardised in 1989. It is validated for adults aged 18 years and above reading at about a sixth-grade level, runs to 567 true or false items and takes 60 to 90 minutes; the restructured form of 2008 has 338 items and takes 35 to 50 minutes.

Scales were built by empirical criterion keying, so items were kept because they separated clinical groups from controls, whatever they looked like.

Scoring uses uniform T-scores with a mean of 50 and a standard deviation of 10, so a T-score means the same percentile on any scale: 65 to 74 is a clinically significant elevation, 75 and above marks acute pathology.

VALIDITY INDICATOR	STRUCTURE	THRESHOLD	THE DISCRIMINATOR
Cannot say, and the consistency pair	Omissions; variable response inconsistency from 47 item pairs; true response inconsistency from 20 opposite-content pairs with 3 scored symmetrically	More than 10 omissions degrades reliability, 20 warrants caution, more than 30 flags re-administration; either consistency index above T 80 invalidates	**Non-content indicators are read before any content indicator**
F and Fb	F is 60 items within the first 370, endorsed by under 10 percent of the normative sample; Fb is 40 infrequent items after item 280	F above T 70 distress, above 90 psychotic states, above 99 exaggeration or error; Fb exceeding F by more than 20 T-points	**A high F alone is not feigning: acute psychosis and profound depression raise it**
Fp and FBS	Fp is 27 items rarely endorsed even by inpatients; the symptom validity scale is 43 somatic and atypical-belief items	Fp at T 100 or above with both consistency indices below 70; symptom validity at T 100 or above	**This combination indicates over-reporting; F high with Fp below 70 reads as genuine severe illness**
L, K and S	L has 15 obvious items keyed false, K has 30 subtle items, S has 50 items with 5 subscales	Read together as under-reporting	**In Indian samples social conformity and family deference inflate L and K**

The 10 clinical scales run hypochondriasis, depression, hysteria, psychopathic deviate, masculinity-femininity, paranoia, psychasthenia, schizophrenia, hypomania and social introversion. Code types take the highest scales, excluding 5 and 0, and a code is defined when the lowest scale in it exceeds the next non-code scale by at least 5 T-points.

Learn four code types.

- **2-7.** Anxious-depressive, and motivated for therapy.
- **1-3 with scale 2 lower.** The conversion V: somatic complaint with denial of affect.
- **4-9.** Antisocial and sensation-seeking.
- **6-8.** Paranoid-psychotic, strongest when both exceed scale 7.

INVENTORY	STRUCTURE AND TIME	THE DISCRIMINATOR
16PF fifth edition	185 items, three-choice, 30 to 40 minutes; from the Allport and Odbert pool of about 4,500 trait terms of 1936, reduced to 171 clusters then 16 oblique primary factors; first published 1949, fifth edition 1993	**Because the primaries correlate, second-order analysis collapses them into 5 global factors mapping onto the Big Five**
NEO-PI-R	240 items on 5 points, 35 to 45 minutes; Costa and McCrae 1992; 5 domains with 6 facets each, 30 in all; domain alphas .86 to .92, replicated across more than 50 cultures	**The facet profile within a domain is what it adds over a global score**
EPQ-R	Eysenck's three biological superfactors: psychoticism, extraversion, neuroticism	**Psychoticism is not clinical psychosis: it indexes nonconformity, hostility, impulsivity and low conscientiousness, and joint factoring in 1995 placed conscientiousness inside it**

Table: the trait inventories. The 16PF and the Big Five are not rival theories; they come from the same 1936 lexical pool.

Personality disorder is diagnosed by interview.

The international personality disorder examination, from Loranger and colleagues under the joint World Health Organization and National Institutes of Health programme, runs parallel modules for the ICD-10 classification with its 10 disorders and the DSM-IV classification with 11.

Its self-administered screener is forced-choice, 77 items in the DSM-IV module and 59 in the ICD-10 module, at a fourth-grade reading level, in 15 minutes or less.

The interview follows, organised by life rubrics, background, work, self and interpersonal relationships, taking 1 to 2 hours with about 30 minutes of scoring for ages 18 to 70 years, and returns a definite, probable or negative classification with a dimensional profile.

RULE **At least 1 criterion must have been recognisably present before age 25 years before a personality disorder is diagnosed on this instrument.** The anchor is what stops a state-dependent mood, psychotic or post-traumatic presentation being scored as trait.

SCREENING CONTEXT	DISORDER	MANUAL CUT-OFF	VALIDATED CUT-OFF
Primary, community detection	ICD-10 impulsive and borderline	3 items	**3 items, preserving sensitivity**
Primary, community detection	DSM-IV borderline	3 items	**5 items, because 3 gives unacceptable false positives**
Secondary, specialist clinic	ICD-10 impulsive and borderline	3 items	**4 items, sparing unnecessary interviews**
Secondary, specialist clinic	DSM-IV borderline	3 items	**6 items, where affective instability coexists**

Table: screener thresholds from a clinical adolescent sample of 125 inpatients, mean age 15.9 years; the primary study supersedes the manual rule.

TRAP Reading clinical scales before validity scales, or offering a scale 4 elevation as antisocial personality disorder: it indexes authority conflict, familial discord and nonconformity. Clinical scales also share a demoralisation factor, so name the Harris-Lingoes subscale that drove the elevation.

VERIFY BEFORE THE EXAM The DSM-5 personality interview is organised criterion by criterion rather than by life rubric, with a screener that lets negatively endorsed criteria be skipped; its per-criterion anchors and screener item count are not in the sources. Neither is any claim of Indian-language field testing of the personality examination, which the library flags as unsupported.

HOLD THIS

Validity scales before clinical scales, interview before inventory, and 1 criterion before age 25 years before any personality disorder is named.

WHAT EARNS THE MARKS

- **Reading validity before content**: the commonest sequencing error in this station
- **Separating real severity from over-reporting**: a high F with a low Fp reads as genuine illness
- **Naming the developmental anchor**: one criterion recognisably present before age 25 years
- **Refusing a diagnosis from one elevation**: name the subscale that drove it instead

SEE ALSO 01 Reliability and validity 05 Intelligence testing 07 Projective tests

07 · Projective tests, and how to defend them

PSYCHOMETRICS · TOPIC 07

Defend the use of the Rorschach in a modern psychiatric assessment.

VIVA TABLE: PSYCHOLOGICAL TESTS; THE PLATES ARE A STANDING SPOTTER

THE SHAPE OF THE ANSWER

1 **The hypothesis**	An ambiguous stimulus forces the person to impose their own organisation
2 **Materials and seating**	Ten plates, fixed order, side by side, latency recorded

3 Two phases	Free association, then inquiry, which is what permits coding
4 Coding dimensions	Location, determinants, form quality, content, popularity, organisation
5 The defensible claim	Formal thought disorder, not categorical diagnosis and not risk prediction

The projective hypothesis holds that an ambiguous stimulus forces the person to impose their own perceptual organisation, needs, conflicts and coping style.

Rorschach developed the inkblots in 1921 and published them from a sample of 300 psychiatric patients and 100 controls as an empirical aid to diagnosing schizophrenia; adoption as a general personality measure came in 1939.

The set is **10 plates, each about 18 by 24 cm, each near-perfectly bilaterally symmetrical**. That symmetry balanced the figures, equalised conditions for right and left-handed examinees and helped blocked subjects respond.

- **Achromatic.** Five plates: I, IV, V, VI and VII.
- **With red added.** Two plates: II and III.
- **Polychromatic.** Three plates: VIII, IX and X.

Examiner and examinee sit side by side with the examiner slightly behind, so facial or postural cues cannot prompt a response. In free association the cards are given in fixed order from I to X, rotation is permitted, and everything said and done is recorded verbatim including latency.

In inquiry each card returns in the same order and the examinee shows where the percept was seen and what made it look that way, which is what permits coding.

CODING DIMENSION	CODES	THE DISCRIMINATOR
Location	Whole, common detail, unusual detail, white space	**Whole reads as integrative effort, unusual detail in quantity as perfectionism or distress, white space as oppositionality**
Determinants	Form, movement (human, animal, inanimate), colour, shading	**Form-colour is affect under intellectual control, colour-form is spontaneity with secondary control, pure colour is poorly restrained affect**
Form quality	Fit of percept to blot contour	**Exner defined it against empirical frequency tables, replacing artistic impression**
Content, popularity, organisation	27 content categories with idiographic content for the rest; popular responses given by at least one third of normative samples; organisational activity and deviance scores	**Content is under conscious control, so deviant verbalisation and illogical combination carry the weight**

Table: the Exner comprehensive system, built through the 1960s and 1970s to unify the Beck, Klopfer, Hertz and Rapaport systems, with the performance assessment system as its later revision.

THE CARDS THEY ASK ABOUT

Card I is the new and stressful task. Cards II and III carry red, pulling blood, harm, anger and sexual content, and Card III reads interpersonal capacity, with two humans interacting given by 72 percent and 76 percent in the two normative series.

Card IV is the imposing figure seen from below, conventionally the father card, its dense shading burdening depressed examinees. Card V is the easiest silhouette, butterfly at 48 percent and bat at 40 percent. Card VI carries the heaviest texture and pulls closeness. Card VII is conventionally the mother card.

Card VIII is the first fully coloured plate, the climbing four-legged animal given by 94 percent and 93 percent. Card IX has the fewest popular responses, and Card X is the fragmented sign-out card.

The position on validity should be specific. Cronbach held in 1959 that the test failed practical predictive criteria; despite more than 2,000 publications many dynamic interpretations stayed unvalidated, and a moratorium was proposed in 1999. The Chapmans showed illusory correlation, clinicians reporting associations that fitted intuition while missing empirical signs that did not.

Comprehensive system norms classified about 50 percent of healthy subjects as showing distorted thinking, while a later series of 4,704 protocols across 17 countries found thinking disorder elevations in 2 percent, depression and hypervigilance in 12 percent and stress overload in 13 percent, matching non-patient base rates.

A 2003 review found specific indices perform above chance: the real use is as an objective measure of formal thought disorder, it correlates with intelligence through productivity and form quality, and it helps identify borderline organisation, with no support for categorical diagnosis or for predicting suicide or violence.

OTHER PROJECTIVE INSTRUMENTS	WHAT IT IS	THE DISCRIMINATOR
Thematic apperception test	Large cards of ambiguous monochrome interpersonal scenes with one blank card; the examinee builds a story with beginning, middle and end	**Analysis names the hero, the needs, the press and the outcome**
Children's apperception test	An apperceptive test in which animal figures replace human ones	**Name it in the family; its age band and themes sit outside the library sources**
Sentence completion	Semi-structured stems; the Sacks form covers family relations, sexual attitudes, interpersonal relations and self-concept, with a Dubey and Dubey Indian adaptation	**Semi-structured, so it sits between inventory and inkblot; stem counts are unverified notebook entries**
Draw-a-person	A whole human figure, then a figure of the opposite sex	**Scored on graphic features, size, placement, line pressure, proportion, clothing and omissions, not content symbolism**

RULE **Present projective tests as adjunctive and hypothesis-generating,** inside an evaluation with history, mental state examination, an objective inventory and formal cognitive testing. Claiming a categorical diagnosis from an inkblot is the failure point here, and the folk card names are convention, not validated fact.

VERIFY BEFORE THE EXAM The total card count of the apperception series, the number administered per session and the age and gender subsets are not in the sources, nor are the developers and age band of the children's version. Two points the library does settle: the scoring system to name is Exner's comprehensive system, and Indian Rorschach work is traced to Prof. H. S. Asthana as an unverified notebook entry.

HOLD THIS

Structural determinants outrank content, and the defensible use is formal thought disorder, not diagnosis. Side by side, free association, then inquiry.

WHAT EARNS THE MARKS

- **Conceding the validity critique before it is raised**: illusory correlation, and the norms problem
- **Naming Exner's comprehensive system**: form quality against frequency tables, not artistic impression
- **Putting structure above content**: content is under conscious control, deviant verbalisation is not
- **Framing it as adjunctive**: inside history, mental state, an inventory and cognitive testing

SEE ALSO 06 Personality assessment 08 Neuropsychology 09 Spotters

08 · Neuropsychological assessment and the frontal tests

PSYCHOMETRICS · TOPIC 08

Why did you ask for a neuropsychological battery in this patient?

VIVA TABLE: PSYCHOLOGICAL TESTS; THE BENDER CARDS ARE THE USUAL SPOTTER

THE SHAPE OF THE ANSWER

1 **The indications**	Separate functional from organic, profile before treatment, map for rehabilitation
2 **The Bender test**	Nine designs, copied on blank paper without a time limit
3 **Scoring systems**	The adult organicity system, the children's system, and recall protocols
4 **Indian batteries**	Stratified local norms make a low-education profile interpretable
5 **Frontal tests**	Card sorting, trails, Stroop and complex figure, each with its sign

Assessment is requested to separate functional illness from early or atypical organic disorder, to profile cognition before neurosurgery, electroconvulsive therapy or a demanding regimen, to characterise decline in epilepsy, cerebrovascular disease, head injury and metabolic encephalopathy, to map preserved against compromised systems for rehabilitation, and to catch diffuse impairment that bedside screening misses.

The Bender visual-motor gestalt test is the one they hand you.

Bender developed it in 1932 and published it in 1938, with instructions in 1946. It uses 9 designs adapted from Wertheimer, the first labelled A and the rest numbered 1 through 8, of which only A, 3, 7 and 8 closely resemble the originals, copied on blank paper without time limit.

Gestalt reproduction matures by age 12; between ages 5 and 10 it correlates moderately with mental age, and beyond 12 it stops tracking intelligence. Copy loads parietal spatial synthesis and constructive praxis; recall loads temporal visual retention.

SYSTEM	STRUCTURE	METRIC	THE DISCRIMINATOR
Pascal and Suttell, 1951	105 scoring factors, standardised on 271 high-school and 203 college subjects	Raw score to a Z-score, with 9 deviations as organic indicators	**Separates psychiatric patients from non-patients and is cumbersome at the bedside**
Koppitz, 1964	30 objective items from 7 developmental categories; refined on 165 children in 6 schools, normed on more than 1,100 children in 12 schools	Worse than 1 standard deviation below the age mean raises suspicion	**The children's system, for ages 5 to 10 years**
Hain, 1964	15 weighted signs valued 1 to 4 by frequency in brain damage, total 0 to 32, inter-rater r 0.93	Normal 0 to 5, borderline 6 to 12, critical 13 or more, optimal single cut-off at 8 or below	**The adult organicity system: sensitive to diffuse disease, false-negative with focal lesions**
Recall and interference protocols	Designs redrawn from memory; or a second copy onto paper printed with intersecting curved lines, scorer agreement r 0.89	Organic patients recalled 3.69 designs against 5.53 in psychogenic controls, and degrade markedly under visual noise (p below 0.001)	**Recall separates temporal memory failure from parietal constructional failure**
Three-trial and Hutt methods	3 trials with a 2-minute break before the third; the Hutt version adds copy, elaboration and association phases rating 27 personality factors	Non-organic subjects improve by the third trial	**Failure to benefit from repetition is the organic sign**

THE INDIAN BATTERIES

The PGI battery of brain dysfunction was built at the postgraduate institute in Chandigarh for culturally validated assessment, and **its memory scale is the component to know**: standardised in Hindi and English for varying literacy.

- **Key number.** The memory scale covers 10 domains.
- **The first five.** Remote memory, recent memory, mental balance, attention and concentration, delayed recall.
- **The rest.** Immediate recall, retention of similar pairs, retention of dissimilar pairs, visual retention, recognition.
- **Sensitive to.** Postoperative decline, epileptic deficits, head injury sequelae and dementing illness.

The NIMHANS battery from Shobini Rao and colleagues and the AIIMS battery built on Luria's functional-systems model carry stratified Indian norms, which is what stops a low-education patient being read as impaired; their test counts and age bands are unverified notebook entries.

RULE **The memory scale is one component inside the battery, not the battery.** Halstead-Reitan is a fixed quantitative battery inferring damage from statistical deviation, summarised as an impairment index; Luria-Nebraska standardises Luria's probes on functional-systems theory with education-adjusted profiles, and its value is rehabilitation, naming the broken link and the intact components.

FRONTAL TEST	WHAT IT LOADS	THE DISCRIMINATOR
Wisconsin card sorting test	Dorsolateral prefrontal cortex and frontostriatal circuits; cards matched to 4 stimulus cards varying on 3 dimensions, colour, form and number, with the rule shifting unannounced	**Perseverative errors index inflexibility, non-perseverative errors inattention, and failure to maintain set means losing a rule already found**
Trail making test	Speed on A, set-shifting on B, which alternates numbers and letters	**B prolonged with A normal localises executive shifting; both prolonged means generalised slowing, sedation or poor acuity**
Stroop test	Anterior cingulate for conflict monitoring, dorsolateral prefrontal cortex for inhibition; word reading, colour naming, then naming ink colour of a conflicting word	**Interference is the time or error penalty for suppressing the automatic reading response**
Rey-Osterrieth figure	Right parietal configuration, frontal planning, right temporal visual memory; copy, immediate recall, delayed recall, optional recognition, with drawing order tracked by switching pencils	**Frontal patients draw piecemeal without a frame, right parietal patients distort the copy itself, right temporal patients copy well and lose detail on delayed recall**

Table: the executive tests. Their numerical parameters are not in the library sources, so none are printed.

TRAP Reading a normal Bender record as excluding organicity: false negatives are common with focal lesions, localised infarcts, low-grade tumours and circumscribed epileptic foci. Equally, do not overcall rotation of 30 or 45 degrees, which is developmental, present in over 50 percent at age 6 and only 7 percent between 7.5 and 9 years, and can be induced when card and paper are in discordant orientations; perseveration is normal through age 7 and points to injury at or beyond age 8.

VERIFY BEFORE THE EXAM Not printed for want of sources: the card counts, criterion count and maximum categories of the card sorting test; the item counts and cut-off times of the trail making test; the unit count, maximum score and recall intervals of the complex figure; the scale and item counts, cut-offs and durations of the Halstead-Reitan and Luria-Nebraska batteries; and the component count of the PGI battery, including whether the Bender test is separate or sits inside the Nahar-Benson test of visual retention built on Bender figures.

What is the task asking?

Original concept demonstrations — not standardised test items or scoring forms.

1 / Inhibition

Say the ink colour, not the word.

BLUE

Competing word and colour responses.

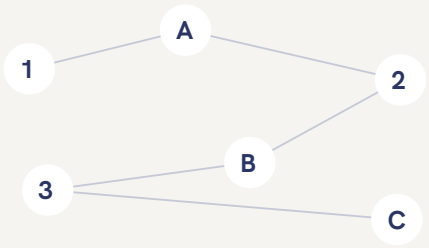
2 / Switch the rule

Sort by colour, then by shape.

A changed rule requires a changed set.

3 / Alternate

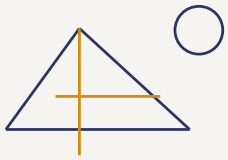
Follow 1 → A → 2 → B → 3 → C.



A novel sequence, not a Trail Making form.

4 / Copy, then recall

Observe → copy → remove → recall.



Original figure: no Rey or Bender design.

Schematic ▪ Original teaching diagram ▪ September 2026

Schematic. Four original concept demonstrations: colour-word interference, a changed sorting rule, alternating symbols, and copying then recalling a novel geometric figure. The coloured word, sorting tokens, alternating sequence and geometric figure are original teaching examples. They are not standardised test forms, validated tasks, scoring materials or substitutes for the appropriate manual and norms.

Name the cognitive demand and explain how instructions change the task.

Schematic prepared by Luna for Weave Viva, based on P5D-05. Original vector diagram.

HOLD THIS

Copy is parietal, recall is temporal, organisation is frontal. Halstead-Reitan localises, Luria-Nebraska rehabilitates, Indian norms make a low-education profile interpretable.

WHAT EARNS THE MARKS

- **Localising by task**: copy is parietal, recall is temporal, organisation is frontal
- **Naming what each battery is for**: one localises by statistical deviation, the other guides rehabilitation
- **Reading a normal record cautiously**: false negatives are common with focal lesions
- **Separating developmental from pathological signs**: rotation and perseveration both have age expectations

SEE ALSO 05 Intelligence testing 07 Projective tests 15 Cognitive screening

09 · Spotters: the test materials on the table

PSYCHOMETRICS · TOPIC 09

Identify this material, and tell me what it measures.

THE SPOTTER TABLE, WHERE SEVERAL OF THESE SIT TOGETHER

THE SHAPE OF THE ANSWER

1 Name it	The material, then the construct it samples
2 Age band	The range it is normed for, and where the ceiling falls
3 Administration	Trials, timing, and what the examiner records
4 Score conversion	What the raw figure becomes, and against which norm
5 The limitation	Volunteer it: each of these is a partial measure

The spotter runs to a fixed script. Volunteer the limitation before it is asked, because each of these is a partial measure.

- **Name it.** The material, then the construct.
- **Place it.** The age band it is normed for.
- **Run it.** Administration and trials.
- **Convert it.** Say how the score converts.

ON THE TABLE	WHAT IT IS	WHAT IT MEASURES	THE ONE LINE
Seguin form board	A wooden board measuring 4 cm by 48 cm by 34 cm with 10 shape recesses and 10 loose blocks: circle, square, rectangle, triangle, cross, rhombus, star, semi-circle, irregular hexagon, oblong	Visual-motor coordination, spatial perception, visual discrimination, matching, psychomotor speed	**3 timed trials with time in seconds and errors; mental age from the shortest time; Indian norms 3 years to 8 years 11 months with the ceiling at 9 years, so no ratio quotient beyond it**

ON THE TABLE	WHAT IT IS	WHAT IT MEASURES	THE ONE LINE
Alexander pass-along test	A shallow wooden frame of flat coloured blocks with graded pattern cards; W. P. Alexander, 1935	Non-verbal practical intelligence, spatial planning, route sequencing, foresight	**Blocks slide within the frame and are not lifted; 15 to 30 minutes**
Kohs block design test	Small cubes with solid and split-colour diagonal faces, with printed design cards	Spatial construction and analysis-synthesis	**Loose cubes rotated freely on an open table, the contrast with the framed pass-along test; both sit in Bhatia's battery, standardised in India in 1955**
Bender gestalt cards	9 geometric designs in black ink on separate white cards	Visual-motor integration, spatial relationships, graphomotor execution	**Rotation, perseveration, distortion, fragmentation and loss of gestalt coherence are the signs read for organicity**
Rorschach plates	10 stiff plates measuring 6 5/8 by 9 1/5 inches, published under copyright by Verlag Hans Huber, Bern	Perceptual organisation, thought quality, affective control, coping style	**Fixed order I to X, side-by-side seating, association of 10 to 15 minutes then inquiry of 20 to 30 minutes, with scoring up to 2 hours**
Thematic apperception cards	Large monochrome or sepia interpersonal scenes, with one blank card	Personality dynamics, interpersonal conflict, motives, defences, needs	**A story with beginning, middle and end, read for hero, needs and press**
Raven's matrices booklet	A bound booklet of abstract matrix problems, each a patterned figure with a missing piece and 6 to 8 options beneath	General ability, non-verbal eductive reasoning, abstraction, pattern deduction	**Minimal linguistic demand, so it works across non-literate and multilingual populations and in motor speech deficits**
Vineland social maturity scale form	A multi-page record form of developmental behaviours in ascending age brackets	Self-sufficiency, social competence, practical adaptive functioning	**Caregiver interview yielding a social age, converted to a social quotient against chronological age**

WHERE THESE SIT IN INDIAN PRACTICE

The form board is the rapid performance screen of paediatric clinics and district early intervention centres, and suits shy or speech-impaired children because verbal load is minimal. It cannot diagnose intellectual disability alone: an adaptive scale sits beside the cognitive measure, and work feeding legal certification is done by psychologists registered with the Rehabilitation Council of India.

RULE **Pass-along slides, Kohs lifts.** Two more pairings: Plate IV is the massive dark heavily shaded figure seen from below, Plate VII is light grey with an open central space and an upward notch, read as women's or children's heads; and the form board is a three-dimensional timed placement task while the Gesell drawing test is two-dimensional copying of graphomotor milestones.

VERIFY BEFORE THE EXAM Not printed for want of sources: the cube and design-card counts of the Kohs kit, the total card count of the apperception series and the number given per session, the item counts and subscale numbers of the Vineland form, the problem count and series breakdown of the Raven's booklet, and the subtest counts of Bhatia's battery and of the PGI battery. Learn one figure for each from a manual.

HOLD THIS

Name it, say what it measures, give the age band, then say what it cannot do. Every performance spotter needs an adaptive scale beside it before certification follows.

WHAT EARNS THE MARKS

- **Answering in the fixed order**: name, construct, age band, administration, conversion, limitation
- **Holding the paired discriminators**: the pass-along blocks slide, the Kohs cubes lift
- **Refusing to certify from a performance test**: an adaptive scale sits beside the cognitive measure
- **Volunteering the ceiling**: no ratio quotient beyond the age at which the norms stop

SEE ALSO 05 Intelligence testing 07 Projective tests 08 Neuropsychology

Rating scales by domain

10 · Psychotic disorder scales

RATING SCALES · TOPIC 10

Rate this patient on the PANSS, and tell me what a response would be.

VIVA TABLE: RATING SCALES; PAIRED WITH THE SCHIZOPHRENIA LONG CASE

THE SHAPE OF THE ANSWER

1	**Provenance and structure**	Three subscales, 30 items, each rated 1 to 7
2	**Enumerate a subscale**	The seven positive items, then the seven negative ones
3	**The floor**	A symptom-free patient scores 30, because each item starts at 1
4	**Response and remission**	A 20 percent reduction after the 30-point correction

5 The companions

Brief rating scale, the negative-symptom pair, the newer negative interview

The positive and negative syndrome scale was published in 1987 by Kay, Opler and Fiszbein.

- **How it was built.** The 18-item brief psychiatric rating scale merged with 12 items from the psychopathology rating schedule, to cover negative symptoms and general psychopathology.
- **Administration.** Through the structured interview, 45 to 50 minutes, with collateral from nursing staff or family.

SUBSCALE	ITEMS	RANGE	THE ITEMS TO ENUMERATE
Positive	7	7 to 49	**Delusions, conceptual disorganisation, hallucinatory behaviour, excitement, grandiosity, suspiciousness, hostility**
Negative	7	7 to 49	**Blunted affect, emotional withdrawal, poor rapport, passive social withdrawal, difficulty in abstract thinking, lack of spontaneity, stereotyped thinking**
General psychopathology	16	16 to 112	**Carries insight, depression, anxiety and impulse control, so totals move when nothing psychotic has changed**
Total	30 items rated 1 to 7	30 to 210	**A symptom-free patient scores 30, not 0, because every item starts at 1**

- RULE **Response is a 20 percent reduction in total score, calculated after subtracting 30 from both baseline and endpoint.** Remission on the brief 6-item version is a total below 14, validated against a global severity impression of 3 or less. Persistent negative symptoms need negative symptoms for at least 6 months during stability with low positive symptoms, and illness duration of at least 6 months.

SCALE	STRUCTURE	THE DISCRIMINATOR
BPRS	Overall and Gorham, 1962; 18 symptom constructs scored 1 to 7; 20 to 30 minutes	**Rates interview plus behaviour over the preceding 2 to 3 days, with collateral from family**
SAPS and SANS	Andreasen, dated 1981 and 1984 in the primary listing; inter-rater intraclass correlations 0.70 to 0.88 on global ratings	**The pair that showed negative symptoms rate as reliably as positive ones; no response or remission cut-offs in the sources**
CAINS	Built after the 2006 consensus conference for 5 subdomains: asociality, avolition, anhedonia, blunted affect, alogia; examined in 281 outpatients	**Two factors, experiential and expressive, and it separates anticipatory from consummatory pleasure**
CGI and PSYRATS	Global impression splits into severity and improvement; the symptom rating scales rate dimensions of hallucinations and delusions	**Global impression is the external anchor other scales are validated against**

TRAP Confusing the two 6-item versions: the Rasch-derived one takes delusions, conceptual disorganisation and hallucinations with blunted affect, social withdrawal and lack of spontaneity, while the correlation-derived brief version includes tension and suspiciousness. And a high negative score is not the deficit syndrome: depression, parkinsonian bradykinesia, deprivation and active persecutory delusions all give secondary negative symptoms.

VERIFY BEFORE THE EXAM The consensus remission criteria for the full 30-item scale, its item-level threshold and required duration, are not in the sources, nor is the item count of the excited component or the structure and cut-offs of the symptom rating scales.

HOLD THIS

Thirty items in 3 subscales, floor 30, ceiling 210, response 20 percent after the 30-point correction.

WHAT EARNS THE MARKS

- **Correcting for the floor before calculating response**: subtract 30 from baseline and from endpoint
- **Enumerating items rather than gesturing at them**: the subscale lists are the examinable content
- **Refusing to read a high negative score as the deficit syndrome**: name the secondary causes
- **Naming the general-subscale problem**: it carries insight, mood and anxiety, so totals drift

SEE ALSO 11 Mood scales 14 Movement scales 16 Function and disability

11 · Mood and mania scales

RATING SCALES · TOPIC 11

What does a Hamilton score of 16 tell you, and what does it not?

VIVA TABLE: RATING SCALES; ALSO THE DEPRESSION LONG CASE

THE SHAPE OF THE ANSWER

1 **The disclaimer first**	Severity and change in a person already diagnosed
2 **Structure**	Seventeen items summed, four more describing subtype only
3 **The bands**	Remission, mild, moderate, severe, on both clinician scales
4 **The change-sensitive scale**	Ten items, 0 to 60, better in medical illness
5 **The limit**	No depression scale separates unipolar from bipolar depression

Open with the disclaimer: the Hamilton depression rating scale, from 1960, measures severity and change in people already diagnosed and cannot make the diagnosis. Its content validity is incomplete, omitting hypersomnia, hyperphagia, reverse vegetative features, indecisiveness and marked anhedonia, while somatic items, anxiety and drug side effects inflate it in the medically ill.

It needs a trained rater and takes 15 to 30 minutes.

- **Items 1 to 17.** Severity over the preceding 7 days, and these are the ones summed.
- **Items 18 to 21.** Diurnal variation, depersonalisation and derealisation, paranoid symptoms, obsessional symptoms: they describe subtype and stay out of the total.

Items score 0 to 2 or 0 to 4, and retardation and agitation are rated from observation.

STATE	HAM-D-17	MADRS	THE DISCRIMINATOR
Remission	Less than 7, and 7 or less in trials	0 to 8, or under 9 in most of the literature	**Remission is an absolute score; response is a 50 percent reduction from baseline**
Mild	8 to 13	9 to 17	**The scales are not interchangeable point for point**
Moderate	14 to 18	18 to 34	**Trial entry sits at 14 to 20 on the Hamilton scale**
Severe	19 to 22, very severe above 23	Above 35	**These Hamilton bands are the Blacker cut-offs**

The Montgomery-Asberg scale, from 1979, was engineered for sensitivity to change: 10 items scored 0 to 6 for a total of 0 to 60, rated over 2 weeks, taking 20 to 40 minutes.

Its items are apparent sadness, reported sadness, inner tension, reduced sleep, reduced appetite, concentration difficulties, lassitude, inability to feel, pessimistic thoughts and suicidal thoughts,

and because it excludes somatic complaint, hypochondriasis and motor retardation it is the better instrument in medical illness.

Equipercentile linking of 4388 patients across 31 trials gives the conversions: Hamilton 10 corresponds to 13, 20 to 26, 30 to 39, 40 to 52 or 53, while percentage change transfers almost unchanged.

Against the Beck depression inventory, 10 corresponds to 13, 20 to 32 and 30 to 50. The self-report pair is that 21-item inventory at 5 to 10 minutes and the 9-item patient health questionnaire at 3 minutes, neither of which records psychomotor change.

RULE **No depression rating scale separates unipolar from bipolar depression.** In 91 patients the area under the curve ran 0.34 to 0.64, below the 0.70 benchmark, so there is no cut-off to quote; item-level differences exist, but the distinction rests on longitudinal history.

TRAP Summing all 21 items for severity, or offering a total as a diagnosis. In an elderly medical inpatient the somatic items raise the total without more depression, which is the case for switching scales.

VERIFY BEFORE THE EXAM Not printed for want of sources: the cut-offs and total ranges of the patient health questionnaire and the Beck depression inventory; the items, timeframe, anchors and cut-offs of the young mania rating scale; the symptom count, co-occurrence rule and positive-screen threshold of the mood disorder questionnaire; the range and cut-off of the Edinburgh postnatal depression scale; and the items and cut-off of the Calgary depression scale for schizophrenia. Give the rationale instead: the postnatal scale excludes the constitutional and somatic symptoms of pregnancy and the puerperium, and the Calgary scale separates depression from negative symptoms and from drug-induced akinesia.

HOLD THIS

Seventeen items for severity, four more for subtype, remission at 7 or less, response at 50 percent.

WHAT EARNS THE MARKS

- **Opening with what the scale cannot do**: it grades severity and does not diagnose
- **Keeping items 18 to 21 out of the total**: they describe subtype rather than severity
- **Naming the content gaps**: reverse vegetative features and marked anhedonia are missing
- **Refusing a cut-off for polarity**: the distinction rests on longitudinal history

SEE ALSO 03 Risk scales 10 Psychosis scales 12 Anxiety and OCD scales

12 · Anxiety, OCD, trauma and dissociation scales

RATING SCALES · TOPIC 12

Give me the Y-BOCS: items, range, entry cut-off, response and remission.

VIVA TABLE: RATING SCALES; THE SYMPTOM CHECKLIST IS SOMETIMES THE SPOTTER

THE SHAPE OF THE ANSWER

1	**The obsessive-compulsive scale**	Ten items, 0 to 40, five dimensions repeated twice
2	**The decision points**	Entry at 16, response by percentage, remission at 12 or less
3	**Checklist against scale**	The checklist sets targets and contributes nothing numerical
4	**The anxiety pair**	Clinician-rated 14 items, self-report 7 items, each with bands
5	**The variants**	Paediatric bands, the autism version, and the trauma checklist

- **Sort them first.** Clinician-rated instruments, then self-report instruments, then the paediatric and disability variants.
- **Then read down.** Type and items, then range and bands, then the one discriminator.
- **Trap.** Bands are scale-specific, so a total means nothing until the instrument is named.

SCALE	TYPE AND ITEMS	RANGE AND BANDS	THE DISCRIMINATOR
HAM-A	Clinician-rated, 14 items scored 0 to 4; Hamilton 1959; 10 to 15 minutes; prior week	0 to 56; below 17 mild, 18 to 24 mild to moderate, 25 to 30 moderate to severe, 31 to 56 severe to very severe	**Items 1 to 6 and 14 are psychic anxiety, 7 to 13 somatic; item 6 rates depressed mood, so it separates anxiolytic from antidepressant effect poorly**
GAD-7	Self-report, 7 items scored 0 to 3, past 2 weeks, plus an unnumbered impairment item	0 to 21; 0 to 4 minimal, 5 to 9 mild, 10 to 14 moderate, 15 to 21 severe	**At 10, sensitivity 89 percent and specificity 82 percent with a positive likelihood ratio of 5.1; at 8, sensitivity 92 percent with specificity 76 percent; at 15, specificity 95 percent with sensitivity 48 percent**
BAI	Self-report, 21 items scored 0 to 3, past month including today	0 to 63; 0 to 21 low, 22 to 35 moderate, 36 and above concerning	**Weighted to autonomic and panic-like sensations, so it correlates r 0.25 with the depression scale and r 0.51 with the HAM-A**
Y-BOCS	Clinician-rated, 10 items scored 0 to 4, prior week to interview; Goodman and colleagues, 1989	0 to 40; 0 to 7 subclinical, 8 to 15 mild, 16 to 23 moderate, 24 to 31 severe, 32 to 40 extreme	**Obsessions and compulsions weighted equally and content ignored; the 5 dimensions repeat for each, time occupied, interference, distress, resistance, degree of control**
CY-BOCS	Clinician interview, same 10 items, past week, children and adolescents	0 to 40, with a lifespan banding of 0 to 13, 14 to 21, 22 to 29 and 30 to 40	**Same architecture, different bands**

SCALE	TYPE AND ITEMS	RANGE AND BANDS	THE DISCRIMINATOR
CY-BOCS-ASD	Caregiver interview; obsession checklist and the 5 obsession items dropped	0 to 20 from the 5 compulsion items	**A child with autism spectrum disorder or intellectual disability cannot reliably report intrusive thoughts, so only repetitive behaviour is rated**
PCL-5	Self-report, 20 items, past month	Probable post-traumatic stress disorder at 33 or above	**Items map onto the four clusters: intrusion, avoidance, negative alterations in cognition and mood, altered arousal and reactivity**

RULE **On the obsessive-compulsive scale, 16 is the trial entry cut-off, response is a reduction of at least 25 percent (35 percent or more in stringent pharmacotherapy and neurosurgical protocols), and remission is a post-treatment total of 12 or less.** Baseline cohort means sit at 24.7, and the time anchors run none, under 1 hour, 1 to 3 hours, 3 to 8 hours and more than 8 hours a day.

TRAP Mistaking the symptom checklist for the scored instrument. The checklist establishes the target obsessions and compulsions across themes, contamination, checking, symmetry, aggressive and sexual obsessions, and contributes nothing numerical; the score comes from the 10 severity items alone.

VERIFY BEFORE THE EXAM Three syllabus instruments have no numbers in the sources, so none are printed: the Liebowitz social anxiety scale, the panic disorder severity scale and the dissociative experiences scale, whose cut-off is asked at nearly every table. Describe them: the social anxiety scale rates fear and avoidance separately across interaction and performance situations, the panic scale rates attack frequency, distress, anticipatory anxiety and agoraphobic avoidance, and the dissociation scale rates the proportion of time absorption, depersonalisation, derealisation and amnesia occur. As an interim proxy the GAD-7 at 10 gives sensitivity 72 percent and specificity 80 percent for social anxiety disorder, and 74 percent and 81 percent for panic disorder.

HOLD THIS

Ten items, 0 to 40, entry at 16, response at 25 percent, remission at 12 or less. The checklist finds the symptoms, the severity items score them.

WHAT EARNS THE MARKS

- **Separating the checklist from the scored items**: the total comes from the severity items alone
- **Giving the three decision points together**: entry, response and remission in one breath
- **Explaining why the autism version drops obsessions**: intrusive thoughts cannot be reliably reported
- **Declaring the unsourced cut-offs**: describe the social anxiety and dissociation scales instead

SEE ALSO 11 Mood scales 13 Substance use scales 17 Child and eating scales

13 · Substance use: screening, severity, withdrawal

RATING SCALES · TOPIC 13

This man scores 22 on the AUDIT. What follows?

VIVA TABLE: RATING SCALES; ALSO THE ADDICTION LONG CASE

THE SHAPE OF THE ANSWER

1 Sort by the question	Screening, severity and withdrawal are three different jobs
2 The screen	Ten items, 0 to 40, with the skip rule
3 Bands and actions	Low risk, hazardous, harmful, likely dependence, each with its action
4 Indian cut-offs	Local thresholds run lower, and give the four reasons
5 Severity and withdrawal	The dependence questionnaire, then the withdrawal scale that doses

Sort by the question first. Screening asks whether hazardous or harmful use is present, severity scales ask how entrenched dependence is and therefore set the treatment setting, and withdrawal scales quantify a state that changes hour by hour to titrate medication. A screening tool cannot dose a detoxification, and a withdrawal score cannot diagnose a use disorder.

The alcohol use disorders identification test is a World Health Organization 10-item instrument for primary care, usable as interview or self-report. Items 1 to 3 cover consumption, 4 to 6 dependence symptoms, 7 to 10 adverse consequences, and a two-factor structure of use and problems fits internationally.

Items 1 to 8 score 0 to 4 while items 9 and 10 score 0, 2 or 4, for a total of 0 to 40; if item 1 is 0, or items 2 and 3 sum to 0, the interviewer skips to items 9 and 10.

SCORE	CLASSIFICATION	ACTION	THE DISCRIMINATOR
0 to 7	Low risk	Alcohol education	**A score of 0 indicates an abstainer**
8 to 15	Hazardous drinking	Simple advice, brief intervention, monitoring	**8 or above is the standard international threshold, chosen for sensitivity**
16 to 19	Harmful drinking	Extended brief intervention, counselling	**10 or above where specificity matters in a high-prevalence clinic**
20 or above	Likely dependence	Diagnostic evaluation, detoxification, addiction referral	**Secondary sources calling 15 or above dependence conflict with the primary classification**

INDIAN AND LOW-INCOME-COUNTRY CUT-OFFS

A systematic review of cut-offs in low- and middle-income countries found 54 studies using other thresholds, India contributing the largest number at 10.

Where psychometric evaluation was done, **effective cut-offs diverged**: hazardous drinking from above 3 to above 5, harmful from above 5 to above 16, dependence from above 7 to above 24.

- **Reason one.** Local standard drink volumes, and home-brewed beverages of variable strength.
- **Reason two.** Lower drinking frequency with heavy episodic intoxication.
- **Reason three.** Item 10, concern expressed by others, scores positive in conservative settings at low intake.
- **Reason four.** Variation in alcohol and aldehyde dehydrogenase activity.

INSTRUMENT	STRUCTURE	BANDS	THE DISCRIMINATOR
SADQ	20 items in 5 sections, physical withdrawal, affective withdrawal, relief drinking, consumption and speed of reinstatement, each scored 0 to 3, rated on a typical heavy-drinking period in the past 6 months	Below 16 mild, 16 to 30 moderate, 31 or above severe	**16 or above is the decision point for a prescribed chlordiazepoxide regimen; mean corpuscular volume and gamma-glutamyl transferase do not track the score**
COWS	Wesson and Ling, 2003; 11 items, 6 objective signs, 1 subjective symptom, 4 mixed	0 to 4 none, 5 to 12 mild, 13 to 24 moderate, 25 to 36 moderately severe, above 36 severe	**Buprenorphine induction needs objective signs at 5 or above, because a high-affinity partial agonist displaces full agonists and precipitates withdrawal**

Table: severity and withdrawal. Inter-rater agreement on the opiate scale was moderate by category (67.5 percent, weighted kappa 0.55) and substantial at the action threshold (82.5 percent, kappa 0.65), best for pulse and worst for pupil size.

RULE **The screening scale says whether, the dependence scale says how much, the withdrawal scale says when to dose.** Each answer is wrong in another instrument's station.

VERIFY BEFORE THE EXAM The revised clinical institute withdrawal assessment for alcohol has no content in the sources: item count, total range, severity bands, the score at which symptom-triggered benzodiazepine dosing starts and the score at which it escalates are all absent, so none is printed. Take all five from the original validation paper or a current addiction text, since examiners pair it with the opiate scale. The item counts and cut-offs of CAGE, the Fagerstrom test, the substance involvement screening test, the drug abuse screening test and the Michigan alcoholism screening test are likewise not stated.

HOLD THIS

Eight or above screens positive, 20 or above suggests dependence, 16 or above buys a detoxification regimen, 5 or above buys buprenorphine.

WHAT EARNS THE MARKS

- **Matching the instrument to the question**: a screen cannot dose a detoxification
- **Quoting the Indian divergence with its reasons**: drink volume, episodic pattern, item 10, enzyme variation
- **Naming the action thresholds**: 16 or above buys a prescribed regimen, 5 or above buys induction
- **Declaring the withdrawal-scale gap**: the alcohol withdrawal instrument has no content in the sources

SEE ALSO 02 Accuracy and cut-offs 12 Anxiety and OCD scales 33 Addiction and liaison

14 · Movement and catatonia scales

RATING SCALES · TOPIC 14

Demonstrate the AIMS examination on this patient.

VIVA TABLE: EXAMINATION AND SCALES; OFTEN A BEDSIDE DEMONSTRATION

THE SHAPE OF THE ANSWER

1 The protocol	Two chairs, mouth cleared, observed at rest, then activated
2 The items	Seven regions rated 0 to 4, then the global items
3 The scoring rule	An activation-only movement scores one point lower than at rest
4 The criterion sets	Distribution and persistence rules, plus withdrawal-emergent dyskinesia
5 The neighbours	Parkinsonism, akathisia and catatonia, each with its own scale

The abnormal involuntary movement scale is a clinician-rated instrument from the United States National Institute of Mental Health taking about 10 minutes, with 12 items in the original and 14 in expanded variants.

Describing the protocol is half the marks: observe unobtrusively at rest before the room; two identical hard armless chairs facing each other; clear the mouth of gum and check dentures; observe seated with hands on knees then hanging unsupported, mouth open, tongue protruded.

Then activate: finger-to-thumb tapping, standing, arms extended palms down, walking, and where equivocal a cognitive load such as counting backwards from 100. Tone and gait are examined but not scored here.

Items 1 to 7 rate regions 0 to 4: muscles of facial expression, lips and perioral area, jaw, tongue, upper extremities, lower extremities, trunk. Item 8 rates global severity from the highest regional score, item 9 incapacitation, item 10 awareness and distress, and the remaining items record dental status and movements in sleep.

The dyskinesia score is the sum of items 1 to 7, and a 2-point reduction is the minimal clinically important difference.

RULE **A movement seen only on activation is scored 1 point lower than the same intensity present spontaneously at rest.** That scoring rule is the most commonly asked detail on this scale.

CRITERION SET	REQUIREMENT	THE DISCRIMINATOR
Schooler-Kane	At least 2 in 2 or more regions, or 3 or 4 in at least 1 region, with at least 3 months of cumulative antipsychotic exposure and other causes excluded	**A total of 7 spread as 1 across all 7 regions does not qualify while 3 in two regions does**
Glazer-Morgenstern	The scale applied at the start and end of each visit, with a total of 3 or more on both applications across 2 successive visits and at least 1 region at 2 or more on both	**Built for persistence, not a single cross-sectional rating**
Withdrawal-emergent dyskinesia	Movements appearing after stopping, substituting or reducing an antipsychotic	**Persisting beyond 8 weeks reclassifies it as tardive dyskinesia**

FEATURE	DRUG-INDUCED PARKINSONISM	TARDIVE DYSKINESIA
Movement	Rhythmic rest tremor at 3 to 6 Hz, rigidity, bradykinesia	**Arrhythmic, jerky choreic or slow athetoid movement**
Timing	Early after starting or increasing the dose	**Delayed, after at least 3 months of cumulative exposure**
Dose reduction, and anticholinergic challenge	Improves with dose reduction, and tremor and rigidity improve with an anticholinergic	**Unmasked or worsened by dose reduction, and dyskinesia worsens with an anticholinergic**

THE OTHER INSTRUMENTS, ONE LINE EACH

The Simpson-Angus scale is a 10-item clinician-rated measure of neuroleptic-induced rigidity and bradykinesia, scored 1 to 5 in the primary specification, with 1-item and 4-item short forms, weighted to upper-limb tone rather than tremor or subjective akathisia.

The Barnes akathisia rating scale rates objective motor restlessness, subjective awareness, subjective distress and global severity, which is how it separates akathisia from pseudoakathisia and from agitation; restless legs worsens at night and eases with movement, while psychotic agitation improves as the antipsychotic dose rises and akathisia worsens.

The Bush-Francis catatonia rating scale has a screening component and a full scale, and **the examination is the examinable part**.

- **Inspect.** Stupor, mutism, staring, grimacing and posturing.
- **Test tone.** Rigidity, waxy flexibility and catalepsy, then elicit Gegenhalten and Mitgehen.
- **Test response.** Automatic obedience, echolalia and echopraxia.
- **Monitor.** Pulse, blood pressure, sweating and temperature, for malignant catatonia.
- **Then.** A lorazepam challenge is both diagnostic and the first therapeutic step.

The unified Parkinson's disease rating scale is the standard in idiopathic disease, leans on historical recall, and gives no regional localisation and no separation of tardive chorea from dystonia or stereotypy.

MONITORING, AND WHAT YOU CAN PRESCRIBE

Screen at baseline and then every 6 months on a first-generation antipsychotic, every 3 months with risk factors such as older age, and every 12 months on a second-generation agent, every 6 months if high risk.

For established tardive dyskinesia the sources name valbenazine and deutetrabenazine, reversible vesicular monoamine transporter 2 inhibitors, as the agents with regulatory approval, and note that lowering or stopping the antipsychotic can unmask rather than relieve movements.

Whether either is obtainable in India, and what is used instead, is not in the sources: carry that from a current Indian formulary, because the follow-up question comes at once.

VERIFY BEFORE THE EXAM Not printed for want of sources: the item names and cut-offs of the Simpson-Angus scale; the item count, range and cut-off of the akathisia scale; the screening and full item counts, anchors and cut-off of the catatonia scale, and the lorazepam challenge dose; and the section and item counts of the Parkinson's disease scale. Clear mechanical artefacts first: edentulousness, ill-fitting dentures and gum mimic orofacial dyskinesia.

HOLD THIS

Sum items 1 to 7, drop a point for activation-only movements, and remember 2 regions at 2 or 1 region at 3 with 3 months of exposure meets Schooler-Kane.

WHAT EARNS THE MARKS

- **Describing the protocol before the numbers**: the examination is half the marks here
- **Giving the activation rule**: the most commonly asked detail on this scale
- **Separating parkinsonism from dyskinesia by anticholinergic response**: one improves, the other worsens
- **Clearing mechanical artefacts first**: dentures and gum mimic orofacial dyskinesia

SEE ALSO 10 Psychosis scales 32 Geriatric psychiatry 33 Addiction and liaison

15 · Cognitive screening and the Indian literacy problem

RATING SCALES · TOPIC 15

This woman has had no schooling. Will you use the MMSE?

VIVA TABLE: RATING SCALES; ALSO THE GERIATRIC LONG CASE

THE SHAPE OF THE ANSWER

1 The original	Six areas, 30 points, with its two cut-offs
2 The confound	Education, language of testing and affective state load the score
3 The Indian rebuild	Five substitutions removing reading, writing and spelling
4 The companion cut-offs	Fluency, naming, praxis and word-list figures
5 Choose by patient	Literate, illiterate, subtype separation, or staging by informant

The mini-mental state examination, formalised by Folstein and colleagues in 1975, grades 6 areas: orientation to time and place, attention and concentration, memory, language (naming, repetition, a three-stage command, reading, writing), visuospatial construction, and following written and verbal instruction. The maximum is 30 points and it takes 5 to 10 minutes.

An optimal clinical cut-off of 26 has been shown, with 25 or below indicating impairment; in community settings 25 or above reads as likely normal and below 24 warrants workup.

It is heavily confounded by education, language of testing and affective state: sociodemographic and affective variables accounted for 13 percent of baseline variance, a low baseline did not predict decline over 4 years, and in preclinical Alzheimer disease future cases had baselines equal to controls, a mean of 27.2 in both across 350 matched pairs, declining only in the final 3 years.

THE HINDI MENTAL STATE EXAMINATION

In the Indo-United States cross-national dementia study at Ballabgarh in Haryana, 28 villages 35 km from Delhi, only 21.7 percent of community elderly were literate, 81 of 374 non-demented subjects aged 55 years and above; illiteracy reached 94.8 percent in women and 64.0 percent in men, and 82.5 percent at 75 years and above.

Ganguli, Chandra and colleagues rebuilt the examination on **five substitutions**.

- **One.** Time of day, for calendar year.
- **Two.** A story requiring serial subtractions, for abstract serial sevens.
- **Three.** Days of the week backwards, for spelling a word backwards.
- **Four.** An oral description of the house, for writing a sentence.
- **Five.** Copying a diamond inside a square, for intersecting pentagons.

The ceiling stays at 30 points and the criterion is a total of 19 or less, the population 10th percentile, or scores at or below the 10th percentile on a memory test and at least one other domain.

Companion cut-offs run 4 for fruit fluency, 6 for animal fluency, 12 combined, 13 for object naming on 15 real objects, 3 for constructional praxis and 9 for word-list learning.

- **RULE** **Use category fluency, not letter fluency, where schooling is limited.** Phonemic segmentation is acquired through reading instruction, so initial-letter fluency fails for want of schooling rather than frontal pathology, while fruits and animals over 60 seconds each measure semantic retrieval independently of literacy.

Free drawing went the same way. Participants could not draw clocks, having no experience of analogue timepieces or writing implements, and substituting a face failed because women drew overlearned folk figures that could not be scored.

Copying survived only as circle, diamond and cross under liberal criteria, while a cube was perceived as an irregular hexagon and became an open box seen from above. Line drawings were replaced by 15 real models, and a 10-word list was printed on bright green paper so recall could be cued to it.

INSTRUMENT	USE IT WHEN	NUMBERS	THE DISCRIMINATOR
MMSE	The patient is literate and you need a fast bedside grade	Top score 30, cut-off 24 or 26, 5 to 10 minutes	**High literacy dependence, and poor sensitivity for mild cognitive impairment**
HMSE	The patient is illiterate or rural	Top score 30, cut-off 19 at the 10th percentile	**No reading, writing or spelling load anywhere in it**
ACE-III	You need dementia subtype separation	Total 100 across five domains; 88 or above normal, below 84 possible dementia; about 15 minutes	**Separates Alzheimer disease from frontotemporal degeneration, at the cost of heavy reading, writing and drawing load**
CDR	You need staging that survives illiteracy	0 denotes no dementia, 4 severe dementia	**Informant interview about everyday function rather than performance on artificial tasks, which is why it crossed into rural India unmodified**

Table: choosing the instrument. A highly educated patient with cognitive reserve can clear the mini-mental ceiling despite early degeneration.

VERIFY BEFORE THE EXAM The total score, mild cognitive impairment cut-off and education adjustment of the Montreal cognitive assessment are not in the sources, nor are the total and cut-off of the frontal assessment battery or the intermediate stages of the dementia rating scale. Describe their roles meanwhile: the Montreal assessment adds executive, visuospatial and abstraction load for mild impairment, and the frontal battery samples conceptualisation, mental flexibility, motor programming and inhibitory control by oral and motor command.

HOLD THIS

Thirty points either way, but the cut-off moves from 24 to 19 when literacy does. Where schooling is the confounder, stage function on the informant scale instead.

WHAT EARNS THE MARKS

- **Naming the five substitutions**: it shows you know why the Indian version exists
- **Using category fluency where schooling is limited**: phonemic fluency needs reading instruction
- **Moving the cut-off with literacy**: 30 points either way, and the threshold shifts
- **Staging on the informant scale**: it survives illiteracy because it rates everyday function

SEE ALSO 08 Neuropsychology 16 Function and disability 32 Geriatric psychiatry

16 · Function, disability and quality of life

RATING SCALES · TOPIC 16

Certify this patient's disability. Which scale, what score, what percentage?

VIVA TABLE: CERTIFICATION AND SCALES; A STANDING FORENSIC CROSSOVER

THE SHAPE OF THE ANSWER

1 **Eligibility first**	A confirmed diagnosis from the named set, and a minimum duration
2 **The four domains**	Self-care, interpersonal, communication and work, each 0 to 4
3 **The duration component**	Months symptomatic adds 1 to 4 and moves the percentage
4 **The global score**	Domain total plus duration, with the certification threshold
5 **Who signs**	A registered psychiatrist, a board carrying one, and the validity period

Neuropsychiatric conditions account for about 14 percent of global disability-adjusted life years lost, and an estimated 2.27 million people in India live with disability attributable to mental illness and intellectual conditions.

Mental illness was 1 of 7 recognised categories under the 1995 legislation, later aligned with the rights of persons with disabilities legislation of 2016.

The instrument to know cold is the Indian disability evaluation and assessment scale. A task force of the Indian Psychiatric Society developed it in 2001 and field-tested it at 8 centres on 1,078 patients; gazette notification adopted it in February 2002, with revisions in 2018 and 2024.

It is not a general screen. The patient must carry a confirmed diagnosis of 1 of 4 disorders, schizophrenia, bipolar disorder, dementia or obsessive-compulsive disorder, with the Government-modified version extended more widely, and total illness duration must be at least 2 years.

COMPONENT	CONTENT	RANGE	THE DISCRIMINATOR
Self-care	Bathing, grooming, toileting, dressing, eating, attending to physical health	0 to 4	**Every domain runs 0 for none to 4 for profound**
Interpersonal activities	Initiating and maintaining relationships, contextually appropriate communication	0 to 4	**Rated from informant interview with records and observation**
Communication and understanding	Comprehension and expression, spoken, written or non-verbal	0 to 4	**Commonly tracks negative symptom burden in schizophrenia**
Work	Rated in 1 of 3 spheres: employment, housework or education	0 to 4	**A student who stopped education because of the illness and cannot resume scores 4**
Duration component	Months symptomatic in the preceding 2 years: under 6 months adds 1, 7 to 12 adds 2, 13 to 18 adds 3, more than 18 adds 4	1 to 4	**Omitting it is the commonest scoring error and it changes the percentage**
Global disability score	Total domain score of 0 to 16 plus the duration score	0 to 20	**0 none, 1 to 7 under 40 percent, 8 to 13 moderate at 40 percent or more, 14 to 19 severe, 20 profound**

WHO SIGNS, AND FOR HOW LONG

Only a registered psychiatrist can make the diagnosis and sign the certificate. Trained clinical psychologists, psychiatric social workers or occupational therapists may administer the scale, working largely from informant interview with records and observation. The certificate is issued by a duly constituted government medical board, central or state, which must include at least 1 psychiatrist.

Temporary certificates run 5 years with reassessment stipulated every 2 years because the course fluctuates, and permanent certification is reserved for irreversible conditions.

- **The standing criticism.** The duration component carries 20 percent of the weight, so chronicity alone can carry a patient past the threshold with moderately preserved function.

INSTRUMENT	STRUCTURE	THE DISCRIMINATOR
GAF	A single-item clinician rating from 1 to 100 in decile anchors, with below 50 denoting serious impairment	**Dropped from DSM-5 because it conflates symptom severity with function and takes the lower of the two, tracks positive psychopathology rather than community functioning, and ignores impairment from medical or environmental causes**
WHODAS 2.0	Published 2012 after testing in 19 countries, grounded in the activity and participation dimensions of the classification of functioning; 6 domains over the preceding 30 days, cognition 6 items, mobility 5, self-care 4, getting along 5, life activities 8, participation 8	**Etiology-neutral, so it rates disability whatever the diagnosis**
WHODAS versions	A 36-item version of about 20 minutes and a 12-item screen of about 5 minutes accounting for 81 percent of the longer version's variance; simple summing of items scored 0 to 4, or weighting onto a 0 to 100 metric	**In schizophrenia raw self-ratings did not correlate with clinician ratings while corrected scores correlated at r 0.60, so anosognosia forces the interviewer-administered form**
SOFAS	Rates social and occupational functioning without regard to symptom severity, and impairment may stem from mental or general medical illness	**The Government-modified Indian scale showed strong concurrent validity with it and with the global assessment of functioning**
WHOQOL-BREF	26 self-report items over 2 weeks, 10 to 15 minutes, in more than 60 languages including 10 Indian ones; 4 domains, physical 7 items, psychological 6, social 3, environment 8, plus 2 general items; items scored 1 to 5 with domain means multiplied by 4 for transformed scores of 4 to 20	**Quality of life is subjective satisfaction, not measured deficit, so it answers a different question from any disability scale**

VERIFY BEFORE THE EXAM Three items to confirm from the statute and the portal rather than from this note: the statutory term for a person certified at not less than 40 percent of a specified disability, and the identity card and national portal through which certification is recorded, neither described in the sources; the exact enumeration of the seven categories under the 1995 Act, where the library corrects its own source and warns that multiple disabilities was not one of them and that blindness and low vision were separate; and the status of the social and occupational functioning scale in DSM-IV, recorded as a proposed alternative in the appendix rather than an adopted axis.

HOLD THIS

Four domains of 0 to 4 give 0 to 16, duration adds 1 to 4, and a global score of 8 out of 20 is the 40 percent certification threshold.

WHAT EARNS THE MARKS

- **Adding the duration component**: omitting it is the commonest scoring error on this scale
- **Naming the threshold arithmetic**: 8 out of 20 is the 40 percent gate
- **Separating disability from quality of life**: measured deficit against subjective satisfaction
- **Naming the standing criticism**: chronicity alone can carry a patient past the threshold

SEE ALSO 15 Cognitive screening 22 Disability Act 2016 27 Rehabilitation

17 · Child, adolescent and eating disorder scales

RATING SCALES · TOPIC 17

This toddler screens at 5 on the M-CHAT. What now?

VIVA TABLE: RATING SCALES; ALSO THE CHILD LONG CASE

THE SHAPE OF THE ANSWER

1 Group the instruments	Broad-band, narrow-band, developmental and adaptive
2 The toddler screen	Twenty items, three risk bands, and the reversed items
3 The attention-deficit forms	Parent and teacher forms, symptom count plus impairment
4 Indian certification	The mandated autism instrument, and the developmental charts
5 Eating screens	The attitudes test, and its three-armed referral index

Group the child instruments by breadth and informant: broad-band scales survey internalising and externalising domains at once, narrow-band scales target one cluster, and developmental and adaptive scales track milestones and real-world functioning. Screening instruments buy sensitivity with specificity and generate false positives by design, and none diagnoses anything without a clinical interview of caregiver and child.

INSTRUMENT	STRUCTURE	SCORING	THE DISCRIMINATOR
M-CHAT-R/F	Robins, Fein and Barton, 2009; parent report for toddlers aged 16 to 30 months, recommended at the 18 and 24 month visits; 20 questions scored in under 2 minutes, with a follow-up interview as the second stage	Total 0 to 20; 0 to 2 low risk, 3 to 7 medium, 8 to 20 high	**8 or above bypasses the follow-up and goes straight to diagnostic referral, while 3 to 7 mandates it, where 2 or more failed items is a positive screen**

INSTRUMENT	STRUCTURE	SCORING	THE DISCRIMINATOR
Reverse-scored items	For 17 items a No indicates risk; for exactly 3 items a Yes indicates risk	Items 2, 5 and 12	**Wondering whether the child might be deaf, unusual finger movements near the eyes, distress at everyday noises**
Vanderbilt scales	Wolraich; parallel parent and teacher forms for ages 6 to 12 years; parent form covers the past 6 months, teacher form since the school year began; symptoms scored 0 to 3, performance 1 to 5	Inattention items 1 to 9, hyperactivity and impulsivity items 10 to 18; a symptom counts at 2 or 3, impairment at 4 or 5	**A subtype needs 6 of 9 positive items in its segment plus documented impairment, and the combined type needs 6 of 9 in both**
Vanderbilt comorbidity screens	Parent: oppositional defiant items 19 to 26, conduct items 27 to 40, anxiety and depression items 41 to 47; teacher: oppositional and conduct items 19 to 28, anxiety and depression items 29 to 35	Positive at 4 of 8, 3 of 14, 3 of 7, 3 of 10 and 3 of 7 respectively, each with impairment	**The impairment rule differs by edition: the original accepts any single 4 or 5, the later guidance requires 4 on at least 2 items or 5 on at least 1**
SDQ	Goodman; parent and teacher forms for ages 3 to 17 years, self-report from 11 to 17; 25 items across 5 scales of 5, rated on 3 points; free for clinical use, in 77 languages	Total difficulties sums the 4 problem scales, 20 items	**The prosocial scale is a strength score and is not added to total difficulties**
SDQ dysregulation profile	A brief 5-item form (2 emotional, 2 conduct, 1 hyperactivity) and an extended 15-item form	Agrees with the checklist dysregulation profile in 81 percent of cases	**Indexes deficient self-regulation across diagnoses and predicts severe presentations and self-injury**
CBCL and Conners	Achenbach's checklist is broad-band, with a dysregulation profile from the anxious-depressed, withdrawn-depressed and aggressive behaviour scales; Conners' scales are narrow-band	Checklist at intake, Conners for focused work	**Broad-band for the first pass, narrow-band for stimulant titration and classroom monitoring**

AUTISM CERTIFICATION IN INDIA

The childhood autism rating scale is a clinician-completed observational scale with caregiver interview, useful for baseline characterisation and not recognised for official certification here.

The Indian scale for assessment of autism, from the national institute for the mentally handicapped, is the legally mandated tool for assessment and certification under the rights of persons with disabilities legislation, with the benchmark for entitlements at 40 percent.

Its domains cover social relationship and reciprocity, emotional responsiveness, speech, language and communication, behaviour patterns, sensory aspects and a cognitive component. The developmental screens in Indian use are the Trivandrum chart, the Baroda test and the Denver test, with suspected delay referred at once for paediatric and psychological testing.

The eating disorder screens belong here because presentation is usually adolescent.

The eating attitudes test in its 26-item form was factor-derived from the 40-item test of 1979, refined in 1982, and takes about 2 minutes.

Items are rated on 6 frequency points. Items 1 to 25 score 3 for the highest frequency down to 0 for the lowest three, with item 26 reversed, for a total of 0 to 78.

Its subscales are dieting (13 items), bulimia and food preoccupation, and oral control.

The referral index has three arms, and any one of them refers.

- **Arm one.** A total of 20 or above.
- **Arm two.** A body mass index below age and sex matched norms.
- **Arm three.** Behavioural symptoms over the preceding 6 months, meaning any of the following.
- **Binge.** Episodes with loss of control 2 to 3 times a month or more.
- **Purge.** Any self-induced vomiting, or any misuse of laxatives, diet pills or diuretics.
- **Exercise.** More than 60 minutes a day for weight.
- **Weight.** A loss of 20 pounds in the period.

RULE **A total below 20 does not clear the patient.** A low body mass index or one positive behavioural probe refers regardless of the questionnaire score, which is the point of a three-armed index.

TRAP Treating the attitudes test and the eating disorder examination questionnaire as interchangeable. In a community study of 1267 young adults, observed agreement was 0.666 with a kappa of 0.281: the 28-item examination questionnaire detected more disordered eating among women and people with overweight or obesity, while the attitudes test correlated more strongly with physical inactivity and can miss binge eating disorder where restriction is absent.

VERIFY BEFORE THE EXAM Not printed for want of sources: the four-tier severity ladder of the Indian autism scale, including the band below the mild cut-off, and its item count; the domain count, scoring range and autism cut-off of the childhood autism rating scale, and the forms of its current revision; the item counts and age bands of the developmental screening charts; and the item count of the five-question eating disorder acronym. Learn the autism ladder as four tiers, because beginning at mild leaves the classification looking incomplete.

HOLD THIS

Three to 7 on the toddler checklist means do the follow-up and 8 or above means refer now; 6 of 9 plus impairment makes an attention-deficit subtype; and 20 or above, a low body mass index, or one behavioural probe each refer an eating disorder.

WHAT EARNS THE MARKS

- **Acting on the band rather than the bare number**: medium risk mandates the follow-up interview
- **Naming the reversed items**: items 2, 5 and 12 are the ones candidates miss
- **Requiring impairment beside the symptom count**: six of nine plus documented impairment
- **Refusing to clear a low eating score**: a low body mass index or one behavioural probe refers

SEE ALSO 05 Intelligence testing 12 Anxiety and OCD scales 29 Child psychiatry

Fig 2 Plate to draw: one page of cut-offs by domain, each instrument a horizontal bar carrying its total range with the severity bands marked along it and the action threshold flagged, so psychosis, mood, anxiety, substance use, cognition and disability can be read against one another.

Forensic psychiatry and the law

The examiner listens for three things in order: the instrument, the provision, and the clinical act it licenses or forbids. Each topic is arranged so a patient turns into a section, and a section turns back into a patient.

18 · Mental Healthcare Act 2017: frame, capacity, advance directive, nominated representative

FORENSIC · TOPIC 18

Take me through capacity under the Mental Healthcare Act.

VIVA TABLE: LAW; THE ACT IS ASKED AT EVERY FORENSIC TABLE

THE SHAPE OF THE ANSWER

1 Name the instrument	The Act, its year, its scale, and what it repealed
2 The definition	Mental illness under Section 2(1)(s), and what sits outside it
3 Capacity, Section 4	Understand, appreciate, communicate: decision-specific and time-specific
4 The two devices	Advance directive and nominated representative, with their chapters
5 The civil-law hinge	Section 3(5): mental illness alone is not unsoundness of mind

Name the instrument before the section. Mental Healthcare Act, 2017, "Healthcare" one word: Act 10 of 2017, assented 7 April 2017, 126 sections, commenced 29 May 2018, repealing the Mental Health Act 1987 and harmonising Indian law with the Convention on the Rights of Persons with Disabilities.

Section 2(1)(s) defines mental illness as a substantial disorder of thinking, mood, perception, orientation or memory that grossly impairs judgment, behaviour, capacity to recognise reality or

ability to meet the ordinary demands of life, with mental conditions associated with the abuse of alcohol and drugs, but not mental retardation.

Capacity, Section 4. Capacity exists if the person can understand the relevant information, appreciate the reasonably foreseeable consequences of a decision or of the lack of one, and communicate the decision by speech, expression, gesture or other means. Capacity is decision-specific and time-specific and is reassessed as the state changes.

RULE Section 3(5) is the hinge between clinic and civil law: determination of mental illness alone does not mean the person is of unsound mind unless a competent court has declared it, and past treatment does not justify a present determination.

DEVICE	SECTIONS	WHAT IT DOES	THE DISCRIMINATOR
Advance directive, Chapter III	5 to 13	How the person wishes, or does not wish, to be treated; preferred establishment	**Operates only while capacity is absent; a capable decision overrides it, and Section 9 excludes emergency treatment**
Overriding a directive	11 and 13	Professional, relative or care-giver applies to the Board, which may uphold, modify, alter or cancel after hearing all concerned	**No unilateral override; Section 13 protects the practitioner who follows a valid directive**
Nominated representative, Chapter IV	14 to 17	Support for treatment decisions, admission applications, discharge planning, research consent	**Supported, not substitute, decision-making; appointing one, or being unable to, is not evidence of incapacity**
Rights, Chapter V	18 to 28	Access, community living, dignity, information and records, confidentiality, free legal aid, insurance parity	**Section 18 is justiciable: Government services as a minimum in each district, free for those below the poverty line and the destitute**

Who may be the nominated representative. Any person not a minor may appoint one, in writing on plain paper with signature or thumb impression. Section 14(3) sets three positive conditions.

- **Condition one.** Not a minor.
- **Condition two.** Competent to discharge the duties.
- **Condition three.** Written consent given to the mental health professional.

Failing appointment, Section 14(4) gives the order of precedence: the person named in the directive under Section 5(1)(c), a relative, a care-giver, a suitable person appointed by the Board,

and last the Director, Department of Social Welfare. For a minor the legal guardian acts unless the Board orders otherwise.

TRAP Asked who is *disqualified*, candidates improvise a list. The Act carries none. Give the three conditions in Section 14(3), then the Board's power to displace under Sections 14(7), 15(2) and 16. The express disqualification list belongs to Board members, under Section 74(2).

HOW THE LAW ARRIVED HERE

Indian Lunacy Act 1912: a custodial statute of 101 sections built on asylum reception, wide magistrate power and management of the patient's property, its only review a monthly visit by the asylum's visitors.

Mental Health Act 1987: drafted in 1950, 37 years to assent, in force across all States only by 1993, medicalising care but keeping magistrate reception orders and silent on rehabilitation and aftercare.

On 6 August 2001 a fire at an unlicensed private mental home at Erwadi killed 28 of the 45 residents chained inside a shed, 5 others badly burnt; the Ramdas Commission recommended unchaining, mandatory licensing and inspection, and upgrading of government hospitals. Ratification of the Convention in 2007 produced the disability Act of 2016 and this Act.

HOLD THIS

Capacity is functional, decision-specific and presumed. Mental illness alone is not unsoundness of mind: that takes a court, under Section 3(5).

WHAT EARNS THE MARKS

- **Naming the instrument before the section**: "Healthcare" as one word, with the year
- **Giving capacity as three functional limbs**: not a diagnosis and not a global status
- **Reaching for Section 3(5)**: unsoundness of mind takes a court declaration
- **Answering the disqualification question correctly**: the Act carries no list for the representative

SEE ALSO 19 Admissions 22 Disability Act 2016 25 Civil capacities

19 · Admissions, discharge and emergency treatment

FORENSIC · TOPIC 19

The brother wants this man admitted tonight. Under which section?

VIVA TABLE: LAW; ALSO THE DISPOSAL QUESTION IN THE LONG CASE

THE SHAPE OF THE ANSWER

1 **The vocabulary change**	Independent and supported replaced voluntary and involuntary
2 **The routes**	Sections 86, 87, 89, 90 and 94, with their examiners
3 **The thresholds**	A risk limb, absent capacity, and the least restrictive option
4 **The clocks**	Reporting, capacity review and Board disposal for each route
5 **Exit and leave**	Discharge on request, the holding limit, leave and absence

Chapter XII replaced voluntary and involuntary with independent and supported. No judicial reception order opens a clinical admission; the judiciary enters afterwards, through the Board.

ROUTE	APPLICANT	EXAMINERS	CEILING	BOARD AND REVIEW CLOCK
Independent, Section 86	The person, not a minor	1 medical officer or mental health professional in charge	Until discharge is requested under Section 88	**Intimation within 72 hours for women and minors, 7 days for other adults**
Minor, Section 87	Nominated representative	2 independently, on the day of admission or in the preceding 7 days: 2 psychiatrists, or 1 psychiatrist with 1 professional or practitioner	Until discharge criteria are met	**Reported within 72 hours, Board review within 7 days, internal review at least every 7 days, re-report at 30 days**
Supported, Section 89	Nominated representative	1 psychiatrist plus 1 other, a mental health professional or medical practitioner	30 days, no renewal under the same section	**Reported within 3 days for a woman or minor, 7 days otherwise; capacity every 7 days; Board findings within 7 days**
Beyond thirty days, Section 90	Nominated representative	2 psychiatrists, both in the preceding 7 days	90 days first, then 120 days, then 180 days each time	**Reported within 7 days; Board permits or discharges within 21 days; capacity every 15 days**
Emergency, Section 94	Any registered medical practitioner	Consent of the nominated representative where available	72 hours or until assessment, whichever is earlier; up to 7 days in a declared disaster	**Electroconvulsive therapy is barred here, and an advance directive does not apply**

The Section 89 thresholds. Severity such that one of three limbs is met.

- **Limb one.** The person has recently threatened or attempted, or is threatening or attempting, bodily harm to himself.
- **Limb two.** He has recently behaved or is behaving violently towards another, or has caused another to fear bodily harm.
- **Limb three.** He has recently shown inability to care for himself to a degree that places him at risk.

Both examiners certify, having taken any directive into account, that admission is the least restrictive option and that the person cannot decide independently and needs very high support.

Section 90 raises the same limbs to conduct shown *consistently over time*. Treatment follows the directive or consent given with the representative's support; where support approaching the whole decision is needed the representative may consent temporarily, and capacity is reviewed every 7 days.

An independent patient is entitled to discharge on request under Section 88, and may be held only up to 24 hours to complete a Section 89 assessment. Section 91 governs leave; no section is headed absconding, Section 92 covering absence without leave or discharge for a person to whom Section 103 applies.

THREE TRAPS IN ONE TABLE

Rolling admissions: Section 89 caps at 30 days and cannot be renewed; continuation goes to Section 90 with two psychiatrists. **Readmission bar:** a person discharged under Section 89 cannot return under it within 7 days, so an early relapse goes straight to Section 90. **Emergency ECT:** Section 94(3) bars it as emergency treatment, whatever the catatonia.

STATION: THE RELATIVE WHO WANTS AN ADMISSION

Synthetic case. A man in his thirties has not slept for four nights, has struck his brother twice, and refuses to come in. The ladder:

1. Is he a minor, and has he capacity under Section 4 to request admission himself?
2. With capacity, this is Section 86, and the brother's consent is not required.
3. Without capacity, and with one risk limb met: who is the nominated representative, and is there a directive?
4. Which two professionals examine him independently today, and is one a psychiatrist?
5. What is the least restrictive option that meets the risk, and why is admission it?
6. What is the reporting clock, and when is capacity next reviewed?
7. If violence is imminent tonight and no representative is reachable, what does Section 94 allow, and for how long?

HOLD THIS

Section 89 is one psychiatrist plus one other for thirty days; Section 90 is two psychiatrists for ninety in the first instance. Risk plus absent capacity opens the door, not diagnosis.

WHAT EARNS THE MARKS

- **Opening the door with risk and capacity**: diagnosis alone admits nobody
- **Keeping the two ceilings apart**: 30 days under one section, 90 in the first instance under the next
- **Knowing the readmission bar**: an early relapse goes to the higher section
- **Barring emergency electroconvulsive therapy**: Section 94(3), whatever the catatonia

SEE ALSO 18 The Act 2017 frame 20 Treatment rules 21 Institutions

20 · Treatment rules, restraint, research, and the presumption of severe stress

FORENSIC · TOPIC 20

What does the Act prohibit outright, and what does it merely restrict?

VIVA TABLE: LAW; THE RESTRAINT QUESTION RECURS IN THE WARD VIVA

THE SHAPE OF THE ANSWER

1 **The four prohibitions**	Section 95, given in order
2 **The restrictions**	Minors and psychosurgery, each with its authorising set
3 **Restraint and seclusion**	Seclusion barred; restraint only as the sole means against imminent harm
4 **The entry**	Method, nature, justification and duration, recorded at once
5 **Section 115**	The rebuttable presumption of severe stress after an attempt

Section 95 is four prohibitions, one conditional. These are not to be performed on a person with mental illness.

- **One.** Electroconvulsive therapy without muscle relaxants and anaesthesia.
- **Two.** Electroconvulsive therapy for minors, which is the conditional one.
- **Three.** Sterilisation intended as treatment for mental illness.
- **Four.** Chaining in any manner or form whatsoever.

PROCEDURE	SECTION	STATUS	WHO AUTHORISES
Unmodified electro-convulsive therapy	95(1)(a)	Prohibited	**Nobody: a statutory bar. Modified treatment stays a valid modality**
Electroconvulsive therapy in a minor	95(1)(b) with 95(2)	Restricted, not absolute	**Three together: opinion of the psychiatrist in charge, informed consent of the guardian, prior permission of the Board**
Psychosurgery	96(1)	Restricted	**The person's own free and informed consent plus Board approval; proxy consent cannot reach it**
Sterilisation as treatment, and chaining	95(1)(c) and 95(1)(d)	Prohibited	**Statutory bars, and chaining is the provision Erwadi is remembered by**
Seclusion or solitary confinement	97(1)	Prohibited	**Statutory bar: a locked side room is not an option**
Physical restraint	97(2)	Restricted	**The psychiatrist in charge, only as the sole means against imminent harm, representative informed within 24 hours**

The restraint entry and research. Section 97 requires method, nature, justification and duration recorded immediately in the notes; no longer than absolutely necessary; not as punishment or deterrent and not for shortage of staff; every instance in the monthly report to the Board. The section names seclusion and physical restraint only, not chemical restraint, and sets no maximum duration.

Section 99: research needs free and informed consent; where capacity is absent and the person does not resist, permission comes from the State Authority, which may approve only with the representative's consent and on five conditions, among them that the research cannot be done on persons with capacity and that no financial or professional conflict of interest exists.

Section 115. Notwithstanding anything in Section 309 of the Indian Penal Code, a person who attempts suicide shall be presumed, unless proved otherwise, to have severe stress and shall not be tried and punished under that Code; Section 115(2) puts the Government under a duty to provide care, treatment and rehabilitation to reduce recurrence.

The Bombay High Court in Shital Dinkar Bhagat v. State of Maharashtra (2024) held that the presumption operates at the threshold and precludes putting the person on trial.

Under the Bharatiya Nyaya Sanhita, 2023 an attempt is not a general offence, the exception being Section 224, an attempt intended to compel or restrain a public servant from discharging duty.

TRAP "The Act repealed Section 309." It did not: Section 115 created a rebuttable presumption that disabled its operation, and "unless proved otherwise" leaves the prosecution room. The Act also leaves severe stress undefined and prescribes no scale or cut-off for it.

Police and magistrate, Chapter XIII. Section 100(1): the officer in charge shall take under protection a person found wandering at large within station limits, believed to have mental illness and to be incapable of self-care, or to be a risk to himself or others by reason of mental illness.

- **Grounds.** Told to the person, or to the representative.
- **Key number.** He goes to the nearest public health establishment not later than 24 hours.
- **Trap.** Under Section 100(4) no person taken into protection shall be detained in a police lock-up or prison in any circumstances.

Section 101: report forthwith to the Magistrate anyone believed ill-treated or neglected. Section 102: the Magistrate may order conveyance for assessment or authorise admission for a period not exceeding 10 days.

Section 103: prisoners, with a quarterly certificate from the prison medical officer to the Board, a special report every 6 months, and an establishment in the medical wing of at least 1 prison per State and union territory.

HOLD THIS

Four prohibitions in Section 95; seclusion barred by Section 97(1); restraint only as the sole means against imminent harm. Section 100 gives twenty-four hours and forbids the lock-up.

WHAT EARNS THE MARKS

- **Separating a bar from a restriction**: nobody can authorise a prohibited procedure
- **Giving the three conditions together**: opinion, guardian consent, prior Board permission
- **Recording restraint as the section requires**: the entry is the defensible part
- **Correcting the repeal claim**: the presumption disabled the old section rather than repealing it

SEE ALSO 19 Admissions 21 Institutions 25 Civil capacities

21 · Institutions, registration, penalties, and the Act in practice

FORENSIC · TOPIC 21

Who registers a nursing home, and who hears an appeal against an admission?

VIVA TABLE: LAW; THE ADMINISTRATIVE QUESTION IN THE FORENSIC STATION

THE SHAPE OF THE ANSWER

1	Three bodies	Central Authority, State Authority, and the district Board
2	Board composition	Six members: the chair, the medical pair, the lived-experience pair
3	Board powers	

	Directives, representatives, admission challenges, records, deficiency of care
4 The clocks	Seven, twenty-one and ninety days, and thirty to the High Court
5 Registration and penalties	Registration under Section 65, then the graded fines

Two authorities regulate, one board adjudicates. Central Authority: Chapter VII, Sections 33 to 44. State Authority: Chapter VIII, Sections 45 to 56, to be established within 9 months of assent; under Section 55(1) it registers establishments, sets norms, supervises, and registers clinical psychologists, mental health nurses and psychiatric social workers as mental health professionals.

Boards: Chapter XI, Sections 73 to 84, constituted by the State Authority for a district or group of districts.

The Board. Section 74(1) gives it 6 members, in four kinds.

- **Chair.** A District Judge, an officer of the State judicial services qualified to be appointed District Judge, or a retired District Judge.
- **Administration.** A representative of the District Collector, District Magistrate or Deputy Commissioner.
- **Medical pair.** Two members, one a psychiatrist and one a medical practitioner.
- **Lived experience.** Two members who are persons with mental illness, care-givers or their representatives.

Disqualifications in Section 74(2), tenure 5 years or the age of 70 whichever is earlier, quorum 3 under Section 76, proceedings judicial under Section 78, applications free of fee under Section 77 and exceptionally oral or by telephone.

Section 82 powers: the advance directive; appointment of a representative; applications against admissions under Sections 87, 89 and 90; non-disclosure of records under Section 25(3); deficiency of care under Section 28; inspection of prisons; and a penalty extending to 5 lakh rupees on a rights violation.

APPLICATION	PROVISION	DISPOSAL LIMIT
Nominated representative; a minor's admission; a supported admission	14(4)(d), 87, 89(10) and 89(11)	**Within 7 days**
Extended supported admission	Section 90	**Within 21 days**
General applications and complaints	Section 80	**Within 90 days**
Written communication of the decision	Section 80(13)	**Within 5 days of the hearing closing**
Appeal to the High Court	Section 83	**Within 30 days**

Registration, penalties, workforce. No establishment runs without registration under Section 65; one registered under the Clinical Establishments Act files a copy with an undertaking on minimum standards.

Section 107: not less than 5,000 and up to 50,000 rupees for a first contravention, 50,000 to 2 lakh for a second, 2 lakh to 5 lakh for each subsequent one, recoverable as arrears of land revenue.

Section 108: up to 6 months or a fine up to 10,000 rupees or both, then up to 2 years or 50,000 to 5 lakh rupees.

The Act cannot be staffed as written: about 0.3 psychiatrists, 0.12 mental health nurses and 0.07 clinical psychologists per 100,000 population against a desirable figure above 3 per 100,000, with a treatment gap of 83%; a 6-member Board is hard to constitute in every district and, with a quorum of 3, can decide a clinical detention with no psychiatrist present.

RULE The Authority registers and regulates; the Board adjudicates. Directives, representatives and admission appeals belong to the Board; registration of establishments and professionals to the Authority. Swapping them loses the mark.

HOLD THIS

Six members, quorum of three, seven days for a supported-admission challenge, twenty-one for an extended one, thirty days to the High Court. The quorum is the flaw worth naming.

WHAT EARNS THE MARKS

- **Keeping the division straight**: the Authority registers and regulates, the Board adjudicates
- **Giving the disposal clock with the application**: each route carries its own limit
- **Naming the quorum flaw**: three members can decide a detention with no psychiatrist present
- **Attaching the workforce figures**: the Act cannot be staffed as written

SEE ALSO 18 The Act 2017 frame 19 Admissions 26 Programmes and policy

22 · Disability: the Rights of Persons with Disabilities Act 2016 and certification

FORENSIC · TOPIC 22

This patient wants a disability certificate. Which Act, and what percentage?

VIVA TABLE: LAW AND CERTIFICATION; CROSSES INTO THE SCALES TABLE

THE SHAPE OF THE ANSWER

1 Two Acts, two jobs	One governs entitlement, the other governs treatment
2 The structure	Sections, chapters, one Schedule, and 21 specified disabilities
3 Benchmark disability	Not less than 40 percent, as certified by the authority
4 The entitlements	Employment and education quotas, social security, grievance routes
5 Guardianship and assessment	Limited guardianship, and when to assess for permanence

One Act for entitlement, another for treatment. The 2016 Act replaced the Persons with Disabilities Act 1995, moving from charity and welfare to rights and social barriers.

It runs to 102 sections across 17 chapters with one Schedule, and recognises 21 specified disabilities where the 1995 Act recognised 7; mental illness and intellectual disability are

separate Schedule entries, intellectual disability there including specific learning disabilities and autism spectrum disorder.

A person with benchmark disability has not less than 40% of a specified disability where that disability is not defined in measurable terms, as certified by the certifying authority, which is the gate to reservation and welfare.

Guidelines are notified under Section 56, certifying authorities designated under Section 57, and Section 58 carries the procedure, the certificate being valid across the country.

ENTITLEMENT	PROVISION	WHAT IT GIVES	DISCRIMINATOR
Public employment	Section 34(1)	Not less than 4% of vacancies in each group of posts	**Mental illness sits in clause (d) with autism, intellectual disability and learning disability, and (d) shares 1% with (e), while (a), (b) and (c) carry 1% each**
Higher education	Section 32	Not less than 5% of seats	**With an upper age relaxation of 5 years**
Social security	Section 24	Schemes at least 25% higher than comparable ones: disability pension, unemployment and care-giver allowance, free aids and medicines on an income ceiling	**Addressed to persons with disabilities generally, not only benchmark disability**
Workplace grievance	Section 23	A Grievance Redressal Officer, inquiry within two weeks	**Then the District-Level Committee under Section 72**
Discrimination or non-implementation	Sections 74 to 83	Inquiry by the State or Chief Commissioner, the recipient of a recommendation bound to act within three months	**The route for refused accommodation or denied admission, and not the Section 14(3) appeal**

Limited guardianship and assessment. Under Section 14, a District Court or State-notified authority may provide a limited guardian where a person with disability, despite adequate and appropriate support, remains unable to take legally binding decisions.

- **How it works.** The guardian decides in consultation with him.
- **Its bounds.** Confined to a specific period, decision or situation.
- **Its standard.** Following the person's will and preference, with total support only where required, and then reviewed periodically.

Every guardian appointed under earlier law is deemed limited, and appeal lies under Section 14(3). For mental illness the notified instrument is the Indian Disability Evaluation and

Assessment Scale, with certificates issuable from primary health centre level upwards. Global scores are expressed as mild, moderate, severe and profound, as notified in the Gazette.

Assess when further treatment is unlikely to reduce the impairment, a period of 6 months normally being considered; where improvement remains possible even after 6 to 12 months a temporary certificate may be issued, valid for 5 years; a permanent certificate is lifelong. A certificate is not to be denied for want of psychiatric intervention.

RULE Three grievances, three doors. A refused *certificate*: the Section 59 appellate authority. A *guardianship* order: the Section 14(3) appellate authority. *Discrimination or non-implementation*: the State or Chief Commissioner, or the district committee. And a limited guardian is not a nominated representative: the first is for legally binding civil decisions, the second for mental healthcare.

VERIFY BEFORE THE EXAM The examiner may want the four domains of the disability scale, the ordinal range of each, the duration-of-illness score, the arithmetic converting a global score into a statutory percentage, and how bipolar disorder is scored differently. The library marks all of it unverified, so none is printed here. Read it off the notified Gazette schedule and the scale itself; at the table, give the Gazette's category language and say the conversion is read off the notified table.

HOLD THIS

Forty per cent of a specified disability is benchmark; four per cent of posts and five per cent of higher-education seats follow. Mental illness shares its one per cent with the rest of clause (d) and with clause (e).

WHAT EARNS THE MARKS

- **Naming the benchmark correctly**: not less than 40 percent of a specified disability
- **Getting the quota arithmetic right**: mental illness shares its one percent inside its clause
- **Sending each grievance to the right door**: certificate, guardianship and discrimination differ
- **Separating a limited guardian from a nominated representative**: civil decisions against mental healthcare

SEE ALSO 16 Function and disability 18 The Act 2017 frame 25 Civil capacities

23 · Criminal responsibility, fitness to stand trial, and the psychiatrist as witness

FORENSIC · TOPIC 23

Was he responsible at the time, and is he fit to face trial now?

VIVA TABLE: LAW; THE COMMONEST FORENSIC VIGNETTE

THE SHAPE OF THE ANSWER

1	**Two questions, two moments**	Responsibility at the act, fitness in the courtroom now
2	**The provision**	Section 84, carried into the new code, and its three ingredients
3	**The burden**	Prosecution beyond reasonable doubt, the accused on the balance

4 The cases	Three judgments, and the conduct each one weighed
5 Fitness procedure	Examination, expert report, judicial finding, disposal and appeal

Two questions, two moments. Responsibility asks about the mind at the act; fitness asks about the mind in the courtroom now.

Section 84 of the Indian Penal Code is carried into the Bharatiya Nyaya Sanhita, 2023 as Section 22 in Chapter III, without substantive change.

It provides that **nothing is an offence which is done by a person who, at the time of doing it, by reason of unsoundness of mind, is incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law.**

- **Ingredient one.** Unsoundness of mind.
- **Ingredient two.** Present at the time of the act.
- **Ingredient three.** Producing cognitive incapacity of that magnitude.

The McNaughten rules give a presumption of sanity until the contrary is proved, a defect of reason from disease of the mind such as not to know the nature and quality of the act or that it was wrong, and a test applied to the very act committed.

Burden: the prosecution proves the offence beyond reasonable doubt, while under Section 105 of the Indian Evidence Act the accused proves the exception on the preponderance of probabilities.

CASE	CONDUCT WEIGHED	OUTCOME
Dayabhai Chhaganbhai Thakkar (1964)	44 knife injuries to his wife; statements to police showing no cognitive loss	**Rejected, and the rule set: weigh the circumstances preceding, attending and following, the crucial time being the moment of commission**
Ratanlal (1965)	Set fire to grass, said openly "I burnt it; do whatever you want", no concealment, manic depressive psychosis	**Acquitted: incapable of knowing what he was doing**
Srikant Anandrao Bhosale	Paranoid schizophrenia with family history, hospital 25 times in the preceding months, killed his wife in daylight without hiding	**Conviction set aside: cognitive impairment established**

TRAP Three near-misses. *Brutality* is not evidence of insanity: a crime is not excused by its own atrocity. *Absence of motive* is corroborative only. *Irresistible impulse* is no defence here, and neither is diminished responsibility, the English partial defence from R v. Byrne (1960), which the Law Commission of India rejected in its 42nd Report: the section tests cognition, not volition.

Automatism and intoxication. Insane automatism from an internal disease of the mind, such as a psychomotor seizure, falls within the exemption and yields a special verdict with safe custody;

non-insane automatism from an external factor, such as concussion, negates the voluntary act and yields an unqualified acquittal.

Substance use that has produced established brain disease is settled insanity, judged under the exemption; involuntary intoxication is a complete defence on the same standard, while a voluntarily intoxicated person is dealt with as if he had the knowledge he would have had sober, as Basdev v. State of Pepsu (1956) sets out.

ELEMENT	CRIMINAL RESPONSIBILITY	FITNESS TO STAND TRIAL
Provision	Section 84 of the Penal Code, now Section 22 of the Bharatiya Nyaya Sanhita, 2023	**Sections 328 and 329 of the Code of Criminal Procedure, custody and bail under Section 330**
Focus	Retrospective, at the moment of the act	**Present, at the inquiry or trial**
Question	Did he know the nature of the act, or that it was wrong or contrary to law?	**Can he follow proceedings, instruct counsel, enter a rational defence?**
Stage	Raised by the accused, who carries the burden under Section 105 of the Evidence Act	**Section 328 at the inquiry, before charges; Section 329 once charges are framed; the court's own duty**
Disposal	Acquittal under Section 334, safe custody or delivery to a relative under Section 335	**Discharge where no prima facie case exists, else postponement for treatment, bail considered under Section 330(1)**
Appeal on the report	Not applicable	**A Medical Board of 2: head of psychiatry at the nearest government hospital, and a psychiatry faculty member at the nearest medical college**

The fitness sequence. Examination by the Civil Surgeon or a designated medical officer on oath; referral to a psychiatrist or clinical psychologist reporting unsoundness of mind or mental retardation; then a judicial finding on incapacity to enter a defence, with inpatient observation typically 1 to 6 weeks.

Mental retardation diverges: the inquiry closes under Section 328(4), and at trial the court shall not hold the trial under Section 329(3), proceeding under Section 330(3) to a residential facility.

Before 2009 postponement could be indefinite: in Veena Sethi (1982) undertrials were found detained from 19 to 37 years, and in 2007 Machal Lalung was found to have spent 38 years as an undertrial.

The 154th Report of the Law Commission held that **treatability and not dangerousness must govern postponement**.

Kaliyappan v. State (2020) held that an act covered by the exemption is not an offence at all, so counsel may place material of sterling quality before a court acting in parens patriae under Section 329(2).

Under Section 45 of the Evidence Act the expert opinion is relevant and advisory: fitness is a judicial finding.

VERIFY BEFORE THE EXAM The Code of Criminal Procedure is recodified as the Bharatiya Nagarik Suraksha Sanhita, 2023, and the examiner may want the successor numbering of the fitness provisions and of Section 105 of the Evidence Act. That mapping is unsourced in the library, so no number is printed. Cite the Code section, name the recodification, read the concordance off the bare Sanhita.

HOLD THIS

Responsibility is cognition at the moment of the act; fitness is capacity in the courtroom now. India has neither diminished responsibility nor irresistible impulse; the expert advises, the court decides.

WHAT EARNS THE MARKS

- **Holding the two moments apart**: retrospective cognition against present courtroom capacity
- **Naming the three near-misses**: brutality, absent motive, irresistible impulse
- **Stating the standard for the exception**: preponderance of probabilities, carried by the accused
- **Leaving the finding to the court**: the expert opinion is relevant and advisory

SEE ALSO 20 Treatment rules 24 POCSO, Juvenile Justice, NDPS 25 Civil capacities

24 · Children and drugs: POCSO, Juvenile Justice, NDPS

FORENSIC · TOPIC 24

A fifteen-year-old discloses abuse and the family asks you not to report. What do you do?

VIVA TABLE: LAW; ALSO THE CHILD AND ADOLESCENT LONG CASE

THE SHAPE OF THE ANSWER

1	**Three statutes, three roles**	Reporter, assessor, and treater holding a statutory shield
2	**POCSO duties**	Reasonable suspicion, immediate report, good-faith immunity, examination rules
3	**The refusal**	Work the protocol, then weigh ongoing risk and the environment
4	**Juvenile Justice**	Sixteen years with a heinous offence opens the preliminary assessment
5	**NDPS**	Consumption, the immunity in Section 64A, and substitution therapy

Three statutes, one clinical reflex each. POCSO makes you a reporter, the Juvenile Justice Act an assessor, the NDPS Act a treater holding a statutory shield for the patient.

Under POCSO 2012, amended 2019, a child is any person below 18 years, the Act is gender-neutral, and the age of consent is 18 years, so a consensual adolescent relationship falls

technically within the offences.

OFFENCE	SECTION	PUNISHMENT AFTER 2019	DISCRIMINATOR
Penetrative sexual assault	3 and 4	Minimum 10 years, raised from 7; minimum 20 years if the child is below 16 years	**The child's age sets the floor**
Aggravated penetrative sexual assault	5 and 6	Minimum 20 years rigorous imprisonment, raised from 10, to life for the remainder of natural life, or death	**Aggravation is position of trust or authority, gang assault, injury, pregnancy, HIV, death, or a child below 12 years**
Sexual assault, and aggravated sexual assault	7, 8, 9 and 10	Minimum 3 years up to 5; aggravated, minimum 5 up to 7 and fine	**2019 added drugs, hormones or chemicals to induce early sexual maturity**
Use of a child for pornography, and storage	13, 14 and 15	Minimum 5 years, 7 on repetition; storage fines of 5000 and 10000 rupees, up to 3 years for storage for transmission, 3 to 5 years commercial	**Section 2(1)(da) of 2019 covers computer-generated images indistinguishable from a real child**
Failure to report	21 with 19	Up to 6 months, or fine, or both; up to 1 year for the head of an institution whose subordinate offended	**Section 21(3) exempts the child**
Disclosing identity	23	Between 6 months and 1 year, or fine, or both	**Name, address, photograph, family and school are all identity**

The clinician's duties. Section 19(1): any person with knowledge or an apprehension that an offence has been or is likely to be committed informs the Special Juvenile Police Unit or local police immediately. The threshold is reasonable suspicion, not proof, and reporting to a hospital committee does not discharge the duty.

Section 19(7) gives immunity for a report made in good faith. Section 27: examination proceeds although no First Information Report exists, by a woman doctor where the victim is a girl child, before a parent or trusted person, with free emergency care within 24 hours.

Sections 29 and 30 presume commission and culpable mental state, which the accused must displace beyond reasonable doubt. Section 37 makes the trial in camera, and Section 35 requires the child's evidence within 30 days of cognizance and the trial to finish, as far as possible, within 1 year.

When a family refuses reporting, work the seven steps of the structured protocol, then the two decision parameters: ongoing risk of re-abuse, and whether the environment can be changed. With the alleged perpetrator in the household, reporting cannot be withheld.

Juvenile Justice Act 2015. A child in conflict with law has not completed 18 years on the date of commission.

Tiers by sentence: petty, up to 3 years; serious, minimum above 3 and up to 7 years, or maximum above 7 with no minimum or a minimum below 7, a limb added in 2021 after Shilpa Mittal; heinous, minimum of 7 years or more.

Only a heinous offence alleged against a child who has completed 16 years triggers the preliminary assessment under Section 15, **an inquiry and not a trial**, into four things.

- **One.** Mental capacity to commit the offence.
- **Two.** Physical capacity to commit it.
- **Three.** Ability to understand the consequences.
- **Four.** The circumstances of commission.

The psychiatrist enters by the proviso to Section 15(1): in Barun Chandra Thakur v. Bholu the permissive "may" was held to operate as mandatory unless the Board itself has a practising professional with a degree in child psychology or child psychiatry.

The examinable distinction is cognitive knowledge against emotional competence, since an adolescent of average intelligence knows the rule while immaturity leaves impulsivity and peer susceptibility overriding it. X v. State of Bihar (2026) held the report advisory and not binding, and vitiated a decision that ignored the Social Background and Social Investigation Reports.

PARAMETER	JUVENILE JUSTICE BOARD	CHILD WELFARE COMMITTEE
Population	Children in conflict with law	**Children in need of care and protection, the 12 categories under Section 2(14)**
Composition	A Principal Magistrate with 2 social workers, at least 1 a woman, at least 1 with child psychology or welfare background	**5 members, at least 1 a woman and at least 1 an expert on children's issues; quorum of 3 for final disposal, sitting at least 20 days a month**
Power over police	May direct registration of a First Information Report under Section 8(3)(l)	**No such power; must write to the Board**
Interface	Refers a child also in need of care and protection to the Committee under Section 17(2)	**Placements and the individual care plan, with aftercare from 18 to 21 years, extendable by 2 years to 23**

TRAP Two mistakes with "may": **mandatory** in the proviso to Section 15(1) where the Board lacks a child psychology or psychiatry member, **directory** in Section 101(2) on appeal. A third: murder is heinous, because life imprisonment is the implied minimum where the court has no discretion to go lower, and the bar in Section 21 on death or life without release operates at sentencing and does not downgrade the classification. And POCSO's cross-references to the old Penal Code and Criminal Procedure Code predate the 2023 recodification: the concordance is unsourced in the library, so name the POCSO section and supply no converted number.

NDPS Act 1985. Commenced 14 November 1985 and amended in 2001, 2014 and 2021; the 2001 amendment built three sentencing tiers on quantity, and since a notification of 2009 the weight counted is the whole mixture, not the pure drug.

Section 27 punishes consumption: up to 1 year or a fine up to 20,000 rupees or both for cocaine, morphine, diacetylmorphine or another notified drug, and up to 6 months or up to 10,000 rupees or both for any other substance, cannabis included.

Section 64A: an addict charged under Section 27, or with a small quantity offence, who voluntarily seeks and undergoes de-addiction treatment at a hospital or institution maintained or recognised by the Government is not liable to prosecution; the immunity is withdrawable if the full course is not completed, and it does not reach intermediate or commercial quantity however severe the dependence.

Section 71 lets the Government establish, recognise or approve centres for identification, treatment, management, education, aftercare, rehabilitation and social re-integration.

The 2014 amendment added "recognise or approve" and, decisively, "management", the statutory foundation for opioid substitution therapy, and created essential narcotic drugs under Section 2(viiia) under central rules, unwinding the state excise licensing that had followed a 97 percent fall in medical morphine consumption.

Under Section 37 commercial quantity offences are cognizable and non-bailable on a twin test: the Public Prosecutor is heard, and the court must be satisfied on reasonable grounds that the accused is not guilty and is not likely to offend on bail.

SUBSTANCE	SMALL	COMMERCIAL	WHY IT MATTERS
Buprenorphine	1 g	20 g	**The figure candidates miss: 1 g, not 2 g**
Methadone	2 g	50 g	**A notified essential narcotic drug**
Morphine and heroin	5 g	250 g	**Identical figures: remember them as a pair**
Ganja	1 kg	20 kg	**Flowering or fruiting tops; bhang from leaves and seeds falls outside Section 2(i)**

SUBSTANCE	SMALL	COMMERCIAL	WHY IT MATTERS
Charas	100 g	1 kg	**Separated resin, crude or purified, including hashish oil**
Cocaine	2 g	100 g	**A coca derivative includes preparations above 0.1 percent cocaine**
Amphetamine, methamphetamine	2 g	50 g	**Scheduled psychotropic substances**
LSD	2 mg	100 mg	**Milligrams, not grams: the unit error being fished for**
Diazepam, methaqualone	20 g	500 g	**Prescription agents with notified thresholds, so dispensing records matter**

Fig 3 The forensic decision spine as one path: the clinical event, the statute it engages, the provision, the act it licenses or forbids, the person who authorises it. Cover the right three columns and rebuild them from the clinical event alone; the rows that stall are the sections to reopen.

HOLD THIS

POCSO: reasonable suspicion, immediate report, immunity in good faith. Juvenile Justice: sixteen years plus a heinous offence opens the assessment, and the expert advises the Board. NDPS: Section 64A covers consumption and small quantity only, and "management" in Section 71 licenses substitution therapy.

WHAT EARNS THE MARKS

- **Reporting on suspicion rather than proof**: an internal committee does not discharge the duty
- **Calling the assessment an inquiry**: four parameters, and the report is advisory
- **Drawing the cognitive against emotional distinction**: knowing a rule differs from resisting an impulse
- **Bounding the drug immunity**: consumption and small quantity only, and the course must be completed

SEE ALSO 23 Responsibility and fitness 29 Child psychiatry 30 Abuse and self-harm

25 · Civil capacities, other statutes, and confidentiality

FORENSIC · TOPIC 25

Can a man with schizophrenia make a will, marry, and vote?

VIVA TABLE: LAW; THE CIVIL-CAPACITY VIGNETTE IN THE FORENSIC STATION

THE SHAPE OF THE ANSWER

1 **The principle**	Capacity is functional, decision-specific and time-specific
2 **The capacities**	Testamentary, contractual, marriage, divorce, adoption, driving, voting

3 Void against voidable	A contract falls away, a marriage stands until annulled
4 Negligence	Duty, breach against the competent practitioner, and damage
5 Confidentiality	The exceptions, the duty to protect, and the Indian position

In civil law capacity is functional, decision-specific and time-specific. A diagnosis settles nothing: the question is the specific test for the specific decision at the specific moment.

CAPACITY	INSTRUMENT	THE TEST	THE DISCRIMINATOR
Testamentary	Indian Succession Act 1925, Section 59, with Banks v Goodfellow (1870)	Four limbs: the nature of a will and its effects; the extent of the property; the claims of those who might expect to benefit; freedom from a delusion that perverts the disposition	**A delusion defeats capacity only if it causally shapes the disposition; an eccentric will made with capacity stands**
Contractual	Indian Contract Act 1872, Sections 11 and 12	Capable of understanding the contract and of forming a rational judgment as to its effect upon his interests	**A person usually of unsound mind may contract in a lucid interval; the agreement of one without sound mind is void**
Marriage	Hindu Marriage Act 1955, Section 5(ii); Special Marriage Act 1954 in similar terms	Neither party incapable of valid consent from unsoundness of mind, nor with mental disorder of a kind or extent making him unfit for marriage and the procreation of children, nor subject to recurrent attacks of insanity	**Breach makes the marriage voidable under Section 12(1)(b), not void; Section 11 voids only bigamy, sapinda and prohibited degrees**
Divorce	Hindu Marriage Act 1955, Section 13(1)(iii)	Incurably of unsound mind, or continuous or intermittent mental disorder of such a kind and extent that the petitioner cannot reasonably be expected to live with the respondent	**Two elements: the disorder and the unreasonableness of cohabitation**
Adoption and driving	Hindu Adoptions and Maintenance Act, with the Central Adoption Resource Authority statutory under the Juvenile Justice Act 2015; Motor Vehicles Act	Sound mind in the adopter and in the person giving the child; and no disease or disability making driving a source of danger to the public	**Advise suspension during acute psychosis, mania or severe depression and during rapid titration, resuming on sustained stability**

CAPACITY	INSTRUMENT	THE TEST	THE DISCRIMINATOR
Voting	Representation of the People Act 1950, Sections 16(1)(b) and 16(2), with Article 326	An adult citizen not judged to be of un-sound mind	**Diagnosis, admission or treatment does not disqualify: it takes a court declaration, Section 3(5) of the 2017 Act read from the other side**

The Golden Rule. From Kenward v Adams (1975): where an elderly or seriously ill testator makes a will, a medical practitioner should examine him, satisfy himself of capacity, witness or approve the will and record the findings at the time. It is good practice rather than law, so a will made without it stands unless incapacity, undue influence or fraud is proved.

NEGLIGENCE, IN THREE ELEMENTS

A claim needs a duty of care, a breach measured against a reasonably competent practitioner, and damage caused by the breach, on the Bolam standard affirmed in Jacob Mathew v. State of Punjab, which also separated civil from criminal negligence, the latter needing gross negligence or recklessness.

The exposed areas in psychiatry are four.

- **One.** Undocumented suicide risk assessment.
- **Two.** Discharge of a severely suicidal patient without family communication.
- **Three.** Non-disclosure of predictable serious adverse effects.
- **Four.** Contravention of the statutory bars on unmodified electroconvulsive therapy and on restraint.

Registration under the Clinical Establishments Act 2010 does not exempt a psychiatric facility from registering under Section 65 of the 2017 Act.

Confidentiality and the duty that qualifies it. Confidentiality is a professional and ethical duty in every setting; testimonial privilege is a rule of evidence belonging to the patient, operating in court, waivable by the patient and not the clinician.

Tarasoff I (1974) created a duty to warn the identifiable victim; Tarasoff II (1976) replaced it with a duty to protect, holding that a therapist who determines, or under professional standards should determine, that a patient presents a serious danger of violence to another must use reasonable care to protect the intended victim, the protective privilege ending where the public peril begins.

Discharging it is clinical before it is forensic: admission, intensified contact, medication optimisation, removal of lethal means, and notification of the victim or police when less intrusive measures will not contain the risk.

India has no domestic equivalent: the sources are the rights chapter of the 2017 Act, disclosure being restricted to a threat to life or public safety or an order of the Board or a court, and the mandatory reporting statutes, which override confidentiality outright.

MNEMONIC

Five Cs, then DEAL

Exceptions: **C**onsent, **C**ontinued treatment, **C**ourt order, **C**omply with the law, **C**ommunicate a threat. The algorithm: weigh the **D**uty against competing duties, check whether an **E**xception applies, **A**sk seniors, the ethics committee or counsel, confirm the **L**egal status in your jurisdiction. Then disclose the minimum necessary and record the reasoning.

TRAP A careful disclosure does not immunise the clinician. In Viviano v. Moan the psychiatrist disclosed only after consulting colleagues and a health-law attorney, notifying the threatened judge on unanimous advice, and was sued anyway for breach of confidentiality; the case turned on the meaning of "imminent" and ran 7 years as the first reverse Tarasoff case before the defendants prevailed. Document the credibility assessment, the timing, the consultation and the reasoning.

Records and certificates. The patient owns the information, the institution owns the medium. Entries are contemporaneous; corrections go in as separate labelled, dated, initialled notes, and once a claim is known no retrospective alteration is permissible. Psychotherapy process notes are kept separate and lose that protection once filed into the general record.

For a dying declaration the clinician certifies a sound disposing state of mind before it begins, stays present, and certifies again at the end, the statement recorded verbatim, preferably by an Executive Magistrate, without leading questions.

A wound certificate carries administrative data, two identification marks, the history in the patient's own words without attributing guilt, each injury by site, type, dimensions, margins, direction and age, an opinion on the weapon, and classification as simple or grievous.

VERIFY BEFORE THE EXAM Three numbers the library leaves open and the examiner asks for: the section of the 2017 Act carrying confidentiality and the adjacent one restricting release of a photograph or information to the media, with its police exception for tracing a missing person; the statutory hook admitting a dying declaration, under the Evidence Act of 1872 and its successor the Bharatiya Sakshya Adhiniyam, 2023; and the provision defining grievous hurt after the Penal Code was replaced by the Bharatiya Nyaya Sanhita, 2023. None is printed here.

HOLD THIS

Void against voidable is the civil discriminator: a contract made without sound mind is void from the start, a marriage in breach of the mental-health condition is voidable and stands until annulled. A clinical diagnosis disenfranchises nobody: that takes a court.

WHAT EARNS THE MARKS

- **Answering per decision, not per diagnosis**: the test belongs to the decision in front of you
- **Giving the four testamentary limbs**: and the causal role a delusion has to play
- **Holding void apart from voidable**: the civil discriminator in this station
- **Naming the Indian confidentiality position**: the rights chapter, and the mandatory reporting statutes

SEE ALSO 18 The Act 2017 frame 22 Disability Act 2016 23 Responsibility and fitness

Community psychiatry

Examined as a chain: a policy, a programme, a district team, and a patient who reaches none of them.

26 · Programmes, policy, money and the treatment gap

COMMUNITY · TOPIC 26

What is the National Mental Health Programme, and what has it delivered?

VIVA TABLE: COMMUNITY PSYCHIATRY; A STANDING POLICY QUESTION

THE SHAPE OF THE ANSWER

1 **Three instruments**	Policy states vision, programme moves resources, Act creates rights
2 **The programme**	Objectives, current components, and the district model that seeded it
3 **The district team**	The cadres, the beds, and the satellite clinics
4 **The statutory spine**	The Section 18 duties the programme is meant to meet
5 **Gap and money**	Quote a gap with its population, then the utilisation figures

Policy, programme and Act are three kinds of instrument. A policy states vision and is not justiciable; a programme is machinery that moves drugs, training and money; an Act binds and creates enforceable rights.

India holds one of each. The National Mental Health Policy of October 2014, grounded in equity, social justice and a rights-based approach and naming the populations needing tailored models; the National Mental Health Programme of 1982; and the Mental Healthcare Act 2017.

- **Objective one.** Minimum mental healthcare accessible to all, particularly the most vulnerable.
- **Objective two.** Application of mental health knowledge in general healthcare and social development.
- **Objective three.** Community participation.
- **Objective four.** Enhancement of human resources.

Its four current components: integration into general healthcare through the district programme, training of human resources, Centres of Excellence, and tele-mental health.

Bellary and the district programme. The Bellary district model in Karnataka showed that general duty medical officers and multi-purpose health workers could identify severe mental disorders, start basic treatment and maintain patients locally under periodic specialist supervision.

The district programme entered the national programme in 1996 with four components: early detection and treatment with essential psychotropics under specialist oversight; training of medical officers on a limited formulary and of community health workers in case identification; information, education and communication; and simple monitoring through case registers.

It began in 4 districts, reached 27 in the ninth plan period, and carries central approval for 767 districts. Tele-MANAS, launched on 10 October 2022, runs round the clock on short code 14416 and toll-free 1800-89-14416.

TIER	SANCTIONED STAFF	THE FIGURE TO QUOTE
District hospital or medical college	The district team of 7 cadres: psychiatrist, clinical psychologist, psychiatric social worker, community or psychiatry nurse, monitoring and evaluation officer, case registry assistant, ward assistant	**A 10-bedded inpatient unit and 4 satellite clinics a month at community or primary health centres**
Community health centre	1 medical officer with 1 clinical psychologist or psychiatric social worker	**The first level at which the Act requires basic and emergency mental healthcare, under Section 18(6)**
Primary health centre	2 community health workers	**Case finding and follow-up on a district plan; severe disorders go up for confirmation**
Sub-centre and wellness centre	Accredited social health activists and community health officers	**School work trains 100 teachers a block as master trainers covering 25 to 30 schools**

THE STATUTORY SPINE

Section 18(1) gives a right to access mental healthcare from services run or funded by the appropriate Government, affordable, of good quality, sufficient, geographically accessible and non-discriminatory.

Section 18(5) carries the duties that map onto the district programme.

- **Integrate.** Mental health into general healthcare at primary, secondary and tertiary levels.
- **Treat.** So as to support living in the community and with the family.
- **Institutional care.** Only in exceptional circumstances, for as short a duration as possible, and as a last resort after community treatment has been tried and has failed.
- **Provide.** Government services as a minimum in each district.

Section 19 gives the right to community living and requires less restrictive establishments including half-way homes and group homes;

Burden and gap. The older-adult analysis of the national survey gives 84%, from 76.9% for neurotic and stress-related disorders to 90.9% for severe mental disorders, with a recognition rate of 15.7% and a median duration of illness before care of 24 months. Workforce: about 0.75 psychiatrists per 100,000 population against a desirable minimum of at least 3 per 100,000.

Money. About 10.6% of the population lives with a mental health condition, and the gap across conditions is reported between 70% and 92%, with other reviews citing 76% to 85%.

Mental health takes less than 1% of the central health budget: an outlay of 1035.39 crore rupees for 2022 and 2023, of which 670 crore was direct.

Of that direct allocation 94% went to two centrally funded institutions, leaving about 7% for the programme.

Utilisation is the other half: 96% and 93% of programme funds were surrendered unutilised in the two financial years from 2018, and of 52,225 crore rupees allocated to States between 2015 and 2021 only 38.11% was used. The district programme runs on a 60:40 Union and State split.

RULE Quote a gap with its population attached: 84% with its 76.9% to 90.9% range belongs to older adults in the Indian national survey, 78.6% is Singapore's twelve-month figure, 85% the general low and middle income country figure. Then name the critique: the metric counts only formal psychotropic treatment and ignores non-allopathic, private and community psychosocial care, which is why "treatment difference" and "care gap" were proposed.

HOLD THIS

Programme 1982, district programme 1996, seven cadres, ten beds, four satellite clinics a month. Less than one per cent of the health budget, and most programme money returned unspent.

WHAT EARNS THE MARKS

- **Separating policy from programme from Act**: only one of the three is justiciable
- **Attaching the population to the gap figure**: a bare percentage invites the follow-up
- **Naming the utilisation problem**: most programme money was returned unspent
- **Citing the district model by name**: it is why task-sharing entered the programme

SEE ALSO 21 Institutions 27 Rehabilitation 28 Community models

27 · Rehabilitation: work, day care, halfway homes, family

COMMUNITY · TOPIC 27

What rehabilitation will you offer this man with residual schizophrenia?

VIVA TABLE: COMMUNITY PSYCHIATRY; ALSO THE LONG CASE MANAGEMENT PLAN

THE SHAPE OF THE ANSWER

1 **Define the target**	Rehabilitation treats the disability, not the symptom
2 **The four domains**	Personal, social, vocational, and civil or community
3 **Train then place against place then train**	Whether placement precedes training is the discriminator
4 **The eight principles**	Competitive work, integration, benefits planning, zero exclusion, unlimited support
5 **Settings and family**	The ambulatory centre, the residential home, and family work

Rehabilitation treats the disability, not the symptom. It helps a person impaired by a mental disorder reach an optimal level of independent functioning in the community, addressing secondary disability, demoralisation, institutionalism and social exclusion across four domains:

personal and self-care, social and interpersonal, vocational and educational, and civil and community.

PARAMETER	TRAIN THEN PLACE	PLACE THEN TRAIN, THAT IS SUPPORTED EMPLOYMENT
Philosophy	Skills acquired before open employment	**Immediate placement in a real job, training delivered on it**
Setting	Sheltered workshops and simulated work in institutional settings	**Integrated competitive employment in the open labour market**
Entry	High barrier: symptom stability, cognitive readiness, abstinence	**Zero exclusion, on client choice and motivation to work**
Pay	Sub-minimum wages, stipends, unremunerated craftwork	**At least the minimum wage, or the rate co-workers get for the same task**
Timeline and support	Prolonged pre-placement training; support ends with the programme	**First face-to-face employer contact within 30 days of entry; support individualised and time-unlimited**

Individual placement and support. Eight principles: competitive employment; services integrated into the clinical team, the specialist attaching to 1 or 2 multidisciplinary teams; benefits planning, so fear of losing a pension does not block work; zero exclusion whatever the diagnosis, symptoms, admissions, substance use, housing or legal involvement; time-unlimited support with face-to-face contact at least monthly; and worker preference driving job choice.

Fidelity runs on a 25-item scale, each item scored out of 5, with caseloads ideally below 20 clients a specialist and caseloads of 50 reported among delivery problems.

Day care, halfway homes, family. A day care centre is ambulatory: structured therapeutic, social and prevocational engagement for stable outpatients living with family, building routine against apathy and withdrawal, with skills training, cognitive remediation, caregiver respite and conditioning of work tolerance.

A halfway home is residential and non-hospital, bridging inpatient care and independent or family living for stable people with residual impairment who lack family support or need transitional living-skills training, with shared chores, budgeting, peer support and medication self-management.

Because public halfway homes are scarce, people who have remitted stay on in long-stay hospitals, and those discharged without a step-down relapse or become wholly dependent on carers.

Family work is the Indian backbone: psychoeducation on illness, relapse signs and realistic expectations; work on high expressed emotion, meaning critical comments, hostility and emotional over-involvement; attention to financial strain and out-of-pocket costs carried by the

family; and management of structural stigma, including the effect on marriage prospects for siblings, which drives concealment and delay.

THE DEPENDENCY TO NAME

Because public infrastructure concentrates on acute biological treatment, halfway homes, day centres and vocational units have largely fallen to non-governmental organisations, with **four vulnerabilities**.

- **Financial fragility.** Donor funding fluctuates.
- **Quality variation.** Little standardisation of care, staff qualification or fidelity monitoring.
- **Geographic inequity.** Services cluster in cities while most of the population is rural.
- **Limits of scale.** No statutory authority and no capital to go national.

The answer is intersectoral, health with social justice and labour, linking clinical stabilisation to skill development, job reservation and disability pension.

Community-based rehabilitation is built for the constraint: task-sharing to trained lay workers under regular specialist supervision, local adaptation instead of transplanted institutional models, home-based family psychoeducation and skills training in the person's own environment, and stigma reduction through local bodies and self-help groups.

Named Indian organisations and halfway homes are not carried in the library here, so prepare one worked local example rather than inventing a name at the table.

TRAP Describing a sheltered workshop when asked about supported employment: the discriminator is whether placement precedes training. And requiring remission before vocational work, when zero exclusion means symptoms, readiness and diagnosis do not bar entry. Repetitive basket or rug making without wages or social measures is not rehabilitation.

HOLD THIS

Place then train, zero exclusion, employer contact within thirty days, support without a time limit. Day care is ambulatory, halfway homes residential, and both fail by becoming permanent.

WHAT EARNS THE MARKS

- **Naming the discriminator**: whether placement precedes training separates the two models
- **Applying zero exclusion**: symptoms, readiness and diagnosis do not bar entry
- **Putting family work at the centre**: expressed emotion, out-of-pocket cost, and structural stigma
- **Naming the non-governmental dependency**: fragility, quality variation, urban clustering, limits of scale

SEE ALSO 16 Function and disability 26 Programmes and policy 28 Community models

28 · Community models: assertive treatment, mhGAP, task shifting, school and disaster work

COMMUNITY · TOPIC 28

What is assertive community treatment, and would it work in your district?

VIVA TABLE: COMMUNITY PSYCHIATRY; PAIRED WITH THE REHABILITATION QUESTION

THE SHAPE OF THE ANSWER

1 Define by organisation	A shared caseload, care given in vivo, cover round the clock
2 Who qualifies	Diagnosis, duration and disability criteria, with the revolving-door pattern
3 The contrasts	Case management brokers, this team treats; then the adaptations
4 What the evidence shows	Fewer admissions and better engagement, not better employment
5 Task shifting	Three tiers, with the safeguards that make it defensible

Assertive community treatment is defined by its organisation, not its drugs. Severe mental illness is operationalised on three dimensions.

- **Diagnosis.** Non-organic psychosis, major affective disorder or disabling personality disorder.
- **Duration.** Two years or longer, with functional deterioration over the preceding 6 months despite symptom remission.
- **Disability.** At least 2 of 5 criteria: employment, public assistance, interpersonal support, help with daily living, behaviour requiring intervention.

Eligibility prioritises the revolving-door pattern: people who cannot engage with clinic-based care and are repeatedly readmitted, including those homeless, justice-involved or with co-occurring substance use.

DOMAIN	CLASSIC ASSERTIVE COMMUNITY TREATMENT	THE CONTRAST TO DRAW
Team and caseload	Multidisciplinary team sharing one caseload, with no individual lists	**Case management brokers appointments with outside agencies; this team treats. Ratio typically 1:6 to 1:15**
Setting and intensity	In vivo, in homes, workplaces and public spaces	**Contact reaching up to 2 visits a day by acuity**
Availability	Crisis cover round the clock by the team's own clinicians	**Time-unlimited responsibility, no programmatic discharge date**
Adaptations	Flexible teams step care up and down on a tracking board; Housing First adds immediate scattered-site housing; forensic teams integrate probation	**The early-intervention model gives 2 years of specialised intervention then 3 years of standard care**

What the evidence says. Across 129 studies published from 1990, the replicated outcomes are fewer admissions, fewer bed-days and fewer emergency attendances, with gains in engagement and adherence. In India high-fidelity ratios are unreachable, so the model is modified, a South Indian evaluation in schizophrenia showing effectiveness and positive consumer views.

mhGAP and task shifting. The Mental Health Gap Action Programme exists because up to 85 percent of people with mental disorders in low and middle income countries receive no treatment; its intervention guide gives algorithmic decision trees for non-specialists.

Task shifting runs three tiers.

- **Specialists.** Complex and refractory cases, forensic assessment, and supervision.

- **Primary medical officers.** Start and monitor pharmacotherapy, and manage side effects.
- **Community workers and lay counsellors.** Screen, psychoeducate, support adherence and refer.

The safeguards are the ethics answer: competency-based training on standardised protocols, protected specialist supervision, explicit referral criteria for acute psychosis, active suicidal intent and treatment resistance, and an uninterrupted supply of essential psychotropics.

School and disaster work. School mental health is three-tiered.

- **Universal.** Life-skills education on the ten core skills, with anti-bullying work and mental health literacy, delivered through the school health and wellness programme with nominated teachers as ambassadors.
- **Targeted.** Work for the child with an academic drop, chronic truancy, withdrawal or disruptive behaviour.
- **Indicated.** Referral to a child guidance clinic or the district child clinic, carrying the mandatory reporting duty on reasonable suspicion of sexual abuse.

Disaster mental health runs up a pyramid from basic services and security, through community and family supports such as tracing relatives and mourning rituals, to focused non-specialised support by primary care workers, to specialised services for post-traumatic stress disorder, severe depression and psychosis.

The acute phase is psychological first aid: **Look, Listen, Link**.

- **Look.** For safety, injury and severe distress.
- **Listen.** Respectfully, asking about immediate needs without forcing recollection.
- **Link.** To basic needs, family reunification and social support.

The intermediate phase screens for acute stress reactions, depressive episodes, anxiety and escalating substance use; the long-term phase treats chronic post-traumatic stress disorder, prolonged grief and major affective disorders.

TRAP First aid against debriefing. Psychological first aid is humane, non-intrusive practical help focused on safety, stabilisation and linkage, and does not probe the trauma. Mandatory debriefing forces ventilation soon after exposure, does not prevent post-traumatic stress disorder, and can worsen outcomes. A second acute error is indiscriminate benzodiazepine prescription in bereavement instead of containment and sleep hygiene.

HOLD THIS

A shared caseload, a low ratio, care given at home, cover round the clock: the replicated gain is fewer admissions and better engagement, not better employment. Task shifting without supervision is task dumping.

WHAT EARNS THE MARKS

- **Defining the model by its organisation**: not by its drugs or its diagnostic mix
- **Stating the outcome honestly**: admissions and engagement improve, employment does not follow
- **Naming the safeguards for task shifting**: training, supervision, referral criteria, drug supply
- **Separating first aid from debriefing**: one links to needs, the other forces ventilation

SEE ALSO 26 Programmes and policy 27 Rehabilitation 30 Abuse and self-harm

Child, women, geriatric, addiction, liaison, psychotherapy, culture, sex and sleep

Eight sub-specialties, one question shape: the criterion count, the discriminator against the near neighbour, and the first-line treatment. Answer in that order and the follow-up is usually the trap named below.

29 · Child and adolescent psychiatry

SUB-SPECIALTY · TOPIC 29

Give me the criteria for attention-deficit hyperactivity disorder, and your first step.

VIVA TABLE: SUB-SPECIALTY; ALSO THE CHILD LONG CASE

THE SHAPE OF THE ANSWER

1 **The counts**	Six symptoms up to 16 years, five from 17, over six months
2 **The developmental gate**	Onset before age 12 years, and across two settings
3 **Treatment by age band**	Behavioural work first in preschool, then medication with support
4 **Learning disability**	The Indian battery, the age it becomes measurable, concurrent intelligence
5 **Enuresis**	Developmental figures, behavioural work, alarm, then the drug and its fluid rule

Give the counts, then the developmental gate.

- **Count.** At least 6 inattention or 6 hyperactivity-impulsivity symptoms up to age 16 years.
- **From 17 years.** At least 5 of either.
- **Duration.** Persisting at least 6 months, to a degree inconsistent with developmental level.
- **The gate.** Several symptoms present before age 12 years, and across 2 or more settings.

GROUP	FIRST LINE	MEDICATION NOTE
Preschool, 4 to 5 years	Parent training in behaviour management, with behavioural classroom intervention	**Methylphenidate only if behavioural work fails and impairment is severe; a second specialist opinion is advised below 5 years**
School age, 6 to 12 years	Medication plus parent training and classroom intervention, with formal educational support	**Immediate-release methylphenidate 0.3 to 0.5 mg per kg a dose, to a maximum of 60 mg daily**
Adolescent, 12 to 18 years	Medication with the patient's assent, plus behavioural therapy and accommodations	**After a 6-week trial at an optimal methylphenidate dose without response, consider lisdexamfetamine**

Learning and developmental disability. Specific learning disorder is one of the 21 specified disabilities. A clinical psychologist administers the standard Indian battery one to one, across

reading, writing and spelling, arithmetic, and the supporting cognitive abilities.

It runs from about age 8 years onwards, since formal instruction must run for some years before a reliable discrepancy between capacity and achievement can be measured.

Intelligence is assessed concurrently on a standardised Indian scale to exclude generalised impairment.

Enuresis. Continence is developmental: at age 2 years 25% are dry by day and 10% by night; by 2.5 years, 85% and 48%; by 3 years, 98% and 78%. Active treatment is not recommended below 6 or 7 years.

Behavioural work runs up to 3 months: timed voiding on waking, at transitions, about every 1.5 to 2 hours and before bed; access to school toilets; liberal daytime fluids; a diary and no punishment.

Then alarm therapy, which raises functional bladder capacity or arousal rather than reducing urine output, with two-thirds dry during use, about half relapsing and a third sustained at 6 months.

Desmopressin is the first-line drug, oral tablets from 0.2 mg at bedtime to a maximum of 0.6 mg, with a strict fluid rule: at most 1 cup with the evening meal, at most 1 cup between dinner and bed, and nothing within 2 hours of sleeping, stopping the drug at once for headache, nausea or vomiting.

Intranasal formulations carry a boxed warning for fatal hyponatraemic seizures, and the World Health Organization does not recommend imipramine for enuresis given cardiotoxicity and a narrow therapeutic index.

TRAP School refusal against truancy. In refusal the parents know the child is at home, the child stays with them, and mornings bring anticipatory anxiety, panic or somatic complaints, with academic motivation intact; separation anxiety, social anxiety, panic, depression or bullying sit underneath, and management is a rapid coordinated return with graded exposure and treatment of the comorbidity. In truancy the absence is concealed, the child is away from home with peers, distress is absent, and antisocial features cluster; the disorder is conduct or oppositional defiant. Oppositional defiant disorder is angry or irritable mood, argumentative or defiant behaviour and vindictiveness towards authority without violating others' basic rights; conduct disorder violates them, across aggression to people and animals, destruction of property, deceitfulness or theft, and serious rule violations. The library marks this comparison unsourced, so it is given as clinical grammar and not as a sourced count.

HOLD THIS

Six symptoms to 16 years, five from 17; onset before 12; two settings; six months. Behaviour management first under 6 years, and desmopressin is unsafe without the evening fluid rule.

WHAT EARNS THE MARKS

- **Giving counts with the age break**: the threshold falls at 17 years
- **Putting behavioural work first under six years**: medication follows failure and severe impairment
- **Attaching the fluid rule to desmopressin**: the drug is unsafe without it
- **Separating refusal from truancy**: concealment, company and distress are the discriminators

SEE ALSO 17 Child and eating scales 24 POCSO, Juvenile Justice, NDPS 30 Abuse and self-harm

30 · Abuse, adolescent self-harm and gaming

SUB-SPECIALTY · TOPIC 30

This child has bruises the father cannot explain. How do you proceed?

VIVA TABLE: SUB-SPECIALTY AND LAW; ALSO THE EMERGENCY STATION

THE SHAPE OF THE ANSWER

1 The frequencies	Neglect is the commonest form, with the abuse prevalence figures
2 Inflicted against accidental	Site, pattern, timing, behaviour, and the mimics to exclude
3 Exclude before naming	Blood count, coagulation profile, skeletal survey
4 Self-harm	The definition, the no-scale rule, and the psychosocial assessment
5 Gaming	The two classifications, their criteria counts, and their status

Neglect is the commonest form, at nearly 78% of reported cases in surveillance data. A national study documented child sexual abuse prevalence of 10.14%, 24.8% in males and 75.2% in females. Separate inflicted from accidental injury and exclude mimics before naming abuse.

FEATURE	INFLICTED INJURY	ACCIDENTAL INJURY
Site	Bilateral, and over soft tissue: cheeks, neck, abdomen, genitalia, both sides of the head	**Unilateral and over bony prominences: forehead, elbows, knees, shins**
Pattern	Marks mirroring an implement: belt, hand, bite, grab marks, cigarette burns	**Diffuse irregular abrasions without geometric borders**
Timing	Multiple fractures or contusions in varying stages of healing	**Single-stage injury matching one documented event**
Behaviour	Hypervigilance, flinching, fear of going home; caregiver account conflicting or incompatible with the injury	**Normal comfort-seeking; account consistent**
Mimics to exclude	Osteogenesis imperfecta, inherited coagulopathy, thrombocytopenia	**Blood count, coagulation profile and skeletal survey before the word abuse is used**

Self-harm. On the current guideline, self-harm is intentional self-poisoning or injury irrespective of apparent purpose, excluding repetitive stereotyped self-injury. Non-suicidal self-injury is

usually about affect regulation: relief from intolerable feeling or numbness, self-punishment or regaining control, without an intent to die.

- **Rule one.** Clinicians do not rely on risk assessment tools or scales alone to predict recurrence or to decide admission or discharge.
- **Rule two.** Assessment is a personalised psychosocial evaluation of intent, access to lethal means, comorbid depression or trauma, and home safety.

Then safety planning with distress-tolerance alternatives and a crisis contact the adolescent can actually reach, safer prescribing with restricted supply and supervised medication, and trauma-focused cognitive behavioural therapy or child-parent psychotherapy where abuse is the antecedent. The Indian crisis contact to write into the plan is the national tele-mental health line, on short code 14416 or toll-free 1800-89-14416.

TRAP Foreign resources and foreign statutes. The text line quoted in much of the self-harm literature serves North America and Europe and does not reach India, so it cannot go into an Indian safety plan; the mandated-reporting penalties in the same literature are Californian. The Indian anchors are the statutory presumption of severe stress in Section 115 of the 2017 Act after an attempt, and POCSO for abuse.

PARAMETER	INTERNET GAMING DISORDER, DSM-5-TR	GAMING DISORDER, ICD-11
Status	A condition for further research in Section III	**A formal diagnosis among disorders due to addictive behaviours**
Criteria	At least 5 of 9: preoccupation, withdrawal, tolerance, failed control, lost interests, continued use despite problems, deception, mood relief, jeopardised relationships or work	**All 3: impaired control; gaming taking precedence over other interests and activities; continuation or escalation despite negative consequences**
Duration and yield	Over 12 months; prevalence 3.83% in a representative young-adult sample	**Normally at least 12 months; 2.48% in the same sample, a smaller but clinically more severe group**

HOLD THIS

Bilateral and soft-tissue, patterned, multi-stage: think inflicted; unilateral and bony, single-stage: think accident. No scale predicts self-harm; the assessment is psychosocial and the plan names a reachable crisis contact.

WHAT EARNS THE MARKS

- **Excluding the mimics before the word abuse**: coagulopathy and bone disease come first
- **Refusing to triage self-harm on a scale**: the assessment is a personalised psychosocial evaluation
- **Writing a reachable crisis contact into the plan**: a foreign text line does not reach India
- **Distinguishing the two gaming constructs**: one is for further research, the other a formal diagnosis

SEE ALSO 03 Risk scales 24 POCSO, Juvenile Justice, NDPS 29 Child psychiatry

31 · Women's mental health

SUB-SPECIALTY · TOPIC 31

How will you confirm premenstrual dysphoric disorder, and treat it?

VIVA TABLE: SUB-SPECIALTY; ALSO THE OUTPATIENT LONG CASE

THE SHAPE OF THE ANSWER

1 **The criteria**	Five symptoms in the final week, one from the affective core
2 **The diary gate**	Prospective daily ratings across at least two symptomatic cycles
3 **The quiet window**	A noisy mid-follicular phase means exacerbation, not this disorder
4 **Treatment ladder**	Serotonergic first, then hormonal and adjunctive, then the refractory options
5 **Perimenopause and postpartum**	The steroid mechanisms, and what each one licenses

Premenstrual dysphoric disorder is a depressive disorder with a prospective-diary gate.

- **Count.** At least 5 symptoms from 11 domains in the final week before menses.
- **Timing.** Improving within a few days of onset, and minimal or absent in the week after.
- **Course.** Across most cycles in the preceding year, with at least 1 from the affective core.
- **Impact.** Causing distress or impairment.
- **The gate.** Confirmed on prospective daily ratings across at least 2 symptomatic cycles.

Community prevalence runs 1.3% to 5.3% and Indian studies about 6%. Circulating oestrogen and progesterone are normal: the disorder is a differential central response to physiological steroid shifts.

QUESTION	ANSWER	DISCRIMINATOR
Confirming the diagnosis	Daily record of severity of problems, scored 1 to 6, over at least 2 cycles: at least 4 for at least 2 days on at least 5 symptoms, with an impairment item	**The mid-follicular window, days 6 to 10, must be quiet: an average above 3 there means premenstrual exacerbation of another disorder, not this one**
First-line drug	A serotonergic antidepressant, continuously or in the luteal phase, from ovulation to menses	**Response comes within days, not weeks, which is what permits luteal-only dosing; bupropion is ineffective**
Hormonal and adjunctive	Continuous low-dose combined oral contraception; low-dose quetiapine 25 mg for residual luteal anxiety and lability	**Calcium carbonate 1,000 to 1,200 mg daily halves symptoms; pyridoxine 80 mg daily gives modest benefit**
Refractory	Gonadotropin-releasing hormone analogue with add-back, then bilateral oophorectomy as a last resort	**Surgery follows a successful agonist trial and completed childbearing**

Perimenopause and the postpartum. Perimenopause precedes menopause, defined as 12 consecutive months of amenorrhoea, and typically falls in the forties; erratic follicular depletion

produces unpredictable steroid swings, falling oestrogen compromises central serotonergic transmission, and vasomotor symptoms fragment sleep, severe insomnia raising the risk of clinical depression by up to 10 times.

Mild symptoms tied to vasomotor instability may respond to a very low-dose combined pill or transdermal oestrogen with oral progesterone; hormonal treatment is contraindicated with active smoking, uncontrolled hypertension, venous thromboembolism or pulmonary embolism, active liver disease, hormone-dependent breast or endometrial malignancy, and after the menopause.

Where it is contraindicated or criteria for major depression are met, treat with a serotonergic antidepressant and cognitive behavioural therapy.

Allopregnanolone rises steeply in pregnancy and falls at delivery, and that withdrawal destabilises GABA-ergic tone in susceptible women, which is the rationale for the neuroactive steroid treatments of postpartum depression: an intravenous allopregnanolone formulation requiring continuous pulse oximetry and on-site monitoring for excessive sedation, and an oral analogue given as a short course.

TRAP Diagnosing on retrospective recall, which over-reports luteal specificity, and treating premenstrual exacerbation as the disorder itself. Two more: separating an infant from a mother with postpartum psychosis as a routine measure rather than arranging supervised joint care, and admitting her without following Section 89. Verify the 2022 Supreme Court resolution of the conflict between the confidentiality provision for a pregnant minor's termination and mandatory reporting under POCSO before quoting it: the library carries neither the ruling nor its provisions.

HOLD THIS

Five of eleven in the last week, confirmed over two cycles, with days six to ten quiet. Serotonergic response in days permits luteal dosing; calcium and pyridoxine are the supported supplements.

WHAT EARNS THE MARKS

- **Requiring the prospective diary**: retrospective recall over-reports luteal specificity
- **Checking the mid-follicular window**: it separates the disorder from premenstrual exacerbation
- **Explaining luteal-only dosing**: response within days is what permits it
- **Keeping mother and infant together where safe**: supervised joint care rather than routine separation

SEE ALSO 11 Mood scales 24 POCSO, Juvenile Justice, NDPS 32 Geriatric psychiatry

32 · Geriatric psychiatry

SUB-SPECIALTY · TOPIC 32

This woman of seventy has new persecutory delusions. What is your differential?

VIVA TABLE: SUB-SPECIALTY; ALSO THE GERIATRIC LONG CASE

THE SHAPE OF THE ANSWER

1 Define the onset bands	After 40 years is late-onset, after 60 very-late-onset
2 Secondary first	Neurodegeneration, delirium, medical illness and polypharmacy before a primary label
3 The phenomenology split	Partition delusions and visual hallucinations mark the later group
4 Dementia and delirium figures	Psychosis rates by disease, and the course that separates them
5 Prescribing and protection	A quarter of the adult dose, and the protective statute

Late-onset psychosis means a first episode after age 40 years.

- **Key number.** Population prevalence of psychosis in older adults is about 1.7%, with a lifetime incidence of 4.7%.
- **Trap.** Much of it is secondary to neurodegeneration, delirium, medical illness or polypharmacy.

FEATURE	LATE-ONSET SCHIZOPHRENIA	VERY-LATE-ONSET SCHIZOPHRENIA-LIKE PSYCHOSIS
Onset	Between 40 and 60 years; about 20% of older patients with schizophrenia began after 40	**After 60 years**
Phenomenology	Persecutory delusions and delusions of reference, auditory hallucinations, lower positive and negative symptom severity, better premorbid adjustment	**Partition delusions, visual, tactile and olfactory hallucinations, sensory impairment common, negative symptoms typically absent**
Trajectory and dose	Better memory and executive function, greater deficits in attention, fluency and visuospatial skill; lower antipsychotic dose, often a quarter	**High risk of progression to dementia; very low dose requirement**

Depression, dementia and delirium. Psychotic depression has a lifetime prevalence of about 0.35%, makes up 20% to 45% of hospitalised depressed patients and 3.6% of outpatients.

Its delusions are mood-congruent, of guilt, worthlessness, somatic decay or nihilism, with prominent retardation.

Combining an antidepressant with a second-generation antipsychotic is first-line: the trial evidence gives 41.9% remission on olanzapine with sertraline against 23.9% on olanzapine alone. Electroconvulsive therapy is the treatment for severe suicidality, food refusal or failure of drugs.

Psychosis occurs in 30% to 50% of Alzheimer disease across its course, with delusions in 15% to 30%, commonly theft, infidelity or the phantom boarder, and hallucinations in 5% to 15%. In Lewy body disease psychosis runs 30% to 80%, with well-formed visual hallucinations as a core feature alongside passage hallucinations, presence phenomena and pareidolias, and extreme antipsychotic sensitivity.

Psychosis complicates up to 60% of Parkinson disease, often dopaminergic in origin.

Delirium occurs in 25% to 35% of hospitalised older adults, with psychotic features in 42.7% to 49%; it is acute and fluctuating with impaired attention and altered arousal, against the insidious

stable course of late-onset schizophrenia and the gradual course of dementia-related psychosis, and antipsychotics do not reduce its severity or mortality.

Prescribing and protection. Ageing reduces total body water and muscle and raises fat, so lipophilic psychotropics distribute more widely and linger. Start at about a quarter of the young adult dose and titrate slowly, using the lowest effective dose for the shortest time.

For behavioural and psychological symptoms of dementia, **exclude the physical causes first**: pain, infection, constipation, retention, hypoxia, electrolyte shifts and sensory deprivation.

Then use caregiver psychoeducation, scheduled toileting, task simplification and environmental calming. Only then an off-label atypical antipsychotic, risperidone 0.5 to 2 mg daily carrying the strongest evidence, against a boxed warning of increased mortality with an odds ratio of 1.6.

The Maintenance and Welfare of Parents and Senior Citizens Act 2007 is the protective statute. A senior citizen is a citizen aged 60 years or above, while a parent may apply whether or not a senior citizen.

Section 23 voids a property transfer made on condition of basic amenities where the transferee then refuses them, and Section 24 punishes abandonment with imprisonment up to 3 months or a fine up to 5,000 rupees or both.

HOLD THIS

Forty to sixty is late-onset, after sixty is very-late-onset with partition delusions and visual hallucinations. A quarter of the adult dose, and behavioural causes before behavioural drugs.

WHAT EARNS THE MARKS

- **Excluding secondary causes before naming a primary psychosis**: much of this is secondary
- **Naming partition delusions**: the phenomenological marker of the very-late-onset group
- **Treating behavioural causes before behavioural drugs**: pain, infection, retention, sensory loss
- **Quoting the mortality warning with the drug**: the odds ratio belongs beside the prescription

SEE ALSO 15 Cognitive screening 31 Women's mental health 33 Addiction and liaison

33 · Addiction psychiatry and consultation-liaison

SUB-SPECIALTY · TOPIC 33

How common is alcohol dependence in India, and how do you explain relapse?

VIVA TABLE: SUB-SPECIALTY; ALSO THE ADDICTION LONG CASE

THE SHAPE OF THE ANSWER

1 Lead with the numbers	Current use, dependence, harmful use, and the treatment figure
2 The three-stage model	Basal ganglia, extended amygdala, prefrontal cortex
3 The four models	Incentive sensitization, habit, allostasis, and the relapse process
4 Harm reduction	Substitution, needle programmes, overdose education and antagonist distribution
5 Liaison	Two models, the referral pattern, and the commonest diagnosis

Lead with the Indian numbers. In the population aged 10 to 75 years, current alcohol use is 14.6%, with dependence in 2.7% and harmful use in 2.5%, making alcohol the commonest psychoactive substance; past-year cannabis use is 2.8% and opioid use 2.1%, and only 0.55% of those with opioid problems receive treatment or support.

Genetic factors account for an estimated 40% to 70% of vulnerability.

The three-stage model localises the cycle.

- **Binge and intoxication.** Basal ganglia, with phasic dopamine release and desensitisation producing tolerance.
- **Withdrawal and negative affect.** Extended amygdala, where anti-reward and stress activation drive use for relief, that is negative reinforcement.
- **Preoccupation and anticipation.** Prefrontal cortex, where impaired executive control leaves craving in charge, the reason adolescents are vulnerable.

MODEL	CORE CLAIM	WHAT IT EXPLAINS THAT OTHERS DO NOT
Incentive sensitization	Sensitised mesolimbic circuits dissociate wanting from liking	**Craving that intensifies while pleasure declines**
Habit and compulsion	Control shifts from ventral to dorsal striatum as behaviour automates	**The move from impulsive to compulsive seeking, and trait impulsivity as a marker**
Allostasis	Reward hypofunction with brain stress hyperactivation resets the hedonic set point	**Persistent dysphoria in protracted abstinence and stress-induced relapse**
Relapse process	A high-risk situation meets coping that either holds or fails	**The abstinence violation effect: attributing a lapse to a stable personal flaw converts it into relapse**

Motivation and harm reduction. Harm reduction minimises health, social and economic consequences without demanding immediate abstinence: opioid substitution with a long-acting agonist or partial agonist under supervised programmes, needle and syringe programmes against blood-borne infection, overdose education with community distribution of the opioid antagonist.

VERIFY BEFORE THE EXAM Naloxone is the antagonist used for opioid overdose reversal in Indian take-home and substitution-linked programmes, but its formulation, route, repeat interval and the structure of Indian take-home programmes are unsourced in the library, so no dose is printed. Read them off the national programme guidance. Do not answer this question with nalmefene, which is a different agent marketed outside India. The same caution applies to the transcranial stimulation clearance quoted for smoking cessation, which is not an Indian approval and is not standard Indian practice.

Consultation-liaison psychiatry. Two models: the reactive one waits for a referral, is asked late, and hands recommendations back to a ward team that often does not implement them; the

proactive integrated model screens new inpatients, intervenes early and co-manages as part of the ward team.

Internal medicine generates most referrals, 52.7% in Indian tertiary audits, and the commonest reasons are a general psychiatric assessment, medication review, sleep disturbance and agitation.

Psychiatric morbidity is present in about a third of general hospital inpatients, and delirium is the commonest diagnosis at 31.7% of consultations and around 15% of admissions; 24.7% of referrals are managed with no medication at all.

Delirium is secondary to physical illness until proven otherwise. Search for sepsis, electrolyte disturbance, hypoxia, organ failure and anticholinergic burden, and take a collateral history to separate it from dementia.

Screen on the confusion assessment method, which rates nine characteristics including acute onset, fluctuation, inattention, disorganised thinking and altered consciousness.

Treat the cause, with reorientation and sensory aids. Reserve low-dose antipsychotics for agitation that threatens care, and avoid benzodiazepines outside alcohol or sedative withdrawal.

Capacity is decision-specific and time-specific under Section 4, and a fluctuating state such as delirium impairs it transiently, so reassess when the medical state improves.

RULE Diabetes distress is not depression. Distress is emotional burnout tied to the regimen itself, present in 33% to 50% of people with diabetes over an 18-month period, and it does not respond to medication: it needs diabetes education, goal restructuring and endocrinology input. Depression is pervasive, is 2 to 3 times commoner than in people without diabetes, and responds to therapy and antidepressants. And check capillary glucose before calling autonomic symptoms panic, because hypoglycaemia mimics it exactly. In severe mental illness, premature mortality of up to two decades is about 60% attributable to preventable physical disease rather than suicide.

HOLD THIS

Alcohol 14.6%, cannabis 2.8%, opioids 2.1%, and only 0.55% of opioid users in treatment. A lapse is data, not failure, and delirium stays the commonest liaison diagnosis until the physical cause is excluded.

WHAT EARNS THE MARKS

- **Leading with Indian prevalence**: the treatment figure is the one that lands
- **Naming the abstinence violation effect**: a lapse becomes relapse through attribution
- **Treating delirium as secondary until proven otherwise**: search the physical causes first
- **Separating diabetes distress from depression**: one answers to education, the other to treatment

SEE ALSO 13 Substance use scales 30 Abuse and self-harm 32 Geriatric psychiatry

34 · Psychotherapy, culture, sexual medicine and sleep

SUB-SPECIALTY · TOPIC 34

Name the defence, then tell me which therapy targets it.

VIVA TABLE: SUB-SPECIALTY; THE DEFENCE QUESTION OPENS MANY TABLES

THE SHAPE OF THE ANSWER

1 **Classify by level**	Pathological, immature, neurotic, mature, with the discriminators
2 **The modalities**	Mechanism first, then the one discriminator for each
3 **Culture and stigma**	Knowledge, attitude and behaviour, with yoga as an adjunct
4 **Sexual medicine**	Psychogenic against organic, dhat, and the dysphoria criteria
5 **Sleep**	Insomnia thresholds, the first-line therapy, and the narcolepsy figures

Defences classify by level, and the level is the answer. The hierarchy carries four of them.

- **Pathological.** Grossly alter external reality: denial, distortion, delusional projection.
- **Immature.** Partly distort it and burden others: acting out, hypochondriasis, passive aggression, projection.
- **Neurotic.** Preserve reality testing while altering internal feeling: displacement, dissociation, intellectualisation, reaction formation, repression, isolation of affect.
- **Mature.** Integrate reality, relationships and impulse: altruism, anticipation, humour, sublimation, suppression.

The discriminators: projection keeps reality testing while delusional projection does not; repression is unconscious and neurotic while suppression is conscious and mature; splitting oscillates between idealisation and devaluation while compartmentalisation lets two conflicting systems coexist unexamined.

MODALITY	MECHANISM	THE DISCRIMINATOR
Cognitive therapy	The triad of negative views of self, world and future, generated by schemas and expressed as automatic thoughts, tested on a written record in 5 to 10 minutes	**Arbitrary inference draws a conclusion with no evidence or against it; selective abstraction lifts one negative detail out of a context that contains contrary evidence**
Systematic desensitisation	Reciprocal inhibition: relaxation is incompatible with anxiety, paired with a graded hierarchy	**Graded and imaginal, with relaxation deliberately present; suited to 4 fears or fewer with good imagery**
Flooding	Respondent extinction at maximum intensity without relaxation	**Ungraded and intense: faster, with far more distress**
Exposure and response prevention	Prolonged exposure to the obsessional trigger with strict prevention of the ritual	**60% to 85% improve among completers, about 75% hold the gain, refusal about 25% and dropout 3% to 12%**
Dialectical behaviour therapy and interpersonal psychotherapy	Acceptance balanced against change across mindfulness, distress tolerance, emotion regulation and interpersonal effectiveness; or the four interpersonal problem areas of grief, role dispute, role transition and interpersonal deficit	**Dialectical therapy targets emotion dysregulation and self-harm; interpersonal therapy treats the current relationship problem rather than the personality**

Culture and stigma. The treatment gap reported in the transcultural literature reaches about 95%. Stigma is knowledge, attitude and behaviour, that is ignorance, prejudice and discrimination.

Yoga is adjunctive, with benefit shown in depression, anxiety, obsessive-compulsive disorder and schizophrenia. The mechanism is framed as autonomic rebalancing, slow breathing at 2 to 4 breaths a minute raising vagal tone and central GABA.

Lifetime adverse events reach 22.7% in observational series, mostly minor musculoskeletal strain, against 2.2% in supervised trials. It is not a monotherapy in acute mania, catatonia or acute psychosis.

Sexual medicine. Psychogenic erectile dysfunction is sudden and situational with preserved nocturnal and morning erections and primary performance anxiety; organic disease is gradual and constant with diminished spontaneous erections and vascular or endocrine comorbidity, and most organic cases acquire secondary anxiety, so the two are not exclusive.

Dhat syndrome is distress attributed to semen loss: exclude infection and depressive or anxiety disorder, then give physiological psychoeducation and cognitive restructuring without dismissing the distress, and avoid repeated genitourinary investigation.

Gender dysphoria needs incongruence of at least 6 months, with at least 6 of 8 criteria in children including the core insistence and at least 2 of 6 in adolescents and adults, plus clinically significant distress: identity itself is not a disorder.

Under the Transgender Persons (Protection of Rights) Act 2019 the certificate of identity is issued by the District Magistrate, the screening committee of the 2016 draft having been dropped, and listed offences carry imprisonment between 6 months and 2 years with fine.

Sleep. Chronic insomnia disorder needs difficulty initiating or maintaining sleep, or early waking, on at least 3 nights a week for at least 3 months despite adequate opportunity; prevalence is about 6% to 10% of adults, and cognitive behavioural therapy for insomnia is first-line with or without psychiatric comorbidity.

Hypnotics are not routine monotherapy and are approved for about 4 to 5 weeks, and failure to remit within 7 to 10 days should prompt a search for apnoea, restless legs, depression, substance use or pain; in bipolar disorder sleep restriction is modified, because sleep loss can switch mood.

Narcolepsy type 1 needs excessive daytime sleepiness for at least 3 months, with either of two findings.

- **Route one.** Cataplexy plus a mean sleep latency of 8 minutes or less and at least 2 sleep-onset rapid eye movement periods.
- **Route two.** Cerebrospinal hypocretin-1 below 110 pg per mL, with the same findings.
- **Mechanism.** Autoimmune loss of hypothalamic orexin neurons, which is why dual orexin receptor antagonists are contraindicated in it.

Obstructive sleep apnoea is bidirectional with psychiatric illness and must be treated before hypersomnolence is attributed to depression or wake-promoting drugs are escalated; screen depression here on the two-item instrument, since longer scales inflate on fatigue and fragmented sleep.

TRAP Non-rapid-eye-movement parasomnia against rapid-eye-movement sleep behaviour disorder. The first arises from deep slow-wave sleep in the first third of the night, with automatic clumsy non-goal-directed behaviour, confusion, difficulty rousing and amnesia; the second arises from rapid eye movement sleep in the last third, with vigorous dream enactment, rapid full alertness and vivid coherent recall, and it is a prodromal marker of the alpha-synucleinopathies. The library marks this comparison unsourced, so it is given as clinical grammar without counts. Cataplexy is emotion-triggered with preserved consciousness and prompt recovery, which separates it from syncope and atonic seizure.

HOLD THIS

Name the level of the defence, the incompatible response in desensitisation, and the ritual prevention in exposure work. Yoga is adjunctive, dhat needs explanation rather than investigation, and eight minutes with two rapid-eye-movement onsets is the narcolepsy threshold.

WHAT EARNS THE MARKS

- **Naming the level, not just the defence**: the level is the answer in this station
- **Pairing mechanism with technique**: reciprocal inhibition, extinction, ritual prevention
- **Keeping yoga adjunctive**: not a monotherapy in acute mania, catatonia or acute psychosis
- **Treating apnoea before attributing hypersomnolence**: and screen mood on the short instrument here

SEE ALSO 25 Civil capacities 31 Women's mental health 33 Addiction and liaison

The whole picture: psychometrics, tests and scales	
PSYCHOMETRICS	Reliability caps validity at its square root. Alpha 0.70 exploratory, 0.80 good, 0.90 for an individual decision. Sensitivity and specificity belong to the test, predictive values to the population, likelihood ratios travel.
RISK SCALES	Pooled positive predictive value 5.5 percent for suicide; at a 0.5 percent base rate even 99 by 99 percent gives 33 percent. Needs assessment and means restriction, not a risk tier.
SCREENING AND DIAGNOSIS	Screening prioritises sensitivity, diagnosis specificity. Semi-structured needs a clinician; fully structured allows lay raters, 21 minutes against 92.
INTELLIGENCE AND DISABILITY	Deviation quotient mean 100, SD 15. Adult scale ages 16 to 90, 10 core plus 5 supplemental. Malin's ages 6 to 16, Binet-Kamat ages 3 to 22. Below 70 plus adaptive deficits plus developmental onset.
PERSONALITY	MMPI-2: 567 items, T mean 50 SD 10, 65 significant; validity scales first, Fp at 100 with consistency intact means over-reporting. Personality examination: 1 criterion before age 25 years.
PROJECTIVE AND NEUROPSYCHOLOGY	Ten plates, side-by-side seating, association then inquiry, Exner coding, defensible for formal thought disorder only. Bender copy parietal, recall temporal, Hain critical at 13 or more.
PSYCHOSIS	Thirty items as 7 plus 7 plus 16, floor 30, ceiling 210, response 20 percent after subtracting 30, brief 6-item remission below 14. The brief rating scale: 18 items over 2 to 3 days.
MOOD	Seventeen items for severity, 18 to 21 for subtype; remission 7 or less, response 50 percent. The change-sensitive scale: 10 items, 0 to 60, remission 0 to 8. No scale separates unipolar from bipolar depression.
ANXIETY AND OCD	Fourteen items 0 to 56; the 7-item screen 0 to 21, cut-off 10 at 89 and 82 percent, or 8 for breadth; the obsessive-compulsive scale 0 to 40, entry 16, response 25 percent, remission 12 or less; autism version 5 items 0 to 20.
SUBSTANCE	The 10-item screen runs 0 to 40: 8 to 15 hazardous, 16 to 19 harmful, 20 or above dependence, Indian cut-offs lower. Dependence questionnaire 16 or above buys detoxification; opiate withdrawal 5 or above buys buprenorphine.
MOVEMENT	Sum items 1 to 7, score activation-only movement a point lower, treat a 2-point change as meaningful. Schooler-Kane: 2 regions at 2 or 1 at 3 with 3 months exposure. Anticholinergics improve parkinsonism, worsen dyskinesia.
COGNITION	Thirty points, cut-off 24 or 26; the Hindi version cuts at 19 with no reading load; the Addenbrooke examination totals 100, 88 normal and below 84 possible dementia; the dementia rating runs 0 to 4 on informant report.

The whole picture: psychometrics, tests and scales	
DISABILITY AND FUNCTION	Four domains 0 to 16 plus duration 1 to 4, global 0 to 20, certification at 8 or above, that is 40 percent, on a board with at least 1 psychiatrist, certificate 5 years, reassessment every 2 years. The disability schedule: 6 domains over 30 days.
CHILD AND EATING	Toddler checklist 0 to 20, items 2, 5 and 12 reversed, 3 to 7 follow-up and 8 or above refer. Vanderbilt 6 of 9 plus impairment. Difficulties questionnaire 25 items, prosocial apart. Eating attitudes 0 to 78, three-armed referral index at 20.
The whole picture: law, community and the sub-specialties	
CAPACITY	Understand, appreciate, communicate; presumed and decision-specific. Mental illness alone is not unsoundness of mind without a court, Section 3(5).
ADMISSIONS	86 independent; 87 minors, two examiners; 89 supported, one psychiatrist plus one other, thirty days, capacity weekly; 90, two psychiatrists, ninety then 120 then 180 days; 94 emergency, seventy-two hours, no electroconvulsive therapy.
TREATMENT BARS	Section 95: unmodified electroconvulsive therapy, minors except on three conditions, sterilisation as treatment, chaining. Section 96 psychosurgery, personal consent plus Board. Section 97 bars seclusion; restraint is the sole means against imminent harm.
INSTITUTIONS	Authority registers, Board adjudicates. Six members, quorum three, seven days for a supported-admission challenge, twenty-one for extended, thirty days to the High Court.
DISABILITY	Forty per cent is benchmark; four per cent of posts, five per cent of seats with five years age relaxation; certificate appeal under Section 59, guardianship appeal under Section 14(3), discrimination to the Commissioner.
RESPONSIBILITY AND FITNESS	Section 84, now 22, tests cognition at the act; 328 and 329 test capacity at trial, with a two-member Medical Board on appeal. No diminished responsibility, no irresistible impulse.
CHILDREN AND DRUGS	POCSO: reasonable suspicion, immediate report, Section 19(7) immunity. Juvenile Justice: sixteen plus heinous, four parameters, expert advisory. NDPS: Section 64A immunity, Section 71 management.
CIVIL CAPACITIES	Banks v Goodfellow four limbs; Section 12 of the Contract Act and the lucid interval; marriage voidable not void; voting lost only on a court declaration.
COMMUNITY	Programme 1982, district programme 1996, seven cadres, ten beds, four satellite clinics a month; gap 84% in older adults; under one per cent of the health budget.
REHABILITATION AND OUTREACH	Place then train, zero exclusion, employer contact in thirty days; assertive treatment is a shared caseload,

The whole picture: law, community and the sub-specialties	
	in vivo, round the clock, cutting admissions rather than unemployment.
CHILD AND ADOLESCENT	Six symptoms to sixteen years, five from seventeen, onset before twelve, two settings; behaviour management first under six; refusal is anxious and known, truancy is concealed.
WOMEN AND GERIATRIC	Five of eleven over two prospective cycles with days six to ten quiet; luteal dosing works in days. Forty to sixty late-onset, after sixty partition delusions; quarter dose; antipsychotic mortality odds 1.6.
ADDICTION AND LIAISON	Alcohol 14.6%, opioids 2.1%, treatment 0.55%; lapse against relapse through the abstinence violation effect; delirium is 31.7% of consultations; diabetes distress does not answer to drugs.
THERAPY, CULTURE, SEX, SLEEP	Four defence levels; reciprocal inhibition against extinction against ritual prevention; yoga adjunctive only; three nights for three months defines insomnia; eight minutes with two onsets defines narcolepsy.

HOLD THIS

Every answer in this note has one shape: name the instrument, the criterion or the section, give the number, then give the one discriminator that separates it from its near neighbour. Say what the instrument measures, how it is scored, what the cut-off does, and what it cannot do. Where the number is not in your sources, say so and say where you will read it: an honest gap with a plan to check it costs less than a confident wrong number.

PART IX

IX

Viva Tables IV: General Psychiatry, Recent Advances, Pedagogy; Spotters

Classification and guidelines, the recent advances and trials, the numbers, history, ethics and teaching questions, and the spotters.

01 Classification and guidelines

02 Recent advances and landmark trials

03 Epidemiology, history, ethics, research and pedagogy

04 Spotters

Viva Tables IV: General Psychiatry, Recent Advances, Pedagogy; Spotters

Four sub-areas · 25 topics · the table where breadth is tested

This is the table with the widest mouth. The examiner can open with the year a classification came into force, move to a trial acronym, then to a number you either hold or do not, then to a drug strip, and then to a photograph.

Breadth here is a memory problem, not a reasoning problem, so the material below is arranged as the examiner asks it: the instrument first, then the change, then the discriminator, then the one line to hold.

Where the library carries a question but not its number, the question is printed and the number is not. A figure supplied from memory at this table is the one thing that is worse than saying you will confirm it.

HOW TO USE THIS NOTE

Every topic runs in the same five parts: the examiner's question, the shape of the answer, the body, what earns the marks, and links to related topics.

Inside the body, read each topic as a ladder. The first line is what you say in the first ten seconds. The grid is what you say if pushed. The trap is what loses the mark.

A trap that says "verify before the exam" marks a question examiners ask reliably and a figure the library could not source. It is a deliberate gap for you to close from the Act, the protocol, or a standard text, not an omission.

Classification and guidelines

Classification questions reward a candidate who separates architecture from content. Say what kind of change it is (a coding change, a new grouping, a deleted subtype, a shift from categories to dimensions) before you list the entries.

1 · The ICD-11 architecture and what changed

CLASSIFICATION · TOPIC 01

What is new in ICD-11, and when did it come into force?

VIVA TABLE: CLASSIFICATION, AND THE OPENING QUESTION OF THE RECENT ADVANCES TABLE

THE SHAPE OF THE ANSWER

1	**The three dates**	Released 2018, approved 2019, statistics from January 2022
2	**Architecture, not content**	Coding change first, then the 21 groupings it allows
3	**Guidelines, not criteria**	Essential features of a typical presentation, not symptom counts
4	**Why it was built this way**	Clinical utility and global applicability, tested in a clinician network
5	**Where India stands**	ICD-10 still codes the record, so speak both

Give the three dates first. The pre-final version of ICD-11 was released to 194 World Health Organization member states in June 2018. The World Health Assembly formally approved it in May 2019. Member state reporting of health statistics using the new system began on January 1, 2022.

- **The scale of the revision.** It is the first comprehensive overhaul of the global mental disorders classification in nearly 30 years.

Development was led by the World Health Organization Department of Mental Health and Substance Abuse over more than a decade. It coordinated **4 related chapters**: mental, behavioural and neurodevelopmental disorders; sleep-wake disorders; diseases of the nervous system; and conditions related to sexual health.

- **Why utility led.** Low- and middle-income countries account for more than 80% of the world population, so clinical utility and global applicability were the organizing principles rather than performance in specialist academic centres.

The evidence base, if asked how it was tested. The Global Clinical Practice Network enrolled nearly 15,000 clinicians from 155 countries, more than 50% of them physicians and 30% psychologists. International clinic-based studies ran in 13 to 14 countries across World Health Organization regions.

FEATURE	ICD-10	ICD-11	THE DISCRIMINATOR
Coding	Rigid decimal format, F00 to F99	Flexible alphanumeric stem codes	**The decimal format capped the chapter at a maximum of 10 major groupings, which forced distinct conditions into heterogeneous blocks**
Number of groupings	Capped at 10	21 distinct diagnostic groupings	**Say 21 and then name catatonia as the grouping that was promoted to that tier**
Chapter order	Not developmental	Developmental lifespan continuum	**Neurodevelopmental disorders open the chapter, neurocognitive disorders close it**
Diagnostic rules	Diagnostic criteria with symptom counts	Clinical Descriptions and Diagnostic Guidelines with essential features	**Thresholds and symptom counts are avoided unless empirically supported across cultures**
Added guidance	Minimal	Lifespan and culture-related guidance throughout	**Presentations in children, adolescents and older adults are described, and clinical phenomena are linked to local concepts of distress**

Architecture first, content second: the coding change is what makes the 21 groupings possible.

RULE ICD-11 characterises disorders through **essential features** a clinician expects in a typical presentation, not through operationalised symptom counting. Saying "criteria" for ICD-11 where the manual says "guidelines" is the small error that tells an examiner you have not opened it.

WHERE INDIA ACTUALLY STANDS

ICD-10 remains the active administrative and documentation standard in state health departments, teaching hospitals and university medical colleges: outpatient records, discharge summaries, hospital information management systems and postgraduate logbooks all use ICD-10 codes.

Statutory notification dates and regulatory deadlines for full digital migration are not carried in the library sources, so do not quote one. Indian centres and investigators, including faculty from premier national institutes, contributed substantially to the ICD-11 development committees and the international field trials.

TRAP Presenting an ICD-11 diagnosis into a case sheet that codes in ICD-10. State the formal diagnosis in ICD-10 nomenclature to match the record in front of you, then give the ICD-11 formulation immediately, with its dimensional qualifiers and trait domains. Fluency in both is what reads as recent-advances competence.

Trap. Calling the ICD-11 rules criteria. The manual gives guidelines, and the word you choose signals whether you have read it.

HOLD THIS

Approved May 2019, statistics from January 2022, 21 groupings in place of a decimal cap of 10. Essential features, not symptom counts; lifespan and culture guidance throughout.

WHAT EARNS THE MARKS

- **Dating the instrument correctly**: approval in May 2019 is separate from statistical reporting from January 2022
- **Naming the coding change before the content**: the decimal cap of 10 is why 21 groupings needed a new code format
- **Using the word guidelines**: essential features of a typical presentation, not operationalised counts
- **Coding in ICD-10 and formulating in ICD-11**: it matches the record in front of you and shows both

SEE ALSO 02 New entries and the dimensions 03 DSM-5-TR and the debate 04 Indian Psychiatric Society guidelines

2 · New entries, deleted categories, and the dimensions

CLASSIFICATION · TOPIC 02

Which diagnoses are new in ICD-11, and which categories were taken out?

VIVA TABLE: CLASSIFICATION, AND ANY SHORT CASE WHERE THE DIAGNOSIS HAS MOVED GROUPING

THE SHAPE OF THE ANSWER

1 What is new	Name the entry and the grouping it sits in, not a bare list

2	**What is gone**	Subtypes, somatoform, conversion, the organic dichotomy
3	**What became dimensional**	Psychosis qualifiers, personality severity then trait domains
4	**The threshold changes**	Depressive episode 5 of 10, entry by mood or interest
5	**The traps**	No anankastic personality disorder, and no search for unexplained symptoms

Answer in three lists, in this order: what is new, what is gone, what became dimensional. Candidates who mix them produce a shapeless answer to a question that has a shape.

NEW ENTRY	WHERE IT SITS	THE DISCRIMINATOR THE EXAMINER LISTENS FOR
Catatonia	An independent grouping at the same tier as mood and psychotic disorders	**3 conditions: associated with another mental disorder, substance or medication induced, and secondary to a medical condition. It is no longer a schizophrenia subtype or an organic category**
Bipolar type II disorder	Mood disorders	**At least 1 hypomanic and at least 1 major depressive episode, with no history of a manic episode. A single lifetime manic or mixed episode confers type I**
Body dysmorphic disorder	Obsessive-compulsive and related disorders	**Moved out of the ICD-10 inclusion under hypochondriasis; preoccupation with slight or unnoticeable flaws plus checking or camouflaging**
Olfactory reference disorder	Obsessive-compulsive and related disorders	**The belief of emitting an odour barely noticeable to others, with checking, camouflaging and avoidance**
Hoarding disorder	Obsessive-compulsive and related disorders	**Accumulation from excessive acquisition or difficulty discarding, with clutter that compromises safety and function**
Excoriation disorder	Body-focused repetitive behaviour disorders, within the obsessive-compulsive grouping	**Sits alongside trichotillomania, not with the self-harm entries**
Complex post-traumatic stress disorder	Disorders specifically associated with stress	**The 3 core features of post-traumatic stress disorder plus disturbances of affect regulation, negative self-concept and relational capacity. Severity of the event alone does not confer it**

NEW ENTRY	WHERE IT SITS	THE DISCRIMINATOR THE EXAMINER LISTENS FOR
Prolonged grief disorder	Disorders specifically associated with stress	**Longing or preoccupation with the deceased and intense emotional pain persisting beyond 6 months, clearly exceeding cultural or religious norms**
Binge eating disorder	Feeding and eating disorders	**Loss of control eating at 1 or more times per week over several months, without regular compensatory behaviours**
Avoidant or restrictive food intake disorder	Feeding and eating disorders	**Insufficient intake with weight loss, deficiency or supplement dependence, and no body shape or weight distortion; applies across the lifespan**
Body integrity dysphoria	Disorders of bodily distress and bodily experience	**A persistent desire to acquire a specific physical disability, beginning in childhood or early adolescence**
Gaming disorder	Disorders due to addictive behaviours	**Impaired control, gaming taking precedence over life interests, escalation despite consequences; online or offline**
Compulsive sexual behaviour disorder	Impulse control disorders	**Failure to control intense sexual impulses over 6 months or more with marked distress. It is an impulse control disorder here, not a paraphilic or addictive one**
Intermittent explosive disorder	Impulse control disorders	**Brief episodes of aggression grossly out of proportion to provocation**
Premenstrual dysphoric disorder	Primary location in the genitourinary chapter, cross-listed in depressive disorders	**Symptoms begin days before menses and remit within 1 week after, across a majority of cycles in the past 1 year. The cross-listing is the point**

The new entries, each with the line that separates it from the category it came out of.

What was removed, and why. Take the deletions as six moves, each with a reason attached, because the reason is what the examiner follows up on.

- **Psychosis.** The schizophrenia subtypes (paranoid, hebephrenic, catatonic, undifferentiated, post-schizophrenic depression, residual, simple) were eliminated for failing to predict treatment response or course, and are replaced by 6 dimensional qualifiers. Schneiderian first-rank symptoms are de-emphasised for lack of diagnostic specificity.
- **Acute and transient psychotic disorder.** Only the polymorphic form is retained, with a duration limit of 3 months. Non-polymorphic presentations code as other primary psychotic disorder.

- **The somatic group.** Somatoform disorders and neurasthenia are replaced by bodily distress disorder, and hypochondriasis moves to the obsessive-compulsive grouping.
- **Conversion is discarded.** The entry is dissociative neurological symptom disorder, carrying 12 presentation-specific subtypes. Acute stress reaction is de-listed as a mental disorder and relocated to factors influencing health status.
- **Personality.** The 10 categorical personality disorder subtypes are gone, replaced by the two-step dimensional scheme in the grid below.
- **The organic dichotomy is dismantled.** Non-organic sleep disorders move to a standalone sleep-wake chapter and non-organic sexual dysfunctions to a unified sexual health chapter.
- **Sexuality and gender.** Gender identity disorders are renamed gender incongruence and transferred out of mental disorders. Fetishism, fetishistic transvestism and sadomasochism are removed, and paraphilic disorders are restricted to behaviours involving non-consenting individuals, intense distress, or substantial risk of injury or death.

DIMENSIONAL DEVICE	STRUCTURE	THE DISCRIMINATOR
Primary psychotic disorder qualifiers	6 symptom dimensions rated present or absent: positive, negative, depressive, manic, psychomotor, cognitive	**They apply across schizophrenia, schizoaffective disorder, delusional disorder and acute and transient psychotic disorder, not to schizophrenia alone**
Personality disorder severity	3 levels: mild, moderate, severe, graded on self and interpersonal functioning	**Subthreshold features that complicate clinical contact are "personality difficulty" under factors influencing health status, not a disorder**
Personality trait domains	5 domains: negative affectivity, detachment, dissociality, disinhibition, anankastia	**As many as are prominent are assigned; a borderline pattern qualifier is retained to preserve access to specialised psychotherapy protocols**
Disorders of intellectual development	Behavioural indicator tables across 3 domains and 3 age groups, graded into 4 severity levels	**The rename from mental retardation is the headline; the indicator tables are what earns the follow-up**
Schizoaffective disorder	Concurrent schizophrenia syndrome and a mood episode in the presenting episode	**Conceptualised as an episodic state, not a longitudinally stable illness**

Mood, anxiety and substance use in one pass. These are the threshold changes an examiner can check in a single question, so hold the numbers and the entry rules.

- **Depressive episode.** At least 5 of 10 symptoms for at least 2 weeks, where ICD-10 required 4 of 9. The symptoms group into 3 clusters: affective, cognitive and neurovegetative.
- **The entry rule changed.** Fatigue no longer suffices as an entry symptom; mandatory entry is depressed mood or diminished interest. Hopelessness is added as a cognitive symptom.
- **Mania and the persistent group.** A manic episode requires increased activity or subjective energy alongside mood elevation or irritability, and a mixed episode indicates bipolar type I. The persistent mood disorders grouping is eliminated: dysthymia moves to depressive disorders, cyclothymia to bipolar disorders.

- **Anxiety is grouped by focus of apprehension.** The hierarchical rules that prevented co-diagnosis of generalised anxiety disorder with depression or obsessive-compulsive disorder are removed.
- **Two redefinitions.** Agoraphobia is fear of situations where escape is difficult or help unavailable because of incapacitating or embarrassing symptoms. Panic disorder is reserved for unexpected attacks, with a "with panic attacks" qualifier available on a parent disorder.
- **The lifespan move.** Separation anxiety disorder and selective mutism move into anxiety disorders.
- **Harmful substance use splits in two.** A single episode of harmful use, and a harmful pattern of use. Harm now includes harm caused to other people.

THREE TRAPS IN ONE BLOCK

Anankastic personality disorder: there is no such ICD-11 diagnosis. Diagnose mild, moderate or severe personality disorder and assign the anankastia trait domain. **Bodily distress disorder:** it does not require symptoms to be medically unexplained, so hunting for a negative workup to confirm it inverts the definition. **Fixed beliefs in obsessive-compulsive disorder:** when the belief is entirely consistent with the disorder's own themes, assign the "with poor to absent insight" qualifier rather than diagnosing delusional disorder.

HOLD THIS

Catatonia up, subtypes out, personality dimensional in two steps. Depressive episode: 5 of 10 for 2 weeks, entry by mood or interest, not by fatigue.

WHAT EARNS THE MARKS

- **Placing each new entry in its grouping**: gaming disorder under addictive behaviours, compulsive sexual behaviour under impulse control
- **Giving the reason a category went**: the schizophrenia subtypes did not predict response or course
- **Doing personality in two steps**: severity first, then as many trait domains as are prominent
- **Quoting the depressive threshold as 5 of 10 over 2 weeks**: with entry by mood or interest, not fatigue

SEE ALSO 01 The ICD-11 architecture 03 DSM-5-TR and prolonged grief 04 Indian Psychiatric Society guidelines

3 · DSM-5-TR, prolonged grief, and the DSM versus ICD debate

CLASSIFICATION · TOPIC 03

What did DSM-5-TR add, and how does its prolonged grief disorder differ from the ICD-11 entry?

VIVA TABLE: CLASSIFICATION, AND THE BEREAVEMENT LONG CASE

THE SHAPE OF THE ANSWER

1	Name the kind of revision	An interim text revision of 2022, not a structural overhaul
2	The lineage of the entry	Section III in 2013, renamed into Section II
3	The criteria side by side	Time since loss, core distress, accessory threshold, what is timed
4	Why prevalence differs	One accessory symptom in ICD against three in DSM
5	Management and culture	Psychotherapy first, and the cultural exceedance requirement

Name the kind of revision before the content. The American Psychiatric Association released the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision in March

2022. It is an interim text revision, not a structural overhaul like DSM-5 itself, which appeared in 2013.

- **What the revision did.** It updated clinical text, modernised terminology, corrected inconsistencies, refined codes across categories, and revised the text on culture, racism and discrimination. Its chief diagnostic addition is prolonged grief disorder, approved into Section II.

The lineage matters more than the label. In DSM-5 the entity sat in Section III, conditions for further study, as persistent complex bereavement disorder. After roughly a decade of field validation and multi-site work, and a review and public comment process spanning 2 years from early 2020 through 2021, it moved into Section II and was renamed.

- **Where it codes.** It maps to the ICD-10 clinical modification code F43.8, other reactions to severe stress, and sits in the trauma- and stressor-related disorders chapter.

CRITERION	DSM-5-TR	ICD-11	THE DISCRIMINATOR
Model	Operationalised polythetic algorithm	Typological clinical descriptive guidance	**DSM prioritised reliability and homogeneous trial cohorts; ICD prioritised utility in primary care and cross-cultural use in low- and middle-income countries**
Time since loss	At least 12 months in adults, at least 6 months in children and adolescents	An atypically long period, more than 6 months at a minimum, for all age groups	**The 6-month threshold in DSM belongs to youth only; quoting it for adults is the commonest error at this station**
Core separation distress	At least 1 of 2: intense yearning or longing, or preoccupation with thoughts or memories	Persistent pervasive longing or persistent preoccupation with the deceased	**In children the preoccupation often centres on the circumstances of the death**
Accessory symptoms	At least 3 of 8	At least 1 of 10 illustrative symptoms, with intense emotional pain	**This single difference drives the prevalence gap between the two manuals**
Symptom duration	Present most days, nearly every day, for at least the last 1 month	The grief response persists for at least 6 months	**DSM times the symptoms; ICD times the response. Counting only elapsed time since the death inflates the diagnosis**
Item composition	Trouble accepting the loss is excluded; avoidance of reminders is an explicit accessory item	Difficulty accepting the death is included; avoidance is not a numbered accessory item	**The single most asked detail: candidates list trouble accepting the loss among the DSM accessory items, and it was removed**

CRITERION	DSM-5-TR	ICD-11	THE DISCRIMINATOR
Culture	Duration and severity clearly exceed expected social, cultural or religious norms	The same requirement, placed one criterion earlier	**Both manuals carry it; only the criterion letter differs**

The two criteria sets side by side, with the accessory-symptom threshold as the hinge.

The numbers behind the debate. Applying the minimal ICD-11 threshold produces higher diagnostic rates than DSM-5-TR. Three figures carry the point.

- **Conditional point prevalence among bereaved adults.** 4.7% by DSM-5-TR against 5.4% by ICD-11 with 6-month symptom persistence, rising to 6.8% when only elapsed time since the loss was counted.
- **Population-wide figures.** 2.0%, 2.3% and 2.9% respectively, in the same order.
- **Agreement between the two sets.** Substantial, kappa 0.75 with a 95% confidence interval of 0.66 to 0.85, and 3.9% of the bereaved sample met both. Raising the ICD-11 accessory requirement from 1 to 3 symptoms brings concordance to kappa 0.79 and aligns prevalence at 4.7%.

In bereaved adult populations, estimated rates of the disorder range between 4% and 15%.

- **Documented risk factors.** Sudden, unexpected or unnatural death, violent circumstances, loss of a child or spouse, death in intensive care rather than home hospice, older age, female sex, lower socioeconomic status, anxious attachment, emotional dependency on the deceased, high pre-loss grief, a prior depressive disorder, absent social support, and suicidal ideation.

Treatment, in one line each. First-line management is psychotherapy.

- **Prolonged grief therapy.** The cognitive-behavioural approach developed by Shear and colleagues, built around two processes: adapting to the loss, and restoring engagement with goals, relationships and identity.
- **Cognitive-behavioural therapy.** It restructures maladaptive grief cognitions and delivers narrative exposure through structured writing; cognitive-behavioural therapy for insomnia is a useful targeted adjunct.
- **In young people.** Developmentally adapted protocols show symptom reduction in bereaved children and adolescents in randomised trials.
- **Medication.** No pharmacological agent holds regulatory approval for the disorder. Comorbid depression or anxiety is treated on its own terms.

THE CULTURAL EXCEEDANCE CRITERION IN INDIAN PRACTICE

Indian mourning involves culturally structured community rituals across faiths, with extended post-death observances, community gatherings and annual remembrance ceremonies. Intense weeping, transient dissociation and temporary disengagement during those customary observances are normative expressions of grief.

The diagnosis is not assigned when symptoms are culturally congruent. The threshold is crossed only when impairment is severe and persistent and the distress remains unremitting well beyond accepted community mourning customs.

TRAP Saying the disorder was created from scratch in the text revision. It was developed by refining and renaming persistent complex bereavement disorder, which had sat in Section III since 2013. Separately, the intrusion discriminator against post-traumatic stress disorder: grief intrusions centre on separation, love, sorrow and yearning for the person; trauma intrusions centre on the incident, terror and horror.

HOLD THIS

12 months in adults, 6 months in youth, 1 of 2 core and 3 of 8 accessory symptoms over the last 1 month. ICD-11 asks 1 of 10, which is why its prevalence runs higher.

WHAT EARNS THE MARKS

- **Keeping the two time rules apart**: 12 months in adults, 6 months only in children and adolescents
- **Naming what each manual times**: DSM times the symptoms, ICD times the response
- **Explaining the prevalence gap mechanically**: 1 accessory symptom against 3
- **Applying the cultural exceedance requirement**: a culturally congruent mourning pattern is not the disorder

SEE ALSO 02 New entries and the dimensions 12 The epidemiology numbers 15 Research methodology

4 · Indian Psychiatric Society guidelines and how to cite them

CLASSIFICATION · TOPIC 04

Which guideline are you following for this patient, and why that one?

LONG CASE DEFENCE, AND THE GUIDELINES VIVA TABLE

THE SHAPE OF THE ANSWER

1 **Indian guideline first**	Name the Society guideline, then the international one
2 **Why context changes the choice**	Family as caregiver, out of pocket cost, Indian statute, comorbidity
3 **Know the recent drafts**	Four drafts, by release date rather than label year
4 **The four-part citation**	Problem, guideline, recommendation, legal and contextual justification
5 **The caveat**	Guidelines assist decisions, they do not replace judgment

Cite the Indian guideline first and the Western one second. International guidelines are developed for systems with third-party payers, high institutional resources and different law.

- **Why the setting changes the recommendation.** In Indian practice the family is caregiver, co-therapist and financial provider. Costs are largely out of pocket, so generic availability decides adherence.
- **Two more reasons.** Practice must conform to Indian statute, and tropical comorbidity, endemic infection, nutritional deficit and metabolic variation shape drug tolerance.

Citing an Indian guideline shows you know the standard of care that actually applies at the bedside you are standing at.

How the series is produced. The Indian Psychiatric Society generates clinical practice guidelines through its guideline committee, specialty sections and expert task forces. Drafts are placed before fellows and members for critical suggestions on the Society web portal, and are then published as dedicated special supplements in the Indian Journal of Psychiatry.

- **What earlier cycles covered.** Standardised management algorithms for schizophrenia, bipolar affective disorder, depressive disorders, substance use disorders, obsessive-compulsive disorder, child and adolescent disorders and geriatric conditions.

DRAFT GUIDELINE	RELEASED	CLINICAL SCOPE	THE DISCRIMINATOR
Consultation-liaison psychiatry	December 2021	Psychiatric disorders in general hospital medical and surgical wards, delirium, somatoform presentations, transplant liaison	**The Indian ward reality it addresses: organophosphate poisoning, metabolic encephalopathy and alcohol withdrawal on general medical wards**
Psychiatric emergencies and brain stimulation techniques	October 2022	Rapid tranquillisation, suicide risk evaluation, catatonia protocols, modified electroconvulsive therapy, repetitive transcranial magnetic stimulation, transcranial direct current stimulation	**Connect it immediately to the statutory bar on unmodified electroconvulsive therapy and to consent under Indian law**
Mental health and wellbeing	October 2023	Preventive interventions, stress management, workplace and school mental health, positive psychological strategies	**This is the guideline to cite for a promotion or prevention question, not a treatment guideline**
Cognition, labelled as the 2025 guideline	November 2024	Neurocognitive impairment across schizophrenia, mood disorders, mild cognitive impairment, dementias, cognitive rehabilitation	**The label year and the release year differ, and saying so is the mark of someone who has seen the portal notice**

The four recent drafts, by release date rather than by label year.

THE FOUR-PART CITATION, SAID OUT LOUD

Use the same shape every time, so it is available under pressure:

1. Name the clinical problem: "for managing this patient with treatment-resistant depression".
2. Name the Indian consensus: "as per the Indian Psychiatric Society guidelines on depressive disorders".
3. State the operational recommendation: the augmentation strategy, the modality, the monitoring.
4. Add the contextual and legal justification: baseline renal and thyroid parameters, feasibility of monitoring, affordability, family supervision, and the statutory consent route.

For an emergency question, the same shape carries the emergency draft guideline, verbal de-escalation and nursing in a quiet environment first, then parenteral rapid tranquillisation with close haemodynamic monitoring.

RULE Guidelines are systematically developed patient-care strategies intended to assist decision-making. They are not rigid standards of care and they do not replace individualised clinical judgment. Say that once, then defend your plan on its merits.

ESSENTIAL MEDICINES AND THE PROGRAMME FRAME

Psychopharmacological selection in a government setting reflects the National List of Essential Medicines, which prioritises agents such as chlorpromazine, haloperidol, risperidone, amitriptyline, fluoxetine, sodium valproate and lithium.

Community delivery runs through the District Mental Health Programme, with satellite clinics at community and primary health centres. Financial toxicity translates directly into non-adherence, so cost is a clinical variable, not an administrative one.

TRAP Two errors that cost marks here. Treating a draft posted on the Society portal for professional input as a finalised journal supplement. And quoting an international guideline without knowing whether the recommended agent is available and affordable in India, which invites the follow-up you cannot answer.

HOLD THIS

Problem, Indian guideline, operational recommendation, legal and contextual justification. Four beats, in that order, every time.

WHAT EARNS THE MARKS

- **Leading with the Indian guideline**: it is the standard of care at the bed you are standing at
- **Dating the drafts by release**: the cognition guideline is labelled 2025 and was released November 2024
- **Adding the legal and cost justification**: monitoring feasibility, affordability, family supervision, consent route
- **Stating the caveat once**: guidelines assist decision-making and do not replace individualised judgment

SEE ALSO 01 The ICD-11 architecture 14 Ethics and the codes 17 Psychiatric emergencies

Recent advances and landmark trials

Recent advances are tested as mechanism, then evidence, then availability. Candidates lose marks by describing a mechanism they cannot attach to a trial, or by implying an agent is available in India when the question of Indian approval has not been checked.

5 · Schizophrenia beyond dopamine D2 blockade

RECENT ADVANCES · TOPIC 05

What is new in antipsychotic pharmacology that does not work through D2 blockade?

VIVA TABLE: RECENT ADVANCES, AND THE DRUG SPOTTER FOLLOW-UP

THE SHAPE OF THE ANSWER

1 **Why the field moved**

	D2 antagonism misses negative symptoms and cognition
2 The muscarinic route	Central M1 and M4 agonism with peripheral blockade
3 The trace amine route	Upstream reduction of dopaminergic tone, not postsynaptic
4 The glutamatergic frame	Interneuron hypofunction drives subcortical dopamine excess
5 Evidence and availability	Quote the trial, then declare the Indian availability gap

Open with why the field moved. For more than 60 years antipsychotic pharmacology has rested on postsynaptic dopamine D2 antagonism. It works for positive symptoms in approximately 70% of individuals.

- **The cost of that mechanism.** Extrapyramidal and cardiometabolic burden and raised prolactin, with poor performance against primary negative symptoms and cognitive deficit. The new mechanisms are attempts to reach those two domains without occupying D2.

AGENT	TARGET	STATUS	THE DISCRIMINATOR THE EXAMINER LISTENS FOR
Xanomeline with trospium chloride	Central muscarinic M1 and M4 preferential agonism, with peripherally restricted muscarinic antagonism	The first approved non-dopaminergic antipsychotic mechanism, approved late in 2024	**M4 activation on striatal and midbrain neurons inhibits presynaptic dopamine release without blocking postsynaptic D2; M1 activation in prefrontal cortex supports cognition; trospium does not meaningfully cross the blood-brain barrier and exists only to block peripheral cholinergic effects**
Ulotaront	Full trace amine-associated receptor 1 agonist with 5-HT1A agonism	Breakthrough Therapy designation in May 2019; oral once daily, 25 mg to 75 mg	**It has no meaningful binding at postsynaptic D2 or 5-HT2A, so it is not a third-generation partial agonist. It reduces dopaminergic tone from upstream, inside ventral tegmental neurons**
Ralmitaront	Partial trace amine-associated receptor 1 agonist	Phase 2 trials completed in 2023	**Partial rather than full agonism, and no 5-HT1A activity; that pair separates it from ulotaront**

AGENT	TARGET	STATUS	THE DISCRIMINATOR THE EXAMINER LISTENS FOR
Iclepertin	Glycine transporter 1 inhibition, raising synaptic glycine at the N-methyl-D-aspartate co-agonist site	Evaluated in phase 3 at 2 mg to 25 mg daily for cognitive impairment; did not achieve improvement on the cognition rating scale	**A negative trial you are expected to know as negative**
Lumateperone	Dopamine D2 and D1, serotonin 5-HT2A, and indirect facilitation of N-methyl-D-aspartate receptor phosphorylation	Administered as a 42 mg capsule daily at bedtime	**Multi-target monoaminergic, so it does not belong in the non-dopaminergic list**
Roluperidone	5-HT2A and alpha-1a adrenergic antagonism	Evaluated at 32 mg to 64 mg daily for negative symptoms	**Negligible extrapyramidal, prolactin or metabolic effect in trial settings, which is the reason it is being developed for negative symptoms**

The pipeline by mechanism, with the line that keeps each agent out of the wrong class.

The ulotaront trial data, if pressed. Give the acute trial, then the extension, then tolerability.

In an acute 4-week phase 2 randomised controlled trial of 245 adults with an acute exacerbation, ulotaront at 50 mg or 75 mg once daily produced a reduction in total Positive and Negative Syndrome Scale score of 17.2 points. That was a treatment difference of 7.5 points against placebo, with a 95% confidence interval of 3.0 to 11.9.

The Brief Negative Symptom Scale change was 7.1 points, a difference of 4.3 points with a 95% confidence interval of 1.8 to 6.8.

In the 26-week open-label extension of 157 enrolled patients on flexible doses of 25 mg, 50 mg or 75 mg, the further change on the syndrome scale was 22.6 points and on the negative symptom scale 11.3 points. Study completion was 67%, against historical completion rates of 39% to 65% for atypical antipsychotics over the same span.

Adverse event frequency was 45.8% for ulotaront against 50.4% for placebo, and extrapyramidal adverse event rates were 3.3% against 3.2%. There was no clinically meaningful change in weight, body mass index, fasting lipids, glucose, glycated haemoglobin or prolactin.

The glutamatergic frame underneath all of it. Hypofunction of the N-methyl-D-aspartate receptor complex on cortical parvalbumin-positive GABAergic interneurons disinhibits glutamatergic pyramidal projections and produces secondary subcortical dopaminergic hyperfunction.

- **Why that frame matters.** Every agent above is an attempt to restore physiological tone somewhere on that loop without excitotoxicity.

Two more advances the examiner may reach for. Both are delivery advances rather than molecules.

- **Prescription digital therapeutics.** These have moved from tracking apps to regulated software interventions. A smartphone therapeutic for negative symptoms targeting avolition and motivational deficit met its primary endpoint in a phase 3 trial and holds a breakthrough device designation, and a reasoning-based mobile therapeutic for paranoia and persecutory ideation shows benefit added to standard care.
- **Accelerated intermittent theta-burst stimulation.** Directed to dorsomedial prefrontal cortex, it reduces negative symptoms and cognitive deficit with good tolerability and documented low seizure risk.
- **On the biomarker side.** Peripheral exosomal microRNA signatures analysed with ensemble algorithms discriminate psychosis risk states, and polygenic risk scores combined with real-world phenotype are being tested to predict treatment trajectory including clozapine responsiveness.

TRAP Expecting atropinic toxicity from the muscarinic combination. Xanomeline stimulates central M1 and M4 while trospium blocks peripheral muscarinic receptors, so the common adverse effects are gastrointestinal, nausea, dyspepsia, constipation and vomiting, without the drug-induced parkinsonism, akathisia or metabolic dysregulation of D2 antagonists.

- **Trap.** Availability. Whether any of these agents is approved, marketed or listed on the National List of Essential Medicines in India is not established in the library sources. Treat Indian availability as an open question and say so; verify before the exam rather than inferring it from the trial data.

HOLD THIS

M4 dampens dopamine release without touching D2; trace amine receptor 1 agonism does it from inside the ventral tegmental neuron. Neither is a partial D2 agonist.

WHAT EARNS THE MARKS

- **Locating the mechanism on the loop**: presynaptic or upstream, rather than postsynaptic D2
- **Explaining what trospium is for**: peripheral blockade only, because it does not meaningfully cross the barrier
- **Knowing the negative trial as negative**: iclepertin did not improve the cognition rating scale
- **Declaring the Indian availability gap**: an unsourced availability claim invites a follow-up you cannot answer

SEE ALSO 06 Clinical high risk and early intervention 11 The landmark trials 18 Neurobiology at the table

6 · Clinical high risk, early intervention, untreated psychosis

RECENT ADVANCES · TOPIC 06

This young person is at clinical high risk of psychosis. What do you do, and what do you not do?

VIVA TABLE: RECENT ADVANCES, AND THE EARLY PSYCHOSIS SHORT CASE

THE SHAPE OF THE ANSWER

1 Define the state	One of three validated clusters, and it is a risk state
2 Give the trajectory	Most do not transition within two years, and half of those remit
3 The instrument	Structured interview, four dimensions, formal training
4 What you offer	Therapy, family support, monitoring, not an antipsychotic

5 If it converts Shorten the untreated interval, then coordinated specialty care

Define the state, then refuse to treat it as a diagnosis. Epidemiological estimates place incident psychotic disorder at between 15 and 100 individuals per 100,000 each year.

- **What high risk means.** It is defined by at least one of three validated syndrome clusters, and the point of identifying it is psychological support before functional decline, not early antipsychotic exposure.

SYNDROME CLUSTER	WHAT IT REQUIRES	THE DISCRIMINATOR
Attenuated psychotic symptoms	Subthreshold positive symptoms, suspiciousness, perceptual distortion, unusual thought content	**Insight is intact and conviction, frequency or severity fall short of a full syndrome**
Brief intermittent psychotic symptoms	Fleeting fully psychotic positive symptoms resolving spontaneously, up to 3 months, lasting less than 1 hour per day	**Fully psychotic but self-limiting; on the structured interview at least one positive item is rated at the top severity anchor**
Genetic risk and deterioration	A first-degree relative with a psychotic disorder or schizotypal personality disorder, plus a significant functional drop	**The exact percentage decline in functioning is not carried in the library sources, so describe the drop and do not quote a figure**

The two-year trajectory, which is the whole point. Prospective follow-up shows that 73% of young people identified at clinical high risk do not transition to full psychosis within the first 2 years, which puts conversion at roughly 27%.

Among the 73% who do not transition, nearly half achieve complete symptomatic remission of their attenuated positive symptoms.

- **The argument against discharge.** Even among remitted non-converters, many continue to carry deficits in social interaction, affective symptoms and role functioning, so engagement continues.

The assessment instrument. The Structured Interview for Psychosis-risk Syndromes, which incorporates the Scale of Psychosis-risk Symptoms, is the primary evaluation standard. It is a semi-structured clinician-rated interview requiring formal training, taking between 30 minutes and 2 hours.

- **How it is administered.** Selected questions are delivered verbatim with flexible probing, to separate overvalued ideas from delusional conviction. It rates four dimensions: positive, negative, disorganisation and general symptoms.
- **The research consortium.** On the international side, the Accelerating Medicines Partnership for Schizophrenia is a public-private consortium with a budget exceeding 117 million dollars, coordinating multi-site networks to identify multimodal biomarkers, exosomal microRNA signatures, neuroimaging and electrophysiology, that predict individual conversion trajectories.

PROGRAMME	STARTED	AGE RANGE	DURATION	CASELOAD	THE DISCRIMINATOR
EPPIC, Australia	1992	12 to 25 years	2 to 5 years	15 to 20 patients per professional	**16 core components, including home evaluation, youth peer support, crisis response and functional recovery**
OPUS, Denmark	1996	18 to 45 years	2 years	10 patients per professional	**Modified assertive community treatment with family workshops and social skills groups of 6 to 8 participants**
RAISE and NAVIGATE, United States	2008	15 to 35 years	2 years	25 patients per team of 4 professionals	**Shared decision-making, personalised pharmacotherapy, family psychoeducation, resilience therapy, supported work**
Early intervention teams, United Kingdom	2011	14 to 35 years	3 years	Low caseload care coordination	**Bridges child, adolescent and adult services, with rapid community assessment that avoids compulsory pathways at first contact**

The four prototype services: the numbers to hold are the age band, the duration and the caseload.

Duration of untreated psychosis. Define it as the interval between the emergence of manifest positive psychotic symptoms and the start of adequate evidence-based antipsychotic pharmacotherapy or specialised psychosocial care.

- **Why it matters.** A longer interval correlates with poorer symptomatic response, greater brain structural change, more hospitalisation and compromised vocational and social recovery.
- **Where the delay sits.** Patient recognition latency, family help-seeking hesitation, referral bottlenecks at the general practitioner, and waiting time for secondary mental health assessment. Early detection

campaigns shorten it through professional education and rapid triage.

- **How long to keep specialised care.** Coordinated specialty care beats standard community care over 2 years, but gains attenuate when patients transition back to generic teams at the 2-year mark. Consensus now favours stepped or extended specialised care across the full 2 to 5 year critical period.

STATION: THE SEVENTEEN-YEAR-OLD BROUGHT BY A WORRIED PARENT

Synthetic case. Two months of suspiciousness, brief and self-limiting voices, and a fall in college attendance. The examiner's ladder:

1. Which of the three high-risk clusters does this presentation meet, and on what evidence?
2. Is the conviction held with insight, and what does that decide?
3. What is the duration of any fully psychotic symptoms, and how long per day?
4. What is the two-year non-transition figure, and how does it change what you offer?
5. What is first-line management here, and why is it not an antipsychotic?
6. What functional measures will you track, and over what period?
7. If conversion occurs, what shortens the duration of untreated psychosis in your setting?

RULE Antipsychotic medication is not first-line for the high-risk prodromal phase. First-line is cognitive behavioural therapy for psychosis, family support and monitoring, because the majority of those identified do not transition and antipsychotics carry metabolic and neurological risk.

EARLY PSYCHOSIS IN A DISTRICT SERVICE

Specialised early psychosis teams have spread more slowly in middle- and low-income settings. In India the delivery vehicle is the District Mental Health Programme team: psychiatrist, clinical psychologist, psychiatric social worker and community nurse.

Around that team sit medical officer triage and basic psychotropic supply at primary health centres, community outreach through accredited social health activists and volunteers, and telepsychiatry through the national tele mental health network and mobile telepsychiatry models.

The landmark Indian task-sharing evidence is the community care trial for schizophrenia, which showed that community interventions delivered by trained lay health workers under specialist supervision reduce disability and improve adherence in resource-constrained settings.

HOLD THIS

73% do not transition within 2 years, and nearly half of those remit. Cognitive behavioural therapy, family support and monitoring, not an antipsychotic.

WHAT EARNS THE MARKS

- **Naming which of the three clusters fits**: with the evidence for it, rather than calling the state a diagnosis
- **Quoting the two-year non-transition figure**: 73%, and using it to justify withholding an antipsychotic
- **Decomposing the untreated interval**: patient, family, general practitioner, service waiting time
- **Adapting the service model to a district team**: task sharing with supervision, and telepsychiatry

SEE ALSO 05 Schizophrenia beyond D2 11 The landmark trials 19 Prevention and positive psychiatry

7 · Rapid-acting antidepressants and the oral glutamatergics

RECENT ADVANCES · TOPIC 07

Why do we need rapid-acting antidepressants, and how does ketamine work?

VIVA TABLE: RECENT ADVANCES, AND THE TREATMENT-RESISTANT DEPRESSION LONG CASE

THE SHAPE OF THE ANSWER

1	**The two failures of monoamines**	Latency of four to six weeks, and incomplete remission
2	**The mechanism chain**	Disinhibition, AMPA, neurotrophin, target of rapamycin, new synapses
3	**The agents by route**	Intravenous, intranasal, oral, and the failed peptide
4	**The monitoring constraint**	Observation period, cardiovascular watch, certified setting
5	**The enantiomers**	S and R differ in affinity, dissociation and mechanism

Start with the two failures of monoamine pharmacology. Depression affected approximately 280 million people worldwide as of 2023, with some global estimates noting 332 million, and women experiencing it 50% more frequently than men.

- **Failure one, latency.** Conventional agents work through monoamine reuptake inhibition or enzymatic blockade, with a therapeutic latency of 4 to 6 weeks. That reflects the time needed for somatic 5-HT1A autoreceptor desensitisation, during which depression, emergent anxiety and elevated suicide risk persist.
- **Failure two, incomplete efficacy.** Only 2 out of 3 patients achieve full remission on first-line treatment, and approximately 30% fail to respond satisfactorily to at least 2 adequate trials, which is the operational definition of treatment resistance.

Ketamine, mechanism in one chain. Ketamine is a non-selective non-competitive antagonist at the ionotropic N-methyl-D-aspartate receptor. Sub-anaesthetic intravenous infusions of 0.5 mg/kg over 40 minutes were shown in 2000 to reduce depressive symptoms within 72 hours and often within hours.

The chain to recite, in order:

1. Blockade of receptors on GABAergic interneurons disinhibits presynaptic glutamate release.
2. Released glutamate activates postsynaptic AMPA receptors, driving sodium influx and brain-derived neurotrophic factor release.
3. Blockade of postsynaptic receptors halts eukaryotic elongation factor 2 kinase, preventing phosphorylation and derepressing translation of that factor.
4. The factor binds tropomyosin receptor kinase B, recruiting the extracellular signal-regulated kinase and protein kinase B pathways, which activate the mammalian target of rapamycin.
5. That drives synthesis of postsynaptic density protein 95 and the GluA1 subunit.

- **Three additional actions.** Ketamine inhibits glycogen synthase kinase 3, preventing density protein phosphorylation and suppressing GluA1 internalisation. It binds the receptor kinase transmembrane domain directly to enhance AMPA throughput and induce long-term potentiation. It also acts as a 5-HT1A agonist recruiting the same downstream cascade.

AGENT	ROUTE AND REGIMEN	EVIDENCE TO QUOTE	THE DISCRIMINATOR
Racemic ketamine	Intravenous 0.5 mg/kg over 40 minutes, under cardiovascular monitoring with a pre-infusion electrocardiogram	An optimised phase 3 trial of 179 patients with treatment resistance receiving twice-weekly injections showed over 50% achieving partial reduction or remission	**Monitor for transient rise in blood pressure and heart rate, sedation, and dissociative or psychomimetic emergence**
Esketamine, the S-enantiomer	Intranasal, single-use device of 28 mg, on-site under direct supervision, with observation for at least 2 hours	Approved in 2019 by the United States and European regulators for treatment resistance with active suicidal ideation, and in Europe as adjunctive treatment in psychiatric emergency	**Distributed in the United States under a risk evaluation and mitigation strategy requiring certified clinics, registered prescribers and enrolled patients**
Dextromethorphan with bupropion	One tablet of 45 mg dextromethorphan hydrobromide with 105 mg bupropion hydrochloride daily in the morning for 3 days, then one tablet twice daily	Approved in August 2022 as the first oral N-methyl-D-aspartate antagonist for major depressive disorder; phase 2 in 43 patients and phase 3 in 327 patients showed symptom reduction beginning at 1 to 2 weeks	**Bupropion inhibits CYP2D6 to prevent rapid dextromethorphan clearance; dextromethorphan supplies uncompetitive antagonism plus sigma-1 agonism. Maximum one tablet daily in moderate renal impairment or a CYP2D6 poor metaboliser**
Rapastinel	Intravenous tetrapeptide, functional partial agonist at the receptor glycine site	Reduced symptoms within 2 hours, sustained for 7 days, without psychomimetic effects in a phase 2 trial of 116 patients, then failed to separate from placebo in phase 3 relapse prevention	**A cautionary tale: rapid onset in phase 2 does not predict phase 3 relapse prevention**

The glutamatergic agents by route, with the regulatory or monitoring constraint that defines each.

TRAP Treating the two enantiomers as interchangeable. Esketamine has 3 to 4 fold higher receptor affinity than R-ketamine and carries higher dissociative, psychomimetic and sedative potential, which is why the observation period exists.

R-ketamine, for the follow-up. It is preclinically more potent, acts through microglial signalling rather than direct receptor blockade, and avoided dissociative effects in human testing. Its metabolite acts as a positive allosteric modulator of the receptor kinase and upregulates synaptic AMPA receptors without dissociation or anaesthesia.

HOLD THIS

0.5 mg/kg over 40 minutes, response within 72 hours, latency of 4 to 6 weeks abolished. Esketamine is 28 mg intranasal on site with observation for at least 2 hours.

WHAT EARNS THE MARKS

- **Naming both failures of monoamine pharmacology**: the 4 to 6 week latency and the remission shortfall
- **Reciting the chain in order**: disinhibition, AMPA, neurotrophin, target of rapamycin, synaptic proteins
- **Attaching the constraint to the route**: on-site observation for esketamine, CYP2D6 caution for the oral tablet
- **Separating the enantiomers**: higher affinity and dissociation for S, microglial signalling for R

SEE ALSO 08 Psychedelics and neuroactive steroids 09 Inflammation and the lithium revival 11 The landmark trials

8 · Psychedelics and the neuroactive steroids

RECENT ADVANCES · TOPIC 08

Where do psilocybin and the neuroactive steroids stand, and what are they approved for?

VIVA TABLE: RECENT ADVANCES, AND THE POSTPARTUM DEPRESSION SHORT CASE

THE SHAPE OF THE ANSWER

1 Psilocybin is a prodrug	Psilocin is the moiety that binds 5-HT2A
2 The circuit claim	Default mode network desynchronised, rigidity disrupted
3 Trials and regulation	The active control arm, and the one country prescribing
4 The steroid mechanism	Tonic inhibition at extrasynaptic delta receptors
5 Brexanolone against zuranolone	Route decides feasibility: infusion against oral course

Psilocybin is a prodrug, and that is the first thing to say. It is an indolealkylamine from mushrooms of the genus Psilocybe, first isolated by Albert Hofmann in 1958.

- **What happens after ingestion.** Dephosphorylation via alkaline phosphatases and non-specific esterases yields the active metabolite psilocin, which crosses the blood-brain barrier and acts as a non-selective 5-HT2A agonist on cortical pyramidal neurons.
- **The intracellular cascade.** Receptor binding activates phospholipase C, producing diacylglycerol and inositol triphosphate and mobilising intracellular calcium, which drives presynaptic glutamate release onto postsynaptic AMPA and N-methyl-D-aspartate receptors.
- **Then transcription.** Calcium influx activates calcium and calmodulin-dependent kinase IV, which phosphorylates the cyclic AMP response element-binding protein to initiate neurotrophic factor transcription. Psilocin also binds the tropomyosin receptor kinase B transmembrane domain directly, stabilising it as an active homodimer.

The circuit-level claim. Depression features hyperconnectivity and overactivity within the default mode network, a distributed network of medial prefrontal cortex, posterior cingulate cortex and angular gyrus that mediates self-referential thought, rumination and morbid introspection.

- **What psilocybin does to it.** It transiently downregulates and desynchronises that network. That is the mechanism offered for disruption of rigid stereotyped thinking, restored cross-network connectivity,

psychological flexibility and synaptogenesis.

AGENT	TRIAL DETAIL TO QUOTE	THE DISCRIMINATOR
Psilocybin in treatment resistance	A phase 2 trial in 233 patients compared single doses of 25 mg, 10 mg and 1 mg; the 25 mg dose achieved rapid sustained reduction against the 1 mg control, with transient headache, nausea, dizziness and fatigue	**The 1 mg arm is the active control, not a placebo, and saying so shows you have read the design**
Psilocybin in major depressive disorder	A phase 2 trial in 24 patients used two sessions at 20 mg per 70 kg and 30 mg per 70 kg with substantial symptom reduction	**Weight-based dosing, and two sessions rather than one**
Psilocybin-assisted psychotherapy	A trial at the Black Dog Institute, where 10 of 15 patients completed, used dose escalation without tapering existing antidepressants	**Background antidepressant treatment was maintained, which is the design question examiners probe**
Ayahuasca and dimethyltryptamine	Oral ayahuasca containing 0.36 mg/kg dimethyltryptamine improved symptoms by the first full day and held at 7 days; a phase 1 trial of intravenous dimethyltryptamine at 0.1 mg/kg then 0.3 mg/kg in 7 patients confirmed rapid action with transient rise in blood pressure and heart rate	**The brew works orally because harmine, harmaline and tetrahydroharmine are reversible monoamine oxidase A inhibitors that prevent gastrointestinal degradation**
5-methoxy-dimethyltryptamine	Inhaled formulations at 6 mg, 12 mg and 18 mg tested in 22 patients, with transient headache and flashbacks	**Acts predominantly through 5-HT1A agonism rather than 5-HT2A**
3,4-methylenedioxymethamphetamine-assisted psychotherapy	Breakthrough designation, with clinical remission in up to 67% of participants in phase 3 trauma trials	**The indication is post-traumatic stress disorder, not depression**

Serotonergic psychedelics by trial, with the design feature each is asked about.

Australia is the regulatory answer. Australia became the first country to permit prescribing of psilocybin for treatment-resistant depression under strict clinical conditions, and specialist psychiatrists are the only clinicians holding prescribing rights.

- **Verify before the exam.** The year of that authorisation is not carried in the library sources, so name the country and the restriction and not the year.

The neuroactive steroids, and why postpartum depression is their indication. These are endogenous steroidal positive allosteric modulators of GABA-A receptors, synthesised from cholesterol in glial and neuronal mitochondrial membranes. Allopregnanolone, androstanediol and pregnanolone sulfate are the endogenous forms.

- **Two receptor populations.** Synaptic receptors, built from 2 alpha, 2 beta and 1 gamma subunits, mediate rapid phasic inhibition. Extrasynaptic receptors, built from 2 alpha, 2 beta and 1 delta subunits, mediate persistent tonic inhibition.
- **How these agents bind.** At transmembrane interfaces between alpha and beta subunits and at the alpha subunit. In nanomolar concentrations they prolong channel opening in the presence of GABA; in micromolar concentrations they open the chloride channel without it.
- **The postpartum mechanism.** During pregnancy circulating allopregnanolone rises steeply, prompting compensatory downregulation of gamma and delta subunits. After delivery it falls precipitously, and in vulnerable individuals the homeostatic upregulation of receptors fails, leaving depleted tonic inhibition.

FEATURE	BREXANOLONE	ZURANOLONE
Molecule and formulation	Synthetic allopregnanolone, continuous intravenous	Synthetic oral neuroactive steroid
Approval	March 2019	August 2023
Regimen	Continuous infusion over 60 hours at 90 mcg/kg/hour, titrated from or to 60 mcg/kg/hour on tolerability	**50 mg orally once daily in the evening for 14 days, with a fat-containing meal**
Dose adjustment	Interrupt or slow the infusion for excessive sedation	Reduce to 40 mg daily for central nervous system depression; reduce to 30 mg daily in severe hepatic impairment, renal disease with an estimated glomerular filtration rate of 60 to 89 mL/min/1.73 m2, or strong CYP3A4 inhibition
Onset and durability	Improvement within 48 to 60 hours, maintained at 4 weeks	Significant improvement on the depression rating scale by the third day of treatment, over a short 2-week course
Key trials	A phase 2 in 21 women, and phase 3 trials in 138 and 108 women	A phase 3 in 150 women at 30 mg daily and a phase 3 in 196 women at 50 mg daily
Safety	**Risk of sudden loss of consciousness or excessive sedation, requiring a mitigation programme and inpatient monitoring; contraindicated at an estimated glomerular filtration rate below 15 mL/min/1.73 m2 and in pregnancy, acceptable in lactation**	**Sedation, somnolence, dizziness; fetal harm in animal models, so avoided in pregnancy and lactation**

The two agents are separated by route and therefore by feasibility: an inpatient infusion against an outpatient two-week oral course.

TRAP Attributing postpartum depression to classical synaptic receptors alone. The deficit is in extrasynaptic delta-subunit receptor upregulation after allopregnanolone withdrawal, that is in tonic rather than phasic inhibition, and that is what these agents restore.

- **Trap.** Calling psilocybin the ligand at 5-HT2A. Psilocybin is inactive and psilocin is the moiety that binds.

HOLD THIS

Brexanolone is 60 hours intravenous with monitoring; zuranolone is 50 mg oral in the evening for 14 days with a fatty meal. Tonic inhibition, not phasic, is the target.

WHAT EARNS THE MARKS

- **Naming psilocin as the active moiety**: psilocybin is the prodrug
- **Reading the trial design**: the 1 mg arm is an active control, and antidepressants were maintained in one protocol
- **Locating the postpartum deficit**: tonic inhibition at extrasynaptic delta-subunit receptors
- **Turning route into feasibility**: a 60-hour monitored infusion against a 14-day oral course

SEE ALSO 07 Rapid-acting antidepressants 09 Inflammation and the lithium revival 18 Neurobiology at the table

9 · Inflammation, precision psychiatry, and the lithium revival

RECENT ADVANCES · TOPIC 09

Is there a place for anti-inflammatory treatment in depression, and where does lithium stand now?

VIVA TABLE: RECENT ADVANCES, AND THE TREATMENT-RESISTANT MOOD DISORDER DISCUSSION

THE SHAPE OF THE ANSWER

1 Name the phenotype	Low-grade inflammation, blunted reward circuits, anhedonia
2 Quote the meta-analysis	Effect sizes on continuous scores, not on remission
3 State the dissociation	Symptom reduction without categorical episode resolution
4 Stratify practically	C-reactive protein, imaging, metaboliser status, low-risk measures
5 The lithium paradox	Most evidenced, least prescribed, and what is reachable here

The inflammatory story has a specific shape: it moves continuous scores, not categorical remission. A subset of depressed individuals carries a chronic low-grade inflammatory state that blunts frontostriatal reward circuits, precipitates anhedonia and impairs monoaminergic transmission.

The meta-analysis to cite. A comprehensive meta-analysis published in the American Journal of Psychiatry examined 19 randomised controlled trials. It focused on the 11 trials that enrolled depressed individuals with an inflammatory phenotype, operationalised by a high-sensitivity C-reactive protein threshold at or around 2 mg/L.

- **The two effect sizes.** Anti-inflammatory agents reduced anhedonia severity against placebo with a Hedges g of 0.40 and a 95% confidence interval of 0.08 to 0.71, and reduced overall depressive severity with a Hedges g of 0.35 and a 95% confidence interval of 0.05 to 0.64.
- **What did not move.** There was no statistically significant difference in categorical treatment response or remission, and serious adverse events did not differ.

Benefits were consistent across 14 trials with varying inflammatory cut-offs. They were not moderated by treatment-resistance status, by monotherapy against adjunctive use, or by

pharmacological class, which spanned non-steroidal anti-inflammatory drugs, minocycline, interleukin-6 inhibitors, tumour necrosis factor inhibitors and mitogen-activated protein kinase inhibitors.

RULE State the dissociation explicitly: anti-inflammatories produce meaningful *continuous* symptom reduction, most of all in the motivational and anhedonic domains, without reliably inducing categorical episode resolution. A candidate who claims full remission has overstated the evidence in a way the examiner is listening for.

What follows clinically. Elevated baseline C-reactive protein predicts poorer response to conventional monoaminergic antidepressants. Because potent biologic immunomodulators carry risks of serious infection and malignancy, current practice emphasises low-risk measures.

- **The low-risk measures.** Omega-3 polyunsaturated fatty acid supplementation, anti-inflammatory dietary patterns such as the Mediterranean diet, structured exercise, whole-body thermal modalities, and a diagnostic search for occult medical contributors such as adiposity, metabolic syndrome and autoimmune illness.
- **Stratification, four markers.** A high-sensitivity C-reactive protein cut-off of 2 mg/L identifies individuals unlikely to respond to a standard selective serotonin reuptake inhibitor. Resting-state functional magnetic resonance imaging of default mode network hyperconnectivity identifies candidates for psychedelic-assisted therapy or circuit-based neuromodulation.
- **And the pharmacogenetic pair.** CYP2D6 metaboliser status guides dextromethorphan and bupropion titration, while a check for CYP3A4 inhibitors avoids excess zuranolone exposure. A prescription digital therapeutic delivering 8 weeks of structured intervention has been approved for maternal depression.

The lithium revival, with its prescribing paradox. Lithium has been used for mental illness since the mid-nineteenth century and remains the gold-standard mood stabiliser, acting through inhibition of glycogen synthase kinase 3 and stimulation of neurotrophic signalling.

- **The registry evidence.** International registries record a substantial fall in prescribing. In Denmark it shifted from first-line to last-line, displaced by second-generation antipsychotics. In Scotland only 5.90% of patients with bipolar disorder received lithium monotherapy while 24.96% received antidepressant monotherapy.
- **The levels to quote.** British Association for Psychopharmacology guidance recommends target serum levels of 0.8 to 1.0 mmol/L for acute mania, and prophylactic mood stabilisation may require up to 12 months of continuous administration before full efficacy.

WHAT IS ACTUALLY REACHABLE IN AN INDIAN SERVICE

Lithium has held uninterrupted listing across the National List of Essential Medicines in the 2011, 2015 and 2022 editions. Standard monitoring of 12-hour trough serum levels, with a maintenance range of 0.6 to 1.2 mM given in the library's textbook source, remains an indispensable requirement.

High-cost therapies such as intranasal esketamine and oral zuranolone face accessibility and financial barriers. So off-label racemic intravenous ketamine at 0.5 mg/kg under structured anaesthetic and psychiatric hospital supervision, electroconvulsive therapy, and essential-list mood stabilisers remain the primary options for treatment-resistant mood disorder.

Repetitive transcranial magnetic stimulation to dorsolateral prefrontal cortex is an established non-pharmacological option. The library sources do not carry its session counts or pulse parameters, so give the target and not a protocol number.

HOLD THIS

Hedges g of 0.40 for anhedonia and 0.35 for severity, with no categorical advantage. Lithium is the most evidenced and the least prescribed agent in the field.

WHAT EARNS THE MARKS

- **Stating the dissociation**: continuous symptom reduction without categorical remission
- **Naming the stratifying threshold**: high-sensitivity C-reactive protein at or around 2 mg/L
- **Preferring low-risk measures**: biologic immunomodulators carry infection and malignancy risk
- **Quoting the prescribing paradox with figures**: the Scottish monotherapy rates make the point in one line

SEE ALSO 07 Rapid-acting antidepressants 10 Digital psychiatry and artificial intelligence 11 The landmark trials

10 · Digital psychiatry, artificial intelligence, translational advances

RECENT ADVANCES · TOPIC 10

Can artificial intelligence diagnose psychiatric illness, and what governs telepsychiatry in India?

VIVA TABLE: RECENT ADVANCES, AND THE ETHICS AND LAW FOLLOW-UP

THE SHAPE OF THE ANSWER

1 Give the honest ceiling	Above chance, below unsupervised clinical use, so decision support
2 Quote one success	Prediction of transition to psychosis performs best
3 Name the three failures	Data leakage, illusory generalisability, sample bias
4 The Indian rules	Consent, modality, drug lists, and the two hard stops
5 Pharmacogenomics and the rest	One mandated allele check, then the translational three

The honest answer about artificial intelligence is the one that scores. Anxiety and depression affect an estimated 29% of the global population across the lifespan, with global economic costs approximating one trillion United States dollars annually.

- **The ceiling on standalone accuracy.** Machine learning algorithms applied to multivariate neuroimaging and polygenic risk data achieve diagnostic accuracies between 48.1% and 62.0%: above chance, and inadequate for unsupervised clinical use.
- **What they are for.** These tools function as decision support over high-dimensional multimodal streams, not as autonomous diagnostic entities.
- **Large language models.** They have matched or exceeded clinicians on standardised question-answering benchmarks and clinical vignettes under test conditions, while hallucination risk, clinical context insensitivity and absent ecological validation constrain real-world integration.

PREDICTION TASK	REPORTED PERFORMANCE	THE DISCRIMINATOR
Transition to psychosis from clinical high risk and recent-onset depression	Sensitivity 76.0% to 88.0% with balanced accuracy 85.5%, using random forest, gradient boosting and deep neural networks on multimodal workflows	**The best-performing psychiatric prediction task in the literature, and the one to quote if asked for a success**
Functional and hospitalisation trajectory in first-episode psychosis	Area under the curve 0.68 to 0.88 for one-year educational or occupational status and hospitalisation	**Prognostic forecasting rather than cross-sectional classification, which is the clinically useful direction**
Onset of attention-deficit hyperactivity disorder	Area under the curve 0.75 with balanced accuracy 0.69 on deep neural networks	**Quote both metrics; accuracy alone overstates it**
Genomic prediction of schizophrenia	Polygenic risk score area under the curve 0.61; biologically guided auxiliary learning architectures reached 0.867 and 0.895; exome models span 0.54 to 0.95, with a gradient boosting model reaching 0.95 in a specific case-control cohort	**The wide span is the point: it reflects cohort selection, not model superiority**
Passive digital phenotyping in bipolar disorder	Adding smartphone accelerometer data improves symptom trajectory prediction by approximately 5% over typing dynamics and standard clinical data	**A modest increment honestly stated beats an inflated claim**
Smartphone applications in depression and anxiety	A meta-analysis of 176 randomised controlled trials found modest but statistically significant symptom reduction in depression and generalised anxiety disorder	**Real-world impact depends on sustained engagement and clinical oversight**

The performance figures examiners ask for, each paired with the caveat that makes the figure usable.

The three methodological failures, which are the real viva question. Name them in this order and give the mechanism of each.

- **Data leakage, also called double dipping.** Information from outside the training set enters feature selection, parameter tuning or model construction. When unsupervised clustering is immediately followed by differential feature selection without strict sample separation, performance is artificially

inflated. A meta-regression found that 46% of genomic machine learning studies used feature selection techniques susceptible to it.

- **Illusory generalisability.** Models with strong internal cross-validation collapse on external cohorts. Models predicting antipsychotic response failed to generalise across independent clinical trial datasets, and aggregating multiple trials did not improve out-of-sample accuracy.
- **Sample constraint and demographic bias.** Limited samples, high acquisition costs, missing variables and non-representative training cohorts create rigid algorithms. Continuous local revalidation and conformal prediction with explicit uncertainty estimates are preferred to static one-time deployment.

Explainable artificial intelligence, in four families. Each family answers a different clinical question about the same prediction.

- **Feature-based.** Quantifies each variable's mathematical contribution to an individual prediction.
- **Example-based.** Identifies training instances resembling the present patient.
- **Model-based meta-models.** Approximate a complex architecture with interpretable rules or decision trees.
- **Counterfactual.** Generates the hypothetical parameter change, in sleep duration or adherence, that would move an individual from high to low risk.
- **Why they matter.** The clinical argument is medicolegal liability, ethical responsibility and unseen demographic bias, not elegance.

Clustering and the dimensional neuroimaging endophenotype. Patients sharing a diagnostic label present with divergent symptom clusters and trajectories, so unsupervised learning groups them without preconceived labels.

- **The methods used.** Centre-based partitioning, model-based mixture and latent class approaches, hierarchical and density-based methods, and multi-view frameworks including nonparametric Bayesian mixtures for depression subtyping and deep autoencoders with clustering ensembles.
- **The imaging framework.** The dimensional neuroimaging endophenotype framework applies clustering to imaging data to capture continuous dimensions of progression alongside categorical neuroanatomical subtypes, and has produced replicable subtypes in Alzheimer disease, schizophrenia, major depressive disorder and autism spectrum disorder.
- **A pharmacological use.** Clustering of antipsychotics by receptor binding profile has produced taxonomies grouped by adverse-effect propensity.

TELEPSYCHIATRY OPERATIONAL GUIDELINES, AND WHAT THEY FORBID

Remote mental health delivery in India is governed by the Telepsychiatry Operational Guidelines released in May 2020 by the Board of Governors in supersession of the Medical Council of India, in collaboration with the national institute. They operationalise teleconsultation within the frame of the Mental Healthcare Act and the Information Technology Act 2000.

The practitioner verifies patient identity, displays their own registration credentials and obtains informed consent. Consent may be implied where the patient initiates the consultation, and must be documented explicitly where the clinician initiates it or where audio-video recordings are stored.

Video is the preferred modality for initial diagnostic evaluation, allowing appraisal of affect, psychomotor activity and nutritional status. Audio and text are reserved for follow-up review and urgent triage.

Medications are stratified into four lists: an over-the-counter list; a first-line refill list for maintenance of an established diagnosis previously assessed in person; an add-on list for optimisation at follow-up; and a prohibited list that includes Schedule X drugs of the Drugs and Cosmetics Rules and restricted psychotropic injectables or habit-forming narcotics.

In an acute emergency the practitioner gives immediate first-aid counselling, advises safe containment and arranges transfer to the nearest physical facility. The national tele mental health programme runs a state network anchored by the apex nodal technical institute, routing callers through a two-tiered triage structure to district teams.

TRAP Two hard stops. Teleconsultation cannot be used to initiate a supported or involuntary admission, which requires in-person examination and institutional procedure, and it cannot bypass patient autonomy or a valid advance directive. And prescribing a psychotropic injectable or a Schedule X stimulant over a follow-up video consultation is prohibited outright, not discretionary.

Pharmacogenomics, held to what it actually delivers. CYP2D6 and CYP2C19 are the major hepatic isoenzymes metabolising selective serotonin reuptake inhibitors, tricyclics and antipsychotics such as risperidone and aripiprazole, with genotypes classifying poor, intermediate, normal and ultrarapid metabolisers.

- **The prevalence figure to hold.** The library's textbook source puts poor metabolism of CYP2D6 at about 6 to 8% of Caucasians and a small proportion of Asians.
- **What the phenotype predicts.** Poor metabolisers show elevated plasma concentrations and greater adverse-effect vulnerability at standard doses, while ultrarapid metabolisers risk therapeutic failure through rapid clearance.
- **The one allele check to name.** HLA-B*1502 screening is the mandated check in individuals of Asian ancestry before carbamazepine, and is recommended for oxcarbazepine, because of the strong association with Stevens-Johnson syndrome and toxic epidermal necrolysis. HLA-A*3101 is associated with carbamazepine hypersensitivity across broader populations.
- **Where panels stand.** Randomised trials of commercial multigene combinatorial panels in major depressive disorder show modest improvement in response and tolerability among treatment-resistant populations, with limited evidence for unselected first-line testing.

The three translational advances. One block each, in the order an examiner tends to walk them.

- **The gut-brain-microbiome axis.** It communicates bidirectionally through the vagus nerve, neuroendocrine pathways, circulating microbial metabolites including the short-chain fatty acids butyrate, acetate and propionate, and the tryptophan-kynurenine shunt.
- **What dysbiosis is said to do.** It contributes to low-grade systemic inflammation, altered intestinal and blood-brain barrier permeability and microglial priming, implicated in treatment-resistant depression, autism spectrum disorder and neurodegeneration. Psychobiotic and dietary interventions show preliminary signals only.
- **Glucagon-like peptide-1 receptors.** Present in the hypothalamus, ventral tegmental area and nucleus accumbens, they modulate central dopaminergic transmission and neuroinflammation. The agonists are used clinically to reverse second-generation antipsychotic-induced weight gain and insulin resistance, with early work exploring craving reduction in substance use disorders and depressive neuroinflammation.
- **Long COVID neuropsychiatry, the presentation.** Cognitive slowing, executive dysfunction, new-onset depression, generalised anxiety, fragmented sleep architecture and dysautonomia.
- **Its putative mechanisms.** Persistent microglial activation, endothelial inflammation with microvascular thrombosis, autoantibody generation, reactivation of latent neurotropic pathogens and persistent viral antigen reservoirs. Management is individualised rehabilitation, pacing, targeted symptom pharmacotherapy and avoidance of premature overexertion.
- **Trap.** Dismissing those cognitive symptoms as purely functional.

HOLD THIS

Standalone machine diagnostic accuracy is 48.1% to 62.0%, and 46% of genomic studies leak data. Artificial intelligence is decision support; the prediction that works best is transition to psychosis.

WHAT EARNS THE MARKS

- **Giving the accuracy band before the enthusiasm**: 48.1% to 62.0% is above chance and below clinical autonomy
- **Naming data leakage by its mechanism**: feature selection that saw the test set
- **Knowing the two teleconsultation hard stops**: no admission initiation, no prohibited-list prescribing
- **Citing the one mandated allele check**: HLA-B*1502 before carbamazepine in Asian ancestry

SEE ALSO 09 Inflammation and precision psychiatry 14 Ethics and the codes 15 Research methodology

11 · The landmark trials examiners cite

RECENT ADVANCES · TOPIC 11

Tell me about the landmark antipsychotic trial, and what it changed.

VIVA TABLE: RECENT ADVANCES AND EVIDENCE, AND THE DRUG CHOICE DEFENCE IN ANY LONG CASE

THE SHAPE OF THE ANSWER

1 Name the endpoint first	Time to all-cause discontinuation, a pragmatic composite
2 Design and cohort	Sites, years, arms, dose ranges, who was excluded
3 The result in one line	Most discontinued, perphenazine matched, olanzapine retained best
4 The later phases	Clozapine enters only in the efficacy pathway
5 Criticisms and transfer	Four design limits, then cost, metabolic watch, clozapine timing

Name the endpoint before the result. The Clinical Antipsychotic Trials of Intervention Effectiveness was an independently funded pragmatic double-blind randomised trial sponsored by the United States National Institute of Mental Health, conducted between October 2001 and December 2004 across 57 clinical sites.

It compared second-generation antipsychotics against a representative intermediate-potency first-generation agent, perphenazine. Participants were adults aged 18 to 65 years with chronic schizophrenia able to take oral therapy.

- **The primary outcome.** Time to all-cause treatment discontinuation, a pragmatic metric that folds efficacy, tolerability, safety and the choices of patient and clinician into one measure.
- **The enrolment figure to prefer.** The primary report documents 1,460 enrolled patients, while secondary literature cites 1,432. Prefer the primary figure.
- **Two design details.** Ziprasidone was added in January 2002 after approximately 40% of the cohort had enrolled, and flexible dosing allowed between 1 and 4 identical capsules daily.

PHASE 1 ARM	DISCONTINUED	MEDIAN MONTHS TO DISCONTINUATION	WEIGHT GAIN OVER 7%	DISCONTINUATION FOR EXTRAPYRAMIDAL EFFECTS
Olanzapine, 7.5 to 30 mg daily	64%	9.2	30%	2%
Risperidone, 1.5 to 6 mg daily	74%	4.8	14%	3%
Perphenazine, 8 to 32 mg daily	75%	5.6	12%	8%
Ziprasidone, 40 to 120 mg daily	79%	3.5	7%	4%
Quetiapine, 200 to 800 mg daily	82%	4.6	16%	3%

Phase 1 in one grid: 74% of the whole cohort discontinued before 18 months, at a median of roughly 6 months overall.

The statistics, if pushed. Olanzapine achieved a significantly longer time to discontinuation than quetiapine, hazard ratio 0.63, and risperidone, hazard ratio 0.75.

The comparisons favouring olanzapine over perphenazine, hazard ratio 0.78 at p of 0.021, and over ziprasidone, hazard ratio 0.76 at p of 0.028, were not statistically significant after adjustment for multiple testing.

- **Hospitalisation for exacerbation.** 11% on olanzapine, 15% on risperidone, 16% on perphenazine, 18% on ziprasidone and 20% on quetiapine.
- **The movement disorder finding, stated precisely.** Extrapyramidal effects led to discontinuation in 8% of the perphenazine group against 2% to 4% among second-generation drugs, at p of 0.002, while incidence of new-onset extrapyramidal symptoms or persistent movement abnormality did not differ significantly across arms.

The metabolic and economic findings, which are the exportable part. Monthly weight change averaged a gain of 2.0 lb on olanzapine, 0.5 lb on quetiapine and 0.4 lb on risperidone, against a loss of 0.2 lb on perphenazine and 0.3 lb on ziprasidone.

Exposure-adjusted glycated haemoglobin change was 0.40% for olanzapine against 0.04% to 0.11% for the others. Triglyceride change rose by 40.5 mg/dl for olanzapine and 21.2 mg/dl for quetiapine, against falls of 2.4 mg/dl for risperidone and 16.5 mg/dl for ziprasidone, with a rise of 9.2 mg/dl for perphenazine. Ziprasidone had the most favourable metabolic profile.

- **The economic result.** Starting with perphenazine reduced average monthly healthcare expenditure by 300 to 500 dollars per patient, a reduction of 20% to 30%, without significant differences in quality-adjusted life years or symptom scores.
- **Cognition and quality of life.** Neurocognitive testing across 817 patients showed modest improvement at 2 months with effect sizes of 0.12 to 0.24 and no significant between-treatment difference. Quality of life scores at 12 months improved by a mean of 0.19 standard deviation units across all groups, with no perphenazine disadvantage.

LATER PHASE	WHO ENTERED	RESULT	THE DISCRIMINATOR
Phase 1B	114 participants who stopped perphenazine, re-randomised to olanzapine, quetiapine or risperidone	Median time to discontinuation 9.9 months for quetiapine and 7.1 for olanzapine against 3.6 for risperidone	**The one phase where quetiapine came out on top, which is why it is asked**
Phase 2E, the efficacy pathway	99 participants who discontinued a second-generation drug for inefficacy, 86% of that group, randomised to open-label clozapine or an alternative second-generation drug	Clozapine median 10.5 months against 3.3 for quetiapine, 2.8 for risperidone and 2.7 for olanzapine, with a 3-month syndrome scale reduction of 11.7 points against 2.5 and 4.1 on a scale running 30 to 210	**Clozapine entered the trial only here. Rotating non-clozapine agents after two failures buys a median of 2.7 to 3.3 months**
Phase 2T, the tolerability pathway	444 participants who stopped for intolerable side effects in 38% or inefficacy in 41%	Median 7.0 months for risperidone and 6.3 for olanzapine against 4.0 for quetiapine and 2.8 for ziprasidone	**The pathway a patient enters is decided by the reason they stopped, and the winner differs between pathways**

The four design criticisms. Give them as four, in this order.

- **Exclusion.** Participants with baseline tardive dyskinesia, 231 individuals or 15% of the cohort, were excluded from perphenazine randomisation, so the neurological safety comparison generalises only to the 85% without it.
- **Endpoint composition.** Time to all-cause discontinuation conflates therapeutic failure, adverse effects and clinician preference, although it correlates with subjective quality of life and reflects real outpatient practice.
- **Dose asymmetry.** Olanzapine was permitted up to 30 mg daily, above usual community practice, while risperidone was capped at 6 mg and ziprasidone at 120 mg, which may have inflated both its retention advantage and its metabolic signal.
- **Power.** The study was powered for superiority rather than non-inferiority, so failure to show second-generation superiority over perphenazine means any true advantage is small.

The other schizophrenia trials. Two, each with its own primary endpoint.

- **The British cost-utility study.** Run in the National Health Service. In its first arm, individuals requiring a medication switch were randomised to a first-generation antipsychotic or a non-clozapine second-generation antipsychotic, and the primary outcome, the quality of life scale, showed no significant difference between classes, with a non-significant trend favouring first-generation agents.
- **Its second arm.** Patients with treatment-resistant illness were randomised to clozapine against non-clozapine second-generation drugs, confirming superior symptom improvement on clozapine.
- **The European first-episode trial.** An open-label randomised trial in 498 participants with first-episode schizophrenia or schizophreniform disorder across European centres, assigning low-dose haloperidol or a second-generation agent: amisulpride, olanzapine, quetiapine or ziprasidone. Haloperidol produced a higher rate of discontinuation driven by extrapyramidal symptoms, while symptom reduction among those who stayed on treatment was comparable.

TRAP Two endpoint confusions that cost a mark each. Clozapine was not an arm in Phase 1; it entered only in the efficacy pathway of Phase 2, for patients who had failed a second-generation drug for inadequate response. And the primary endpoint of the American trial was time to all-cause discontinuation, whereas the British trial's primary endpoint was the Heinrichs and Carpenter quality of life scale. Swapping them tells the examiner you learned the conclusions without the designs.

The mood, depression and paediatric landmarks, stated as conclusions. One block per trial, design then conclusion.

- **The stepped-care depression trial.** The sequence ran citalopram monotherapy; then switch to sertraline, extended-release venlafaxine or sustained-release bupropion, or augmentation with bupropion, buspirone or cognitive therapy; then switch to mirtazapine or nortriptyline, or augmentation with lithium or triiodothyronine; then tranylcypromine, or venlafaxine with mirtazapine.
- **Its conclusion.** Remission rates fell across successive levels, and relapse during follow-up rose with the number of steps needed to reach remission.
- **The bipolar depression trial.** Acute bipolar depression was randomised to a mood stabiliser with an adjunctive antidepressant, paroxetine or bupropion, against a mood stabiliser with placebo. There was no significant advantage for the antidepressant in durable recovery.
- **The bipolar maintenance trial.** Lithium monotherapy, valproate monotherapy and the combination were compared in bipolar I maintenance. Both combination therapy and lithium monotherapy were superior to valproate monotherapy in preventing relapse, with the combination carrying the lowest hazard of recurrence.
- **The therapy augmentation trial.** In United Kingdom primary care, treatment-resistant depression on stable pharmacotherapy was randomised to usual care with or without added cognitive behavioural therapy. Augmentation significantly improved response.
- **The adolescent depression trial.** Adolescents were randomised to fluoxetine alone, cognitive behavioural therapy alone, the combination, or placebo. The combination produced superior response, and suicidal ideation fell across active groups with the greatest reduction under combination treatment.
- **The attention-deficit multimodal study.** Systematic medication management, intensive behavioural therapy, the combination and routine community care were compared. Medication management and the combination were superior for core symptoms, and the combination permitted lower stimulant dosing.

VERIFY BEFORE THE EXAM The library sources for this volume do not carry the participant numbers, follow-up durations or remission and response percentages for the stepped-care depression trial, the bipolar depression trial, the bipolar maintenance trial, the cognitive therapy augmentation trial, the adolescent depression trial, or the attention-deficit trial. The conclusions above are safe; the figures are a deliberate gap. Read the level-by-level remission percentages and the durable recovery rates off a primary report or a standard text before the viva, and at the table give the conclusion with the design rather than approximating a number.

The two network meta-analyses, and the one sentence each earns. The antidepressant network meta-analysis found all 21 antidepressants more efficacious than placebo with modest differences between individual agents: agomelatine, escitalopram, mirtazapine, paroxetine and vortioxetine showed higher relative efficacy, while escitalopram, vortioxetine and agomelatine showed favourable acceptability.

The antipsychotic network meta-analysis of 32 oral antipsychotics for acute schizophrenia found efficacy differences among non-clozapine agents to be small to moderate, with marked distinctions across adverse-effect domains: weight gain, sedation, extrapyramidal symptoms, hyperprolactinaemia and QTc prolongation.

RULE Do not read a rank order as absolute superiority. Efficacy differences among standard compounds are small, so drug choice is driven by medical comorbidity, tolerability profile and affordability, which is exactly the argument an Indian candidate is best placed to make.

THREE THINGS THESE TRIALS CHANGE AT AN INDIAN BEDSIDE

First, cost. Where expenditure is largely out of pocket, the finding that perphenazine matched second-generation agents on effectiveness while costing less supports affordable conventional agents and low-cost generics as viable first-line options under national and district programmes.

Second, cardiometabolic surveillance. Given South Asian vulnerability to metabolic syndrome, type 2 diabetes and coronary disease, the weight and lipid liabilities documented for olanzapine call for waist circumference, fasting glucose and lipid profile monitoring rather than unmonitored escalation.

Third, clozapine timing. Because rotating non-clozapine atypicals after two failures yields a median retention of a few months, two adequate trials should be followed by prompt clozapine with close haematological surveillance and a consent and admission route that complies with the statute.

HOLD THIS

74% discontinued before 18 months; perphenazine matched the second-generation agents and cost less; clozapine was the only agent that changed the curve, and only in the efficacy pathway.

WHAT EARNS THE MARKS

- **Leading with the endpoint**: time to all-cause discontinuation, and what that composite contains
- **Placing clozapine correctly**: Phase 2 efficacy pathway, not Phase 1
- **Naming the dose asymmetry**: olanzapine to 30 mg against risperidone capped at 6 mg
- **Converting the result into local practice**: cost, metabolic surveillance, and earlier clozapine

SEE ALSO 05 Schizophrenia beyond D2 15 Research methodology 20 Drug spotters

Epidemiology, history, ethics, research and pedagogy

This is the sub-area where a candidate is caught out by a single number. The defence is to state which figure you are quoting, from which survey, and whether it is current or lifetime, weighted or crude, before you say the number itself.

12 · The epidemiology numbers examiners ask

EPIDEMIOLOGY · TOPIC 12

What is the prevalence of mental illness in India, and what is the treatment gap?

VIVA TABLE: EPIDEMIOLOGY AND PUBLIC HEALTH, AND THE COMMUNITY PSYCHIATRY DISCUSSION

THE SHAPE OF THE ANSWER

1 Say current and lifetime together	Name the survey, the states, the sample, the instrument
2 Break it by category	Common, severe, substance, suicidal risk, with the gaps
3 Then the global youth data	What rose, what fell, over the thirty years
4 The gradient and the sex ratios	Development index reverses intuition, lethality reverses prevalence
5 Attach the population	A number without its population is not usable

Separate current from lifetime in the same breath. In the published report of the National Mental Health Survey of India, the weighted current prevalence of any mental morbidity was 10.56% with a 95% confidence interval of 10.51 to 10.61, and the weighted lifetime prevalence was 13.67% with a confidence interval of 13.61 to 13.73.

The survey covered 12 Indian states with 34,802 completed interviews in adults aged 18 years and above, assessing morbidity with the brief structured interview against ICD-10 diagnostic criteria for research. The pooled treatment gap for overall mental morbidity was 84.5%.

- **Which figure gets quoted.** The figure usually quoted first in Indian teaching is 10.6%, which is the rounded current prevalence and not the lifetime figure.
- **The four headline categories.** On that same set of candidate records, common mental disorders, comprising depressive, anxiety and neurotic or stress-related conditions, ran at a weighted 10.0%; severe mental disorders, comprising non-affective psychosis and severe bipolar illness, at 0.8%; and high suicidal risk at 0.9%.
- **The design, if asked.** Multi-stage stratified random cluster sampling, with clusters selected with probability proportionate to size.

CATEGORY	WEIGHTED PREVALENCE	TREATMENT GAP	THE DISCRIMINATOR
Any mental disorder	10.6%	84.5%	**The prevalence is a candidate-record figure and the gap is from the published report; say which is which if pushed**
Substance use disorders excluding tobacco	5.0%		**Overlaps with the other groups rather than adding to them**
Alcohol use disorder	4.6%	86.3%	**The largest treatment gap of any category**
Tobacco use disorder	13.1%		**Higher than any other single diagnostic category, and routinely forgotten**
Other substance use disorders	0.6%		**Small, which is the point against overestimating illicit use**
Schizophrenia and other psychotic disorders	0.4%	75.5%	**Not 1%: the clinical impression of 1% comes from lifetime risk estimates, not from this community survey**
Mood disorders	2.8%		**Of which depressive disorder is 2.7% and bipolar affective disorder 0.3%**
Depressive disorder	2.7%	85.2%	**The second largest gap, and the one that sits behind most primary-care screening arguments**
Bipolar affective disorder	0.3%	70.4%	**The lowest treatment gap of the five, which is counter-intuitive and therefore asked**
Neurotic, stress-related and somatoform disorders	3.5%	83.2%	**Phobic anxiety 1.9%, other anxiety 1.2%, obsessive-compulsive disorder 0.8%, post-traumatic stress disorder 0.2%**

The Indian grid. Substance use and depressive disorders carry the largest gaps; bipolar and psychotic disorders the smallest.

The global youth numbers, from the Global Burden of Disease analysis. Among youth aged 10 to 24 years across 204 countries, the age-standardised prevalence of 10 mental disorders reached 14,778.109 per 100,000 population in 2021.

- **The thirty-year direction of travel.** Between 1990 and 2021 the age-standardised prevalence of mental disorders rose by 6.8% with a 95% uncertainty interval of 4.7 to 9.0, and disability-adjusted life years attributable to them rose by 14%.
- **The highest and the lowest.** Anxiety disorders carried the highest prevalence at 4,976.610 per 100,000, and schizophrenia the lowest at 105.694 per 100,000.
- **What rose.** Depressive disorders showed the largest relative increase over the 30-year period, rising 22.5% with an interval of 18.3 to 26.5, with acute escalation between 2019 and 2021. Eating disorders rose 17.2%.
- **What fell.** Attention-deficit hyperactivity disorder fell 12.4% and idiopathic developmental intellectual disability fell 11.0%.
- **The two clear falls.** Substance use disorders stood at 1,573.968 per 100,000, a fall of 20.3%, with alcohol use disorders down 26.4% and drug use disorders down 15.5%. Self-harm stood at 45.136 per 100,000, a fall of 35.8% since 1990.

The socio-demographic gradient, which reverses the intuition. The socio-demographic index combines income per capita, educational attainment and total fertility rate under 25 years.

- **High-index regions.** High-income North America, Western Europe and Australasia carry the highest prevalence of anxiety disorders, autism spectrum disorders, depressive disorders, bipolar disorder, eating disorders, schizophrenia, alcohol use disorders and drug use disorders.
- **High and upper-middle index regions.** These carry the highest rates of attention-deficit hyperactivity disorder and of self-harm.
- **Low-index regions.** These carry the highest rates of conduct disorders, linked to poverty-associated adversity and family disruption.
- **Lower-middle index regions, prominently South Asia.** These carry the highest burden of idiopathic developmental intellectual disability in both prevalence and disability-adjusted life year rankings. In low- and middle-income settings approximately one-third of children under 5 years fail to reach expected cognitive milestones.
- **The extreme value.** The highest self-harm rates globally occur in the Inuit population of Greenland, concentrated in young males aged 20 to 24 years.

MEASURE	ANY MENTAL ILLNESS	SERIOUS MENTAL ILLNESS	THE DISCRIMINATOR
Definition	Any diagnosable mental, behavioural or emotional disorder in the past year, excluding developmental and substance use disorders, at any level of impairment	The same, but resulting in serious functional impairment that substantially interferes with or limits one or more major life activities	**Impairment, not diagnosis, is what separates the two**
Adult prevalence	23.1%	6.0%	**Roughly a quarter against roughly one in sixteen**
Female against male	26.4% against 19.7%	7.1% against 4.8%	**Female excess in both, wider in proportional terms for the serious category**
Age gradient	36.2% at 18 to 25 years, 29.4% at 26 to 49, 13.9% at 50 and above	11.6% at 18 to 25 years, 7.6% at 26 to 49, 3.0% at 50 and above	**Both fall steeply with age, which is the opposite of most chronic disease**
Past-year treatment	50.6%	66.7%	**Treatment contact is higher in the serious category, reflecting greater impairment**
Treatment by sex	56.9% of women against 41.6% of men	71.4% of women against 59.3% of men	**The sex gap in receiving care is wider than the sex gap in having illness**

Sex ratios and the lethality paradox. Take the direction of each comparison, not just the fact that one exists.

- **Female excess.** In the youth analysis, females outnumbered males in prevalence and disability-adjusted life years for anxiety disorders, depressive disorders and eating disorders.
- **Male excess.** Males exceeded females for conduct disorders, autism spectrum disorders, attention-deficit hyperactivity disorder, alcohol use disorders and drug use disorders. Differences were minimal for bipolar disorder, schizophrenia and developmental intellectual disability.
- **The paradox itself.** Self-harm prevalence was higher among females, while disability-adjusted life years and mortality from self-harm were substantially higher in males, attributed to methods of greater physical lethality.
- **The adolescent gradient.** Lifetime prevalence of any mental disorder rose from 45.3% at 13 to 14 years, to 49.3% at 15 to 16, to 56.7% at 17 to 18, with an overall figure of 49.5% and 22.2% showing severe impairment or distress.

VERIFY BEFORE THE EXAM Examiners ask for median age of onset by disorder and for cumulative lifetime risk by age 75, and the library's source for those figures was unusable, so no onset age or cumulative risk percentage is printed here. Specific female-to-male ratios such as two to one for depression are likewise unsourced in this set. Read them off a cross-national onset meta-analysis before the viva. At the table, examiners penalise an invented figure more heavily than an admitted gap.

RULE Say the population with the number. "Ten point six percent" alone is incomplete; "the weighted current prevalence of any mental morbidity in Indian adults aged 18 and above" is the claim. A number without its population is not a number an examiner can accept.

HOLD THIS

Current 10.56%, lifetime 13.67%, treatment gap 84.5%, psychosis 0.4% and severe mental disorders 0.8%. Tobacco at 13.1% is higher than any single diagnostic category.

WHAT EARNS THE MARKS

- **Giving current and lifetime as a pair**: 10.56% and 13.67%, each with its population
- **Naming the instrument and the sample**: a structured interview against ICD-10 research criteria, 12 states
- **Refusing the reflex 1% for psychosis**: the community survey figure is 0.4%
- **Stating the lethality paradox in the right direction**: female prevalence, male mortality

SEE ALSO 13 History and the Indian lineage 15 Research methodology 19 Prevention and positive psychiatry

13 · History, the Indian lineage, and the underused treatments

HISTORY · TOPIC 13

Who are the Indian figures in the history of psychiatry, and what did each contribute?

VIVA TABLE: HISTORY AND COMMUNITY PSYCHIATRY, AND THE CONSENT DISCUSSION IN AN ELECTROCONVULSIVE THERAPY CASE

THE SHAPE OF THE ANSWER

1	**Frame the colonial period**	Administration first, clinical care second
2	**The lineage**	Concept, place, date, for each named figure
3	**Committees to programme**	Bhore, Mudaliar, Srivastava, then the national programme
4	**The critique**	Madness as constituted object, and two colonial set pieces
5	**The underused three**	Electroconvulsive therapy, clozapine, lithium, with their numbers

Colonial psychiatry was an administrative instrument before it was a clinical one. Institutional psychiatry in India began under British rule to manage soldiers, civil servants and destitute Indian subjects, in segregated native lunatic asylums established under the East India Company. Confinement was presented in colonial medical discourse as benevolent rescue from ignorance and superstition.

The dates worth holding. Four, in order.

- **Lahore.** The Punjab Mental Hospital at Lahore, whose trajectory from 1840 to 1930 was documented by C.J. Lodge Patch in 1931.
- **The training turn.** An editorial in the Indian Medical Gazette in 1901 moved Indian facilities toward formal training for asylum attendants and modernised management.
- **Ranchi.** Owen Berkeley-Hill was Medical Superintendent of the Ranchi European Mental Hospital from October 1919 until he stepped down on 24 October 1934. Across that tenure of 15 years he claimed to have transformed a chaotic facility into what he called the finest mental hospital in Asia, and he applied psychoanalytic theory as an instrument of colonial administration.
- **Kanke.** L.P. Varma, first Assistant Superintendent of the separate Indian Mental Hospital at Kanke, published in 1953 the first systematic chronological history of psychiatric institutions across India and Pakistan.

FIGURE	DATES AND BASE	THE CONTRIBUTION THE EXAMINER WANTS
Girindrasekhar Bose	1886 to 1953, Calcutta	**The first psychoanalyst outside the Western world; founded the Indian psychoanalytical association; formulated the theory of opposite wishes in 1921; published on the psychological outlook of Hindu philosophy in 1930 and reviewed 25 years of Indian psychological research in 1938**
A. Venkoba Rao	Presidential address 1978	**Traced Indian psychiatric thought across pre-Vedic and post-Vedic Ayurvedic traditions, and in 1980 published an analysis of the Bhagavad Gita and the mental sciences, mapping ancient concepts of mind to contemporary constructs**
Vidya Sagar	Amritsar Mental Hospital, from the 1950s	**The camp approach: tents on hospital grounds with families living alongside the ill relative and participating in daily therapeutic routine, which broke asylum seclusion and showed family participation improves outcome in a resource-constrained setting**
N.N. Wig	PGIMER Chandigarh, then AIIMS New Delhi	**Rural community psychiatry in the Raipur Rani block of Haryana within the World Health Organization multi-country collaborative study from 1975 to 1981; delineated Dhat syndrome, the Hanuman complex, and acute and transient psychotic disorders**
J.S. Neki	AIIMS New Delhi and PGIMER Chandigarh	**The Guru-Chela relationship as an indigenous psychotherapeutic paradigm, and Sahaja as an Indian cultural ideal of mental equilibrium**

FIGURE	DATES AND BASE	THE CONTRIBUTION THE EXAMINER WANTS
Erna Martha Hoch	Swiss-born, worked in India from 1956 to 1988	**Early child psychiatric services, including the child guidance clinic at the Ranchi institute in 1950, teaching at Lady Hardinge Medical College in 1966 and AIIMS from 1967 to 1968, and psychiatric facilities in Srinagar from 1969 to 1980**
M. Sarada Menon and S. Rajkumar	Chennai	**Founded the schizophrenia research foundation, working on community intervention, mobile telepsychiatry and longitudinal outcome studies**

The lineage as an examiner asks it: the concept, then the place, then the date.

Committees, programmes and the community turn. Three committees, then the programme.

- **Bhore, 1946.** Recommended upgrading mental hospitals, integrating mental health into general healthcare and establishing a premier training institute.
- **Mudaliar, 1962.** Recommended small psychiatric clinics of 5 to 6 beds in each district hospital.
- **Srivastava, 1974.** Introduced the community health volunteer scheme, training village-level workers to identify and help manage mental disorders.
- **The National Mental Health Programme.** First draft in February 1981, first national workshop in July 1981, second workshop and approval by the Central Council of Health in August 1982.
- **Why the Indian path differed.** Western deinstitutionalisation closed large state asylums and frequently produced transinstitutionalisation into prisons or homelessness when community funding lagged. The Indian path diversified services into general hospital psychiatric units, primary health centre integration and family-supported outpatient care, leaving mental hospitals as tertiary regional centres.

The critique, and two colonial set pieces. Michel Foucault's Madness and Civilization, published in 1965, refused the traditional evolutionary history of psychiatry as unbroken humanitarian progress.

- **His argument.** Madness was constituted as an object of knowledge through discursive practice and social exclusion, beginning with the seventeenth-century great confinement. In colonial territories Western psychiatry translated universal scientific claims into tools for managing subject populations.
- **Two who extended it.** Ashis Nandy analysed the same dynamic in India, showing that modern institutions colonised minds as well as bodies. Frantz Fanon, working as a psychiatrist in colonial Algeria, showed colonial oppression producing psychological pathology directly.
- **Set piece one, the Indian Lunacy Act of 1912.** It codified colonial asylum practice around magistrate reception orders, police detention and protection of property rather than therapeutic rehabilitation.
- **Set piece two, the Indian Hemp Drugs Commission of 1894.** It surveyed indigenous practitioners and asylum superintendents on cannabis. Historical analysis shows superintendents routinely applying biased criteria to classify ordinary consumption as toxic insanity, inflating the diagnosis of hemp insanity and reinforcing administrative control.

TREATMENT	ORIGIN	THE NUMBERS THAT SHOW THE UNDERUSE	THE DISCRIMINATOR
Electroconvulsive therapy	Described by Ugo Cerletti and Lucio Bini in 1938	Inpatient administration in South London teaching hospitals fell from 35% in 1956 to 2.2% in 1991; three-month session tallies in England fell from 34,485 in 1985 to 6,782 in 2006; in 2021 the accreditation service documented 2,158 courses across 85 of 93 registered clinics	**After successful acute treatment for depression, stopping without maintenance pharmacotherapy leads to relapse in up to 85%. Modern practice means anaesthesia, muscle relaxation, electroencephalographic monitoring, seizure threshold titration, ultrabrief pulse width and unilateral placement**
Clozapine	Synthesised in the 1950s during antidepressant development; Kane and colleagues confirmed superiority in patients refractory to chlorpromazine in 1988	Response rate in treatment-resistant schizophrenia is 41% against 5% for olanzapine titrated up to 25 mg daily. Starting within a critical window of 2.8 years from psychosis onset yields an 82% response, against 31% when delayed beyond it	**Neutropenia occurs in approximately 3% and agranulocytosis in 0.4%. An interruption exceeding 48 hours requires complete retitration, and smoking cessation reduces clearance enough to require dose reductions of up to 70%**
Lithium	Mid-nineteenth century use documented, and established as the gold-standard mood stabiliser	In Scotland only 5.90% of patients with bipolar disorder received lithium monotherapy while 24.96% received antidepressant monotherapy; in Denmark it moved from first-line to last-line, displaced by second-generation antipsychotics	**Target serum level of 0.8 to 1.0 mmol/L for acute mania under the British Association for Psychopharmacology, and up to 12 months of continuous administration before full prophylactic efficacy. Once-daily dosing reduces the risk of decline in glomerular filtration**

Three treatments with strong evidence and falling use, which is the framing the question is set in.

The treatment-resistance definition, given precisely. Treatment-resistant schizophrenia is symptoms of at least moderate severity failing to respond to 2 trials of antipsychotics at a therapeutic dose, a chlorpromazine equivalent of at least 600 mg daily, for at least 6 weeks each, with adherence confirmed at at least 80%.

- **Give all four elements.** The number of trials, the dose equivalence, the duration, and the adherence threshold.

A FINDING WORTH QUOTING ON ACCEPTABILITY

A public survey in Bangalore by Andrade and Thyagarajan in 2007 showed that the term electrostimulatory therapy achieved significantly higher public acceptability than electroconvulsive therapy. It is a useful answer to a question about consent and stigma in a setting where the family decides whether treatment proceeds.

- **TRAP** Crediting Cerletti and Bini with convulsive therapy in general. They introduced electrical induction in 1938; chemical convulsive therapy with camphor and pentylenetetrazol came earlier, from Ladislas Meduna, and the year of that introduction is not carried in the library sources.

- **Trap.** Confusing the acute antimanic plasma level with the time to full prophylaxis. They are different quantities, and the second is measured in months.

HOLD THIS

Two trials, 600 mg chlorpromazine equivalent, 6 weeks, 80% adherence, then clozapine. Started within 2.8 years the response is 82%; delayed, 31%.

WHAT EARNS THE MARKS

- **Attaching a concept to each name**: opposite wishes, the camp approach, Guru-Chela, Dhat syndrome
- **Dating the committees and the programme**: 1946, 1962, 1974, then 1981 to 1982
- **Giving all four resistance elements**: trials, dose equivalent, duration, adherence
- **Quoting the clozapine window**: 82% within 2.8 years against 31% after it

SEE ALSO 14 Ethics and the codes 21 The electroconvulsive therapy machine 25 Historical figure spotters

14 · Ethics: the principles, the boundaries, and the codes

ETHICS · TOPIC 14

Your patient refuses admission and you believe he will die. Which principles are in conflict, and how do you resolve them?

VIVA TABLE: ETHICS AND LAW, AND THE CONSENT DEFENCE IN ANY INVOLUNTARY CASE

THE SHAPE OF THE ANSWER

1	**Name the four principles**	Autonomy, beneficence, nonmaleficence, justice
2	**Name the conflict**	Illness distorts the authentic and sustained preference
3	**Apply a structure**	Four boxes in order, capacity on a sliding scale
4	**Justify coercion narrowly**	Parens patriae, police powers, least restrictive alternative
5	**Cite a code**	Madrid, the 2020 Code, and the Indian statutory safeguards

Name the four principles, then name the conflict. Modern psychiatric ethics rests on the four prima facie duties of Beauchamp and Childress.

- **Autonomy.** The obligation to honour personal self-governance over body, mind and treatment.
- **Beneficence.** The positive duty to relieve distress, prevent harm and enhance quality of life.
- **Nonmaleficence.** The duty to avoid inflicting unnecessary harm.

- **Justice.** Fair distribution of healthcare resources, parity in mental health coverage and non-discrimination.
- **What Roberts adds.** Fidelity, placing the patient's wellbeing above personal gain, together with veracity, confidentiality, respect for persons, clinical competence, and respect for the law.

MODEL	CORE CONCEPT	WHERE IT EARNS ITS KEEP
Beauchamp and Childress	Four prima facie principles	**Identifies the duties and the structural conflict, but does not resolve it**
Hundert	Resolves latent conflicting values through explicit prioritisation and clarification	**Use it when the conflict is between a societal and a personal duty**
Jonsen, Siegler and Winslade four-box model	Analyses a case in descending order: clinical indications, patient preferences, quality of life, contextual and socioeconomic features	**The bedside structure, and the order is the answer: clinical indications come first in an emergency**
Drane sliding scale	The stringency of the capacity standard varies directly with the gravity of the decision	**A routine examination needs low-level capacity, choosing psychotherapy over medication needs intermediate capacity, refusing life-saving treatment or demanding discharge in delirium needs the most stringent standard**

How the conflict is resolved in an emergency. In severe depression with active suicidality, or in acute psychosis, beneficence and autonomy collide.

- **The reasoning to say out loud.** The clinician intervenes to preserve life because active illness temporarily distorts the person's authentic and sustained preferences. Beneficence and nonmaleficence override the immediately stated wish, with the explicit purpose of restoring long-term autonomy. That last clause is what separates an ethical answer from a paternalistic one.

Boundaries, and why consent cannot cure a sexual relationship. Define the two terms before you judge the case.

- **A boundary crossing.** A benign, non-exploitative departure from standard practice fitted to a clinical context, such as accepting an inexpensive festive card or stepping out of the office to help a fainting relative. It does not harm the patient and is examined by clinical self-reflection.
- **A boundary violation.** A harmful, exploitative transgression in which the clinician uses the therapeutic relationship to meet personal, emotional, financial or sexual needs.
- **Why the asymmetry is decisive.** In psychotherapy the patient discloses deep vulnerabilities, fears and sexual conflicts, and that knowledge gives the clinician exceptional authority. Consent obtained inside a fiduciary power differential is exploitation, so patient consent to a sexual relationship is legally and ethically vitiated, and sexual behaviour with a current patient is categorically prohibited whoever initiates it.
- **The clinical skill.** Recognising internal discomfort and countertransference signals early, before erosion becomes violation.

The ethical justification for coercion. Coercive intervention rests on parens patriae, the state's protection of vulnerable individuals, and on police powers, the prevention of harm to others.

- **The international limit.** It is restricted to exceptional circumstances where withholding treatment would endanger the life or physical integrity of the patient or those around them, and it must apply the least restrictive alternative and serve the patient's best interests.
- **The Indian requirements.** Supported admission requires severe illness, significant risk of harm to self or others, and inability to decide independently, with statutory time limits and independent scrutiny by a review board. Nominated representatives and valid advance directives are the instruments that carry prior autonomous preference into a later crisis.

VERIFY BEFORE THE EXAM Examiners ask for the exact statutory limits in days: the ceiling on an initial supported admission, the permitted extension, the length of each renewal, and the cadence of review board scrutiny. None of the ethics sources in the library carries the Act's text, so no such figure is printed in this topic. Read the numbers off the Act itself, and at the table give the criteria and the safeguards in words rather than approximating a figure.

The history that produced the codes. The Sterilization Act of 1933 in Germany mandated forced sterilisation, and the Action T4 programme from 1939 caused the death of approximately 300,000 mentally ill citizens with the direct participation of medical personnel. Compulsory eugenic sterilisation statutes operated in the United States from 1907 onward.

- **What the Soviet abuses produced.** Political abuse of psychiatry in the Soviet Union in the 1970s led to the Declaration of Hawaii in 1977, comprising 10 points, updated in Vienna in 1983.
- **The Madrid sequence.** After the Athens Declaration of 1989 and United Nations Resolution 46/119 of 1991, the World Psychiatric Association General Assembly endorsed the Declaration of Madrid on 25 August 1996, with additional guidelines added in Hamburg on 8 August 1999 and amendment in Yokohama in August 2002.

The seven general guidelines of Madrid, in order. Learn them by number, because the examiner may ask for the fourth.

1. **The aims of psychiatry:** optimal evidence-based treatment, promotion of mental health, rehabilitation, the least restrictive intervention, and advocacy for equitable resource allocation.
2. **Continuing professional development.**
3. **The patient as a partner by right:** mutual trust, adequate clinical information and rational decision-making in accordance with personal values.
4. **Care of incapacitated patients:** consult families and legal counsel, withhold treatment against their will unless withholding it endangers life, and act in the patient's best interest.
5. **Third-party assessments:** inform the individual beforehand of the purpose, the intended use of findings and the potential repercussions.
6. **Confidentiality:** restrict clinical information to improving the patient's health, do not exploit data for personal, financial or academic gain, and breach it only where serious harm to the patient or a third party would otherwise ensue, with advance notice where possible.
7. **Research ethics:** conform to the canons of science, secure prior approval from an independent ethics committee, and take extra care with vulnerable psychiatric subjects.

SPECIFIC SITUATION	THE REQUIREMENT	WHAT IS PROHIBITED
Euthanasia	Protect life, alleviate distress, and treat the underlying depression that distorts perception	**Any action intended to terminate the life of an incapacitated person**
Torture	Refuse involvement in physical or mental mistreatment	**Participation in any process of torture, even under state coercion**
Death penalty	Refuse involvement in execution protocols	**Participating in an execution, and assessing competency to be executed**
Sex selection	Support non-biased reproductive health	**Participating in termination of pregnancy for sex selection**
Organ transplantation	Advise on psychosocial factors and support autonomous decision-making	**Acting as a proxy decision maker, or using psychotherapy to persuade donation**
Media contact	Preserve dignity, reduce stigma, convey validated scientific data	**Diagnosing or publicising the presumed psychopathology of an individual in the media**
Ethnic discrimination	Ensure equitable respectful access regardless of cultural background	**Supporting or endorsing ethnic cleansing**
Genetic research	Obtain rigorous informed consent and explain the limits of current genetic science	**Testing without adequate counselling resources and privacy protection**
Psychotherapy practice	Maintain explicit consent, professional boundaries, and pharmacotherapy where indicated	**Exploiting countertransference, and breaching confidentiality without legal mandate**
Industry relations	Disclose trial funding to review boards and to research participants	**Accepting gifts that introduce undue prescribing influence**
Third-party payers	Advocate for insurance parity and access to evidence-based care	**Compromising the standard of care to accommodate profit-driven cost containment**
Sexual boundaries	Uphold therapeutic neutrality and emotional safety	**Sexual behaviour with a patient, categorically**

The twelve specific directives of the Madrid Declaration with its Hamburg and Yokohama revisions.

The 2020 Code, and how it differs. The World Psychiatric Association Code of Ethics was approved by the virtual General Assembly in 2020, succeeding the Hawaii and Madrid declarations. It was developed over nine years following presentation at the Berlin General Assembly in 2017 and endorsement by the executive committee in September 2019.

The Association unites 140 member societies across more than 120 countries, representing over 250,000 psychiatrists.

- **Its four sections.** Clinical practice, emphasising supported decision-making, autonomy, confidentiality and minimisation of restraint. Education, covering standards for training residents, ethical dissemination of knowledge and teaching boundaries.
- **And the other two.** Research and publication, requiring scientific rigour, protection of vulnerable subjects, transparency of trial data and prevention of ghostwriting or commercial bias. Public mental health, directing psychiatrists to combat stigma, advocate parity, address systemic inequity and avoid public speculation about individuals.
- **Its major addition.** The explicit inclusion of families and informal caregivers as essential collaborators in the therapeutic plan.

Research ethics in a vulnerable population, in six lines. Take them as six separate duties.

- **Method is an ethical matter.** Research that does not conform to rigorous scientific methodology is inherently unethical, because it exposes participants to risk without prospective societal benefit.
- **Review.** Every protocol requires review and approval by an independent institutional ethics committee.
- **Capacity for research consent.** It must be formally examined, with validated instruments where appropriate. Psychosis or severe depression does not by itself imply research incompetence, but it does require calibrated scrutiny.
- **Therapeutic misconception.** Participants must understand that study procedures are designed for scientific enquiry rather than individualised treatment.
- **Disclosure.** Investigators disclose financial affiliations, sponsorships and contractual obligations to the committee and to participants. Contractual restrictions preventing publication of negative findings are unethical.
- **Literacy is not an exemption.** Informed consent cannot be bypassed because of low literacy, and proxy consent is required where the person is incapacitated.

VERIFY BEFORE THE EXAM A complete research-ethics answer opens with the general codes beneath ethics-committee review and informed consent, the Nuremberg Code and the World Medical Association Declaration of Helsinki, with the Indian Council of Medical Research national ethical guidelines as the Indian layer, before moving to the psychiatry-specific Hawaii, Madrid and 2020 instruments. Their dates, clause content and amendment history are not carried in the library sources for this volume, so name the instruments and take the dates from a primary code text before the viva.

INDIAN REGULATORY CONDUCT

Under National Medical Commission regulations, which replaced the earlier council guidelines, a registered medical practitioner is prohibited from receiving gifts, travel facilities, hospitality or monetary grants from pharmaceutical companies, device firms or diagnostic entities, and is barred from endorsing commercial products, formulations or healthcare software publicly or on social media.

Romantic or sexual interaction with a current patient or an immediate family member is gross professional misconduct, attracting suspension or erasure. Prescriptions must use legible generic names in capital letters.

Teleconsultation prescribing is limited to the permitted drug categories, and Schedule X substances and medications with high dependence liability cannot be prescribed remotely.

Statutory patient rights include confidentiality, emergency care, non-discrimination, protection from cruel or degrading treatment, and access to records. Unmodified electroconvulsive therapy and sterilisation as a treatment for mental illness are barred.

CONCEPT	ITS NEAR NEIGHBOUR	THE DISTINCTION THAT SCORES
Boundary crossing	Boundary violation	**A crossing is benign, isolated and serves the patient; a violation is exploitative, serves the clinician, and compromises care**
Beneficence	Paternalism	**Beneficence secures wellbeing with the patient's collaboration; paternalism overrides a valid autonomous decision on the presumption that the clinician knows best**
Third-party assessment	Therapeutic alliance	**An evaluation for a court or employer lacks the usual confidentiality, and the evaluee must be warned beforehand of the limits to privacy**
Psychiatric diagnosis	Decisional incapacity	**A diagnosis does not equate to incapacity: capacity is decision-specific, time-specific and task-specific**
Madrid Declaration	The 2020 Code of Ethics	**Madrid gives seven general and twelve specific guidelines focused on avoiding abuses; the 2020 Code organises duties across four institutional pillars and formally integrates family caregivers**

HOLD THIS

Autonomy, beneficence, nonmaleficence, justice; four boxes in order; capacity stringency rises with the gravity of the decision. Consent inside a power differential is not consent.

WHAT EARNS THE MARKS

- **Adding the purpose clause to any override**: intervening in order to restore long-term autonomy
- **Separating a crossing from a violation**: who the act serves, and whether care is compromised
- **Naming the coercion basis**: parens patriae and police powers, with the least restrictive alternative
- **Keeping diagnosis and capacity apart**: capacity is decision, time and task specific

SEE ALSO 04 Indian Psychiatric Society guidelines 15 Research methodology 17 Psychiatric emergencies

15 · Research methodology and biostatistics

RESEARCH · TOPIC 15

Which study design would you use for this question, and which test for this data?

VIVA TABLE: RESEARCH METHODOLOGY AND BIOSTATISTICS, AND THE THESIS DEFENCE

THE SHAPE OF THE ANSWER

1 Start from the question domain	Therapy, prognosis, aetiology, meaning, each has its design
2 Separate bias from confounding	Name the control that answers each one
3 Handle the pyramid honestly	Design label did not predict accuracy on test
4 Alpha, beta, power, sample size	Give the conventions and what moves the sample
5 Then pick the test	Question, measurement scale, then design structure

Answer a design question with the question domain, not with a rank. Studies divide into observational and experimental.

- **Observational.** Descriptive case reports and case series, alongside analytic cross-sectional, case-control and cohort studies.
- **Experimental.** The investigator allocates an intervention, principally in the randomised controlled trial.
- **Why the analytic ones count.** Analytic observational studies have a defined comparison group and therefore internal control, which separates them methodologically from uncontrolled case reports and clinical anecdote.

QUESTION DOMAIN	OPTIMAL DESIGN	WHY
Therapy	Randomised controlled trial	**Randomisation breaks the prognostic link between allocation and baseline confounders**
Prevention	Randomised controlled trial or prospective study	**Evaluates risk reduction over time against a comparison group**
Diagnosis	Randomised controlled trial or cohort study	**Compares an index test against an established reference standard in a defined population**
Prognosis	Cohort study, with a case-control series where needed	**Evaluates longitudinal course, relapse rates and functional outcome**
Aetiology	Cohort study	**Indispensable where the exposure, substance use or trauma, cannot ethically be randomised**
Meaning and lived experience	Qualitative study	**Evaluates perspective and subjective distress through structured qualitative enquiry**

- **RULE** Statistical tests evaluate associations; the study design alone determines whether a causal inference is justified. Say that sentence before you name any test, because it pre-empts the follow-up question.

Bias against confounding, with the controls that answer each. Define both, then walk the controls in the order a trial applies them.

- **Bias.** Systematic error in design, enrolment, conduct or analysis that shifts the effect estimate away from the true value.
- **Confounding.** An extraneous variable independently associated with both exposure and outcome, creating a spurious association or masking a real one.
- **The comparison group.** It separates intervention effect from natural history, spontaneous remission, regression to the mean and secular trend.
- **Randomisation and concealment.** Randomisation distributes measured and unmeasured baseline confounders across arms. Allocation concealment prevents knowledge of the upcoming assignment and so reduces selection bias at enrolment.
- **Blinding, in two places.** Blinding of participants and clinical personnel reduces performance bias and expectation effects. Blinding of outcome assessors reduces detection bias, which matters most for subjective psychiatric rating scales.
- **Analysis-side controls.** Intention-to-treat analysis with standardised handling of missing data limits attrition bias. In observational work, confounding is controlled at design by restriction and matching, and at analysis by stratification, multivariable regression or analysis of covariance.

The meta-epidemiology of concealment, quoted study by study. Inadequate or unclear allocation concealment is associated with larger estimated treatment effects, and the counts belong to each study rather than to a pooled range.

- **Schulz and colleagues.** 250 controlled trials across 33 meta-analyses.

- **Wood and colleagues.** 1,346 trials across 146 meta-analyses, with exaggeration most marked for subjectively assessed outcomes.
- **Savovic and colleagues.** Seven meta-epidemiological datasets totalling 1,973 trials.
- **Page and colleagues.** 24 meta-epidemiological studies, with a ratio of odds ratios of around 0.90 to 0.93, indicating larger apparent benefits in trials at high risk of bias. They gave no trial count for those 24 studies.

Reviews, and why the top of the pyramid is not safe ground. Separate the three kinds before you judge one.

- **Narrative review.** An author-selected summary prone to subjective sampling bias, with no explicit search protocol or structured appraisal.
- **Systematic review.** A focused question answered with explicit reproducible methodology to identify, appraise and synthesise all eligible studies. Where outcome metrics match, a meta-analysis aggregates effect sizes into a pooled estimate.
- **What both remain.** Secondary syntheses. They generate no primary data and can amplify the biases of the studies they contain.
- **The growth figures.** Annual publications rose by 2,728% for systematic reviews and 2,635% for meta-analyses between 1991 and 2014, against a 153% increase for all indexed items.
- **The discordance in depression research.** 185 meta-analyses on antidepressants appeared between 2007 and 2014, with paroxetine ranking anywhere from first to tenth best and sertraline from second to tenth.
- **The redundancy figures.** Among systematic reviews on overlapping topics, 68% overlapped, with up to 76 overlapping reviews on a single topic, while only 36% cited previous reviews and only 9% reported protocol registration.
- **And the bias-handling figure.** Of systematic reviews, 70% assessed risk of bias and only 15% incorporated that risk into the primary synthesis.

The reporting standards and the pyramid critique. Name the instrument and what it standardises.

- **The trial standard.** The consolidated standards of reporting trials give standardised items for randomised trials, covering sequence generation, allocation concealment, blinding and participant flow.
- **The review standard.** The preferred reporting items for systematic reviews and meta-analyses standardise search strings, screening criteria and risk of bias evaluation.
- **Verify before the exam.** Their checklist item counts are not carried in the library sources, so name the instrument and its purpose rather than a count.
- **Where hierarchies came from.** Evidence hierarchies began with the Canadian Task Force on the Periodic Health Examination in 1979, which introduced 3 levels of evidence and 5 recommendation grades as pragmatic tools for preventive guidelines.
- **And where they went.** The Oxford Centre for Evidence-Based Medicine model of 2011 ranks from level 1 systematic reviews down to level 5 expert opinion. More than 80 variant hierarchies have been catalogued, and often misapplied as rigid surrogates for truth.

EMPIRICAL COMPARISON	WHAT IT FOUND	THE DISCRIMINATOR
Benson and Hartz, 2000	136 reports across 19 clinical topics; only 2 of 19 observational estimates fell outside the 95% confidence interval of the corresponding trial estimate	**Design label did not predict accuracy**
Concato and colleagues, 2000	5 clinical topics; well-designed cohort and case-control studies did not systematically overestimate effect sizes against trials	**The claim of systematic observational exaggeration failed on test**
Anglemyer and colleagues, 2014	14 methodological reviews, 11 of them at 79% observing no significant difference, with a pooled ratio of effect estimates of 1.08 and a 95% confidence interval of 0.96 to 1.22	**The pooled ratio sits at unity, which is the number to quote**
Toews and colleagues, 2024	A pooled ratio of ratios of 1.08 with a 95% confidence interval of 1.01 to 1.15	**Replication of the same estimate a decade later**

Observational designs did not inflate effects; the conditional structure that replaces the pyramid is replication first, then appraised primary research, then structured routine data, then research-supported expert opinion.

Alpha, beta, power and sample size. Define each with its conventional value.

- **Alpha.** The probability of rejecting a true null hypothesis, detecting a difference that does not exist, conventionally set at 0.05 or 5%.
- **Beta.** The probability of failing to reject a false null hypothesis, missing a real difference, conventionally set between 0.05 and 0.2, with a commonly cited cut-off of 20%.
- **Power.** The probability of correctly rejecting the null when a true difference exists, that is 1 minus beta, normally set at 80%, which carries a 20% risk of a false negative.
- **What moves the sample size.** It rises as alpha falls, as power rises, as the targeted effect size shrinks, as population variance grows, and as the baseline incidence of a dichotomous endpoint falls. The required distribution depends on sample size, with the Student t distribution below 60 and normal approximations above 60.

Delta inflation, in one sentence and one consequence. Delta inflation occurs when a power calculation is based on an optimistic or outdated treatment effect size taken from earlier literature.

- **The consequence.** Because sample size varies inversely with the true effect, overestimating the difference produces a trial with inadequate power and a false negative conclusion. In psychiatric research the mechanism is usually improvement in background care or a shift in patient characteristics that narrows the observed difference.

The p value against the confidence interval. A p value is the probability of observing test statistics as extreme as, or more extreme than, the study data under the assumption that the null hypothesis is correct. It yields a dichotomous verdict against the threshold, and it communicates nothing about effect magnitude, direction or precision.

- **The interval.** A 95% confidence interval gives the range of plausible values for the true population parameter: narrow means precise, wide means small sample or high variance.

- **Why the interval decides clinical relevance.** An intervention can reach p below 0.05 while the whole interval falls below the threshold for meaningful symptom improvement.

GOAL	VARIABLES	PARAMETRIC TEST	NON-PARAMETRIC ALTERNATIVE
Compare one group to a known population	Interval and normal outcome	One-sample t-test	**One-sample median test**
Single cohort, binary outcome	Categorical outcome in two categories	Binomial test	**Chi-square goodness of fit**
Compare two independent groups	One nominal predictor in two groups, continuous outcome	Two independent samples t-test	**Wilcoxon and Mann-Whitney test**
Compare two independent groups, categorical outcome	One nominal predictor, nominal outcome	Chi-square test	**Fisher exact test for sparse cells**
Compare three or more independent groups	One nominal predictor, continuous outcome	One-way analysis of variance	**Kruskal and Wallis test**
Compare two paired observations	One within-subject factor at two time points, continuous outcome	Paired samples t-test	**Wilcoxon signed-ranks test**
Compare two paired observations, binary	Matched within-subject factor, binary outcome	McNemar test	**Repeated measures logistic regression**
Compare three or more repeated observations	One within-subject factor at three or more time points	One-way repeated measures analysis of variance	**Friedman test**
Assess linear association	Continuous predictor and outcome	Pearson correlation	**Spearman rank correlation**
Predict a continuous outcome	One or more continuous or categorical predictors	Simple or multiple linear regression, or analysis of covariance	**Non-parametric regression is not specified in the library sources**
Predict a binary outcome	Continuous or categorical predictors, binary outcome	Logistic regression	**Discriminant analysis**
Compare multiple continuous outcomes across groups	One nominal predictor, two or more interval outcomes	One-way multivariate analysis of variance	**Multivariate linear regression**

The selection grid: question, then measurement scale, then design structure. Parametric assumptions are normality of residuals, homogeneity of variance, independence of observations and linearity.

FOUR TRAPS IN ONE BLOCK

Significance against relevance: a very large sample reaches p below 0.05 for a trivial change on a rating scale, and an underpowered study misses a clinically meaningful effect. **Absence of evidence:** failing to reject the null is not evidence of no effect, and is often a beta error from inadequate sample size, high variance or delta inflation. **Concealment against blinding:** concealment shields the sequence before entry to prevent selection bias; blinding conceals assignment after allocation to prevent performance and detection bias. **Paired data with independent tests:** pre-post scores need a paired t-test or a signed-ranks test, and matched binary outcomes need McNemar, not chi-square.

- **Trap.** Testing normality on the raw dependent variable. Test it on the residuals.

HOLD THIS

Alpha 0.05, beta up to 0.2, power 80%, and sample size rises as the target effect shrinks. Design decides causality; tests only measure association.

WHAT EARNS THE MARKS

- **Naming the question domain before the design**: therapy, prognosis, aetiology, meaning
- **Pairing each bias with its control**: concealment for selection, assessor blinding for detection
- **Quoting the pooled ratio at unity**: 1.08 with an interval crossing 1, replicated a decade later
- **Choosing the test from scale and pairing**: McNemar for matched binary, not chi-square

SEE ALSO 11 The landmark trials 12 The epidemiology numbers 16 Medical education and pedagogy

16 · Medical education and pedagogy

PEDAGOGY · TOPIC 16

How would you assess a postgraduate trainee, and how would you give feedback?

VIVA TABLE: MEDICAL EDUCATION, AND THE TEACHING QUESTION IN THE GENERAL VIVA

THE SHAPE OF THE ANSWER

1	Start with the framework	Miller's four levels, each licensing an instrument
2	Separate competence from performance	Shows how against does, and the words are not interchangeable
3	Name the learning theory	Andragogy, the experiential cycle, Bloom's revised verbs
4	Assess programmatically	Many observations aggregated, not one better form
5	Give feedback learner-first	Pendleton's sequence, and why the sandwich fails

Answer a pedagogy question with the framework and the assessment it licenses. Miller's pyramid, introduced by George Miller in 1990, classifies clinical capability into 4 levels, and each level licenses a different instrument.

- **Knows.** Factual recall, tested by multiple choice questions and written papers.
- **Knows how.** Applied knowledge and clinical reasoning, tested by case-based discussion, chart-stimulated recall and vignettes.

- **Shows how.** Demonstration in a controlled environment, tested by objective structured clinical examinations and standardised patients.
- **Does.** Authentic performance in real practice under systemic constraint, tested by workplace-based assessment.
- **The correlation that justifies the pyramid.** Conventional written examinations evaluate the base and correlate only modestly with clinical performance at the apex, r of 0.3 to 0.5, while more than 75% of definitive diagnoses are reached through history and examination alone.
- **The extension.** The pyramid also expands into a three-dimensional prism carrying cognitive, psychomotor and affective domains.

- **RULE** Competence is demonstrated potential in a simulated or testing environment, at shows how. Performance is actual behaviour in routine workplace practice under authentic pressure, at does. Using the words interchangeably is the error the whole framework exists to prevent.

Adult learning, and the experiential cycle. Postgraduate training works under andragogy, pioneered by Malcolm Knowles.

- **What andragogy assumes.** The trainee moves toward self-directed autonomous learning. Accumulated clinical experience becomes an expanding resource, readiness to learn is tied to professional developmental tasks, orientation is problem-centred rather than subject-centred, and internal drivers of mastery, curiosity and patient responsibility predominate over external reward.

Kolb's experiential learning theory operationalises it as a four-stage cycle:

1. **Concrete experience:** managing an acutely agitated patient in casualty.
2. **Reflective observation:** what happened, including one's own emotions and the patient's response.
3. **Abstract conceptualisation:** integrating de-escalation models and psychopharmacological guidance.
4. **Active experimentation:** the next encounter.

- **How identity forms.** Through situated learning in communities of practice, the novice moving from peripheral participation to full integration through deliberate practice.

Bloom, and the revision that changed the verbs. Bloom's taxonomy of 1956 divides learning into 3 domains: cognitive, psychomotor and affective. The original cognitive hierarchy ran through 6 levels of nouns from simple to complex: knowledge, comprehension, application, analysis, synthesis, evaluation.

- **The 2001 revision.** Anderson and Krathwohl recast it into active verbs, remember, understand, apply, analyse, evaluate, create, and paired it with 4 knowledge dimensions.
- **The four dimensions.** Factual, terminology and criteria. Conceptual, theories and diagnostic schemas. Procedural, psychotherapy protocols and treatment algorithms. Metacognitive, self-monitoring and awareness of countertransference.
- **The worked psychiatric example that scores.** Recall the criteria for a manic episode, explain dopamine tract blockade, calculate a stimulus dose, differentiate catatonia from neuroleptic malignant syndrome, critique conflicting trial evidence, formulate an individualised rehabilitation plan.

Competency-based education and programmatic assessment. Competency-based medical education defines the capabilities required for independent practice rather than the duration of training, a shift from behaviourist conditioning to constructivist experiential development.

- **The problem programmatic assessment solves.** No single instrument carries sufficient reliability or validity for a high-stakes credentialing decision.
- **Its three tenets.** Individual assessments are formative instruments for learning and actionable feedback. 6 to 14 aggregated observations across varied presentations and multiple assessors are needed for acceptable generalisability above 0.8. High-stakes progression decisions belong to periodic synthesis by a competency committee reviewing a longitudinal portfolio.
- **Assessment utility.** The multiplicative product of validity, reliability, feasibility, acceptability and educational impact.
- **Entrustable professional activities.** They translate abstract competency lists into operational units of everyday practice that a supervisor can safely entrust once competence is attained, with supervision progressing from observation only, through direct and then indirect supervision, to distant supervision and finally independent practice with supervision of juniors.

VERIFY BEFORE THE EXAM Three examinable specifics are not carried in the library sources for this volume: the number and exact wording of the entrustment supervision levels, the station count and station duration of a standard objective structured clinical examination, and the numeric anchors that divide the rating bands of the mini clinical evaluation exercise. Confirm them from your own programme documents or a medical education text, and at the table describe the structure rather than quoting a count you cannot source.

INSTRUMENT	STRUCTURE	PSYCHOMETRICS TO QUOTE	THE DISCRIMINATOR
Mini clinical evaluation exercise	Direct observation of a focused 15 to 20 minute encounter, followed immediately by 5 to 10 minutes of structured feedback; 7 domains on a 9-point scale, covering medical interviewing, physical examination, professionalism, clinical judgement, counselling, organisation and efficiency, and overall competence	Variance stems from assessor stringency at 15 to 25%, case specificity at 30 to 40%, and true trainee ability at 20 to 35%. Two generalisability studies disagree and should be quoted separately: a multi-institutional study of 1,847 residents across 12 programmes reached 0.81 with 8 observations across different assessors and contexts, while a study of 2,847 assessments needed 14 assessments for 0.80 and fell to 0.72 at 8. Fewer than 4 observations per domain is unreliable, below 0.5	**Assessors give 68% of attention to the encounter and 10% to the rating sheet, so forms of more than 15 items degrade completion accuracy and should be streamlined to 8 to 12 core items**

INSTRUMENT	STRUCTURE	PSYCHOMETRICS TO QUOTE	THE DISCRIMINATOR
Direct observation of procedural skills	Observation of a trainee executing a procedure from start to finish; 6 to 8 domains on a 6-point scale covering preparation, informed consent, aseptic technique, technical execution, post-procedure management and communication	6 to 8 observations achieve 0.75 to 0.82 for common procedures, while 10 to 12 are needed for complex ones. Assessor variance accounts for 18 to 24% of total variance and training reduces it by 18 to 23%	**Predictive validity: trainees in the lowest quartile experienced 1.8 times higher complication rates, with a 95% confidence interval of 1.3 to 2.4, during their first 2 years of independent practice. Procedural expert assessors reach an intraclass correlation of 0.78 against 0.61 for general supervisors**
Case-based discussion	A 15 to 20 minute structured probe of reasoning, investigation and management based on a record the trainee selects	Six to 8 sessions achieve 0.75; structured reasoning frameworks reach an intraclass correlation of 0.72 against 0.51 for unstructured review	**The trainee chooses the record, which is both its strength for reflection and its vulnerability to gaming**
Objective structured assessment of technical skills	7 generic technical skills evaluated on bench models or simulation	Above 0.75 with 4 to 6 assessments	**Simulation-based, so it sits at shows how rather than at does**
Multi-source feedback	15 to 25 items administered across 8 to 12 raters: peers, nursing staff, supervisors and patients	Above 0.7 for reliability; detects 78% of professionalism issues against 34% by direct observation, while direct observation detects 89% of technical deficits against 45% by multi-source feedback	**Peers contribute 18% unique variance on teamwork and nurses 15% on communication, which is the argument for including both**

The workplace instruments. Two structural components define all of them: direct observation in an authentic environment, and immediate contextual feedback.

The Indian regulatory position, stated with its evidentiary weight. The postgraduate regulations of 2000 recommended logbooks for monitoring but did not mandate in-training workplace assessment. In Western settings fewer than a third of clinical encounters were directly observed, with up to 80% of residents having only 1 observed encounter across residency. The National Medical Commission began competency-based reforms in 2019.

A secondary commercial education portal reports that longitudinal workplace assessment is now mandatory under regulations it names as the postgraduate regulations of 2023. The typical requirements it lists are 8 to 12 procedural observations and 8 to 12 clinical evaluation exercises per resident per academic year, sampling across wards, outpatients and emergency, across varied diagnoses and multiple faculty.

The same source adds quarterly portfolio review by the head of department, 4 reviews per year, documented remediation for trainees falling behind, direct observation of teaching for final-year residents, and same-day recording with a trainee self-assessment score and faculty signatures.

- **Verify before the exam.** No primary notification or gazette citation for an instrument of that name appears in the sources, so present the regulation name and the quotas as reported guidance rather than verified statute. The corroborated lineage is the 2000 regulations followed by the commission's competency reforms from 2019.
- **On implementation.** Surveys report that 34% of Indian medical colleges possess validated professionalism assessment tools, 67% rely on paper records and 11% operate comprehensive digital tracking. A 6-hour faculty training workshop raised inter-rater reliability from an intraclass correlation of 0.54 to 0.71.

Feedback, and the model that beats the sandwich. Pendleton's rules, formulated in 1984 and expanded in 2003, run learner-first in 4 steps with a fifth summary, and are designed to conclude in not more than 5 minutes.

1. The trainee identifies 2 or 3 things that went well.
2. The supervisor confirms and elaborates on them.
3. The trainee identifies 2 or 3 things that could be done differently, in their own words.
4. The supervisor offers constructive alternatives and concrete strategies to bridge the gap.
5. Either party summarises the actionable points.

- **Why the ordering works.** Learner-first disrupts the power asymmetry, stimulates self-assessment and promotes ownership. Opening with positives lowers defensiveness, and the phrase "what could be done differently" frames improvement as forward-looking rather than punitive.
- **Its limitations.** Rigidity, a need for 5 or 6 practice encounters before faculty use it naturally, and the risk that the supervisor simply parrots the trainee's self-critique.
- **Why the sandwich fails.** Praise then critique then praise dilutes the corrective content: learners remember the praise and discount the critique, or read the praise as an insincere precursor to criticism. Alternative conversational frameworks that prioritise a learner-defined agenda are also worth naming.

Microteaching, in outline. It is a laboratory technique that reduces teaching complexity by limiting class size, session duration and instructional scope to a single teaching skill.

- **Its target skills.** Set induction, stimulus variation, questioning technique, use of visual aids, reinforcement and closure.
- **Its cycle.** Plan, teach while observed or recorded, structured feedback using Pendleton's rules, re-plan, re-teach to a different peer group, re-feedback.
- **Verify before the exam.** The class size, session length and step count are not carried in the library sources, so describe the cycle and confirm the parameters from your own department's format.

The journal club, in outline. It links literature to bedside care through a focused clinical question and a systematic search of indexed databases, then appraises on three axes.

- **Internal validity.** Randomisation technique, allocation concealment, blinding, completeness of follow-up and intention-to-treat analysis.
- **Results.** Interpreting absolute risk reduction, relative risk, hazard ratios and numbers needed to treat rather than p values alone.
- **External validity.** The transportability of the result to an Indian public health context with its comorbidity, affordability and infrastructure.

- **The appraisal scaffold.** Reporting guidelines: the trial standard for randomised trials, the observational standard for cohort and case-control studies, and the review standard for systematic reviews.

TRAP Three that cost marks. Running a workplace assessment as a one-off summative examination, which induces strategic gaming, cherry-picked easy cases and lenient faculty; it is formative, and high-stakes decisions come only after programmatic aggregation. Relying on a single observation, when case specificity accounts for 30 to 40% of score variance and rater stringency 15 to 25%. And merging the two discordant generalisability studies into a single range when they report 0.81 and 0.72 for the same number of observations.

HOLD THIS

Knows, knows how, shows how, does; written papers correlate with the apex at only 0.3 to 0.5. Reliability comes from 6 to 14 observations across assessors, not from a better form.

WHAT EARNS THE MARKS

- **Pairing each Miller level with its instrument**: written paper at knows, workplace assessment at does
- **Keeping competence and performance apart**: simulated potential against authentic behaviour
- **Locating reliability in sampling**: many observations across assessors, not a longer form
- **Ordering feedback learner-first**: the trainee names the strengths and the changes before you do

SEE ALSO 15 Research methodology 14 Ethics and the codes 23 Scale and test material spotters

17 · Psychiatric emergencies and disaster psychiatry

EMERGENCIES · TOPIC 17

An agitated patient is brought to casualty. Take me through your management.

VIVA TABLE: EMERGENCIES, AND THE AGITATION STATION IN THE CLINICAL EXAMINATION

THE SHAPE OF THE ANSWER

1 Define and classify	Emergency, crisis type, and the three mediating factors
2 Climb the ladder	Safety, de-escalation, offer, boundary, medication, force, restraint
3 Suicide precautions	Direct enquiry, environmental sweep, line of sight
4 The statutory frame	Least restrictive setting, time-limited emergency treatment
5 Disaster psychiatry	Problem grid, three stages, psychological first aid

Define the emergency, then classify the crisis. A psychiatric emergency is an acute disturbance of thought, feeling or action requiring immediate therapeutic intervention, and the patient is in a state of crisis in which baseline coping has been overwhelmed by real or perceived circumstances.

- **Three kinds of crisis.** Maturational, the predicted life transitions of leaving home, marriage or parenthood. Situational, the sudden unanticipated event of acute illness, bereavement or job loss. Adventitious and social, the large-scale accidental or deliberate event of natural disaster, riot, industrial accident or terrorist attack.
- **Three mediating factors.** The subjective perception of the event, the availability of situational supports, and access to functional coping mechanisms.

The common presentations are acute suicidal or homicidal behaviour, severe agitation and aggression, acute psychosis and delirium, severe affective episodes, substance intoxication and

withdrawal, neuroleptic malignant syndrome and serotonin syndrome, and panic attacks and acute conversion reactions.

- **The affective figures.** Depression is the primary complaint in 0.6% of emergency department presentations, while up to 1 in 5 emergency department patients carry depressive illness. Diagnostic evaluation assesses 5 or more symptoms over a 2-week duration.

STEP	ACTION	HOW IT IS DONE
1	Ensure scene safety	Do not act alone; remove hazardous objects; keep exit routes unobstructed
2	Verbal de-escalation	Calm, low, firm tone; preserve personal space; open posture
3	Voluntary time-out	Offer relocation to a quiet, low-stimulus room
4	Clear boundary setting	State clearly and respectfully that aggressive physical action is unacceptable
5	Voluntary pharmacotherapy	Offer oral medication to help restore behavioural control; the library sources do not carry the dose, so name the agent and confirm the dose from a protocol
6	Show of force	**Assemble 4 to 6 trained staff within visible sight; presence alone often deters escalation**
7	Restraint or seclusion	**Emergency sedation or physical restraint as a last resort to avert imminent harm, with continuous or frequent vital monitoring, assessment of circulation, and removal as soon as safety is re-established**
8	Structured debriefing	Review triggers with the patient once calm, and conduct a multidisciplinary staff debriefing

The ascending ladder. Warning signs that precede overt violence: tense or hostile facial expression, pacing, motor restlessness, raised vocal volume, erratic movement, prolonged staring, withdrawal or unresponsiveness, paranoid delusions and direct verbal threats.

Suicide risk and the environmental sweep. Any patient expressing suicidal thought requires immediate stratification through direct non-judgmental enquiry.

- **What the enquiry covers.** Passive ideation, active intent, specific plan, access to lethal means and perceived hopelessness. Asking directly does not induce suicidal thought in a person who is not suicidal.
- **The sweep.** A thorough search of patient and belongings, with systematic confiscation of sharp objects, belts, shoelaces, drawstrings, cords, lighters and medications.

- **The watch.** One-to-one observation with uninterrupted line of sight, and placement in a high-visibility room away from stairwells and unmonitored exits, with finger foods and without standard metal utensils.

The statutory frame, in words. Indian law upholds care in the least restrictive setting, so outpatient management takes priority over inpatient detention wherever clinically feasible.

- **Emergency treatment.** Treatment by a registered medical practitioner is time-limited and exists to prevent imminent loss of life or grievous injury.
- **Supported admission.** For a person needing high support, it requires certification and follows failure of less restrictive alternatives, with review board scrutiny.
- **Restraint.** Restraint as punishment or for staff convenience is barred. Physical restraint requires explicit psychiatrist authorisation, documented justification and regular charting.
- **After an attempt.** An attempt at suicide carries a statutory presumption of severe stress, exempting the person from penal prosecution and directing the state to provide treatment and rehabilitation.
- **Verify before the exam.** The section numbers, the time limits in hours and in days, and the review cadence are not carried in the library sources for this topic, so quote the provision only after reading it off the Act.

DOMAIN OF DISASTER-ASSOCIATED PROBLEM	PRE-EXISTING	EMERGENCY-INDUCED	RESPONSE-INDUCED
Social	Poverty, discrimination	Family separation, loss	**Overcrowding and poor privacy in relief settings**
Mental health	Chronic severe mental illness, substance dependence	Grief, acute stress, post-traumatic stress disorder, depression	**Anxiety generated by poor aid communication**
Service delivery	Limited specialist access	Facility damage, supply disruption	**Uncoordinated aid and untrained staff**

The World Health Organization classification is a problem-domain grid, not a time course. Naming it as a grid and the recovery stages as a timeline is itself examinable.

The epidemiology of mass trauma. Almost everyone affected experiences transient distress, insomnia, sadness, aches, fatigue and anxiety, and most recover spontaneously.

- **The conflict figure.** In populations affected by armed conflict or war within the preceding 10 years, approximately 22%, about 1 in 5, suffer a mental disorder: mild forms of depression, anxiety and post-traumatic stress disorder account for 13%, and moderate to severe disorders for 9%. Depression prevalence rises with age and is more frequent among women.

The operational response runs in three stages:

1. **Acute, immediate to 4 weeks.** Shock, confusion, arousal and physical survival needs. The focus is psychological triage, basic stabilisation and psychological first aid.
2. **Intermediate or chronic, 1 to 6 months.** Persistent pathology, complicated grief, major depression and formal post-traumatic stress disorder emerge, and the focus moves to secondary screening and structured psychotherapy.
3. **Long-term recovery, beyond 6 months.** Rehabilitation, community resilience, after-action review and health system integration.

Triage and the screening instruments by phase. Psychiatric triage separates transient adaptive distress from severe decompensation or high-risk neuropsychiatric emergency requiring immediate diversion.

- **Acute stage, 0 to 4 weeks.** A rapid triage tool identifies individuals with high-risk trauma exposures needing urgent behavioural stabilisation.
- **Chronic stage, 1 to 6 months.** Paediatric screening uses the child post-traumatic stress symptom scale and the adolescent primary care traumatic stress screen. Adult screening uses the post-traumatic stress checklist and the revised impact of events scale.

Psychological first aid, and what it is not. It is an evidence-informed modular acute intervention for children, adolescents, adults and families in the immediate aftermath of disaster, recognised as the acute psychosocial intervention of choice.

- **What it is not.** It is not formal psychiatric treatment, it does not assume that survivors will develop psychiatric illness, and it does not require a licensed mental health specialist.
- **Who delivers it.** Frontline responders, teachers, community volunteers and general health staff, relying on human presence and practical support in field hospitals, shelters, evacuation centres and casualty units.

Its five empirically validated elements:

1. **Safety:** removing people from hazards and shielding them from traumatic sights.
2. **Calm:** reducing autonomic arousal with grounding and slow paced breathing.
3. **Self- and collective efficacy:** engaging survivors in their own welfare decisions and practical problem-solving.
4. **Connectedness:** facilitating reunification with relatives, neighbours and community networks.
5. **Hope:** providing accurate verifiable realistic information about relief and immediate next steps.

- **Its eight core actions.** Contact and engagement, safety and comfort, stabilisation where needed, information gathering on current needs and concerns, practical assistance, connection with social supports, information on coping, and linkage with collaborative services.

PHASE	WHAT IS OFFERED	WHAT IS AVOIDED
Acute, 0 to 4 weeks	Psychological first aid; skills for psychological recovery for protracted distress; for children, child-adult relationship enhancement, parent-child interaction therapy and child-parent psychotherapy	**Routine psychological debriefing: a compulsory single-session recounting of traumatic memory, which lacks evidence of efficacy and can impede natural resilience. A systematic review found little or no evidence to support routine debriefing after traumatic birth**
Chronic, 1 to 6 months	Structured screening, then trauma-focused cognitive behavioural therapy and eye movement desensitisation and reprocessing; the humanitarian intervention guide supplies primary-care algorithms in low-resource emergencies	**Applying exposure-based therapies during the acute impact phase, where they are treatments for established illness rather than field interventions**

The Zunin and Myers phases, which Indian examiners ask by name. The four-phase model of collective disaster reaction runs heroic, honeymoon, disillusionment and reconstruction.

- **The durations.** The heroic phase of altruistic rescuing lasts a day to weeks. The honeymoon phase of relocation to relief camps lasts 2 to 4 weeks. The disillusionment phase, as relief is weaned, lasts 3 to 36 months before restoration to harmony.
- **Trap.** Merging three different structures. Keep this model separate from the problem-domain grid, which is not a time course, and from the three-stage service timeline, which is a delivery schedule rather than a model of collective emotional reaction.

MASS TRAUMA IN AN INDIAN DISTRICT, AND BUILDING BACK BETTER

Community-based psychosocial support aligns with the decentralisation objectives of the national and district programmes. District teams of psychiatrist, clinical psychologist, psychiatric social worker and psychiatric nurse operate district-level inpatient units of 10 beds and conduct 4 satellite clinics monthly.

Those clinics sit at community and primary health centres. The model traces historically to the primary-care psychiatry projects at Sakalwara from 1975 and Raipur Rani from 1977.

The World Health Organization principle to name is building back better, that an emergency converts acute aid into lasting system reform.

The precedents worth quoting: after the 2004 tsunami, Sri Lanka expanded infrastructure so that every district established formal mental health infrastructure where one-third had it before; and after Typhoon Haiyan in 2013, the Philippines trained over 1300 community and health workers across primary facilities.

Two more: Syria expanded services beyond hospitals into primary and social care across more than 12 cities, and the 2024 Ukrainian operational roadmap coordinated over 250 multisectoral actors.

- **TRAP** Confusing psychological first aid with critical incident stress debriefing. First aid is non-intrusive, practical and needs-oriented, and requires no emotional ventilation or recounting of the event; a single-session formal debriefing that forces disclosure is avoided because of the risk of secondary traumatisation.

- **Trap.** Pathologising a normal reaction. Most survivors show distress, and diagnosing major depression or post-traumatic stress disorder within the first few days converts a normal human response into an illness. Formal post-traumatic stress disorder requires persistence beyond 1 month, and the acute task is supportive stabilisation.

HOLD THIS

De-escalate, offer, set boundaries, offer medication, show force with 4 to 6 staff, restrain last.
In conflict-affected populations, 22% carry a disorder: 13% mild, 9% moderate to severe.

WHAT EARNS THE MARKS

- **Climbing the ladder in order**: restraint arrives last, with monitoring and early removal
- **Doing the environmental sweep explicitly**: ligature points, sharps, cords, medication, line of sight
- **Keeping the three disaster structures apart**: problem grid, service timeline, collective phases
- **Refusing to pathologise early distress**: post-traumatic stress disorder needs persistence beyond 1 month

SEE ALSO 14 Ethics and the codes 19 Prevention and positive psychiatry 21 The electroconvulsive therapy machine

18 · Neurobiology at the table

NEUROBIOLOGY · TOPIC 18

Name the dopamine pathways and tell me what each one costs you clinically.

VIVA TABLE: NEUROBIOLOGY, AND THE SIDE-EFFECT EXPLANATION IN ANY DRUG QUESTION

THE SHAPE OF THE ANSWER

1 **Classify the messenger**	Transmitter, hormone, and the ones that are both
2 **Four dopamine pathways**	Origin, target, function, then the clinical consequence
3 **Receptor families**	Which are ionotropic, which are metabotropic, and the polarity
4 **Systems, not molecules**	Stress axis, neurotrophic signalling, default mode network
5 **Close on genomics**	Shared polygenic risk, not a chemical imbalance

Classify before you describe. A neurotransmitter acts locally across the synaptic cleft on postsynaptic receptors; a neurohormone enters systemic circulation to act at a distant tissue. Some messengers, epinephrine and corticotropin-releasing factor among them, do both.

- **By structure.** Monoamines, subdivided into catecholamines from phenylalanine and tyrosine, indolamines from tryptophan, and the imidazole amine histamine from histidine. Amino acids, excitatory glutamate and aspartate from the glial glutamine cycle and inhibitory GABA and glycine. The quaternary amine acetylcholine from choline and acetyl coenzyme A. Purines. Neuropeptides.
- **What defines a monoamine.** One amino group, an aromatic ring and a two-carbon chain. Dietary precursors undergo sequential hydroxylation and decarboxylation, and transmission is terminated by high-affinity presynaptic reuptake and by monoamine oxidase and catechol-O-methyltransferase.

DOPAMINERGIC PATHWAY	ORIGIN AND TARGET	FUNCTION	THE CLINICAL CONSEQUENCE
Mesolimbic	Ventral tegmental area to nucleus accumbens in the ventral striatum	Reward and reinforcement	**Hyperstimulation of D2 receptors here underlies positive psychotic symptoms in schizophrenia and in psychotic mania**
Mesocortical	Ventral tegmental area to dorsolateral and ventromedial prefrontal cortex	Sustained attention, working memory, sequential problem solving	**Underactivation or structural hypofunction produces cognitive deficit, distractibility and negative symptoms**
Nigrostriatal	Substantia nigra pars compacta to dorsal striatum, caudate and putamen	Motor planning and execution	**Hypofunction gives parkinsonian rigidity and bradykinesia; D2 antagonism gives extrapyramidal symptoms, and persistent blockade risks tardive dyskinesia through receptor upregulation. Substantia nigra neuron counts decline progressively after age 65**
Tuberoinfundibular	Arcuate and periventricular hypothalamic nuclei to the median eminence	Dopamine acts as prolactin-inhibiting factor	**Pathway underactivity or pharmacological blockade elevates prolactin; the pathway also participates in endocrine stress reactivity and sleep regulation**

Dopamine acts on 5 receptor subtypes: D1-like, D1 and D5, stimulate adenylate cyclase and raise cyclic AMP; D2-like, D2, D3 and D4, inhibit it. D2 receptors sit postsynaptically and presynaptically as autoreceptors, where activation lowers cyclic AMP and reduces presynaptic firing.

Noradrenaline, with the ageing figures. Norepinephrine is synthesised from dopamine within vesicles by dopamine beta-hydroxylase. Nearly all noradrenergic innervation of cortex and forebrain arises from the pigmented locus coeruleus in the rostral pons, whose neurons begin to be lost between age 30 and 40.

- **Alpha-1.** Postsynaptic, activating phospholipase C, raising intracellular calcium and excitability.
- **Alpha-2.** Postsynaptic in prefrontal networks, and presynaptic as autoreceptors that suppress adenylate cyclase and reduce calcium influx, acting as a brake on burst firing.
- **Beta.** Stimulates adenylate cyclase. Frontal beta receptors remain stable with age, while precentral, temporal and cingulate cortices decline linearly.
- **The depression finding.** Post-mortem studies show compensatory upregulation of postsynaptic beta-adrenergic receptors, reflecting prolonged transmitter depletion.

Serotonin, family by family. Serotonin is synthesised from tryptophan in the midbrain and pontine raphe nuclei.

- **The 5-HT1 family.** Couples to inhibitory G proteins. 5-HT1A receptors act as somatodendritic autoreceptors in the raphe and as postsynaptic receptors in the limbic system, dampening defensive rage. 5-HT1D receptors are presynaptic terminal autoreceptors.
- **The 5-HT2 family.** Couples to Gq, activating phospholipase C and raising calcium. Postsynaptic limbic 5-HT2 receptors facilitate defensive rage and behavioural arousal, with increased receptor density in suicidal and depressed cohorts post-mortem.
- **The ageing figures.** With ageing, 5-HT2 density falls by 20% to 50% and 5-HT1 by up to 70%.
- **The 5-HT3 receptor.** The sole ionotropic serotonin receptor, a ligand-gated cation channel permeable to sodium and potassium, mediating rapid depolarisation and the peripheral emetic reflex.
- **The rest.** The 5-HT4, 5-HT6 and 5-HT7 families couple to Gs.

The permissive hypothesis, stated as a mechanism. Central serotonin depletion creates a permissive neurochemical state in which fluctuations of catecholaminergic transmission dictate affective polarity: a concurrent fall in noradrenaline precipitates depression, and a surge in catecholaminergic activity precipitates mania.

- **The supporting observation.** Reduced cerebrospinal fluid 5-hydroxyindoleacetic acid in depressed and suicidal cohorts.

Excitation, inhibition and excitotoxicity. Glutamate is synthesised from glutamine and recycled through astrocytes in the glutamate-glutamine cycle, acting on 3 ionotropic families, N-methyl-D-aspartate, AMPA and kainate, and on metabotropic receptors.

- **How excitotoxicity happens.** The N-methyl-D-aspartate and AMPA channels admit calcium and sodium, producing rapid depolarisation. Sustained firing without adequate repolarisation intervals allows excessive intracellular calcium accumulation, activating destructive proteases and endonucleases and causing neuronal death.
- **Two clinical translations.** Unsynchronised paroxysmal depolarising shifts occurring approximately 1 second apart generate clinical seizure activity. Deficient glutamatergic extinction in hippocampal memory traces has been implicated in persistent intrusive recollection.
- **GABA.** Synthesised from glutamate by glutamic acid decarboxylase. Ionotropic GABA-A receptors gate chloride channels, and inward chloride hyperpolarises the membrane, while metabotropic GABA-B receptors open potassium channels for prolonged hyperpolarisation.
- **Too much and too little.** Excessive GABAergic stimulation gives sedation, respiratory suppression and coma. Deficient tone allows runaway glutamatergic excitation as mania, panic or convulsion.
- **Glycine has two jobs.** Inhibitory at strychnine-sensitive chloride channels in brainstem and cord, and in cortex and hippocampus an obligate co-agonist at the N-methyl-D-aspartate receptor complex.

Cholinergic circuits and the striatal balance. Acetylcholine is synthesised by choline acetyltransferase and inactivated by acetylcholinesterase, projecting from two hubs.

- **Hub one, the basal forebrain complex.** The nucleus basalis of Meynert and diagonal band of Broca innervate hippocampus, amygdala and cortical mantle for memory encoding, sustained attention and sensory gating. Their cells atrophy after age 60 with a 10% to 30% reduction in cortical muscarinic receptor density, and their degeneration is the hallmark of Alzheimer dementia.
- **Hub two, the mesopontine tegmental complex.** It projects to striatum, thalamus and cerebellum for motor modulation and sleep architecture.
- **Receptors.** Metabotropic muscarinic M1 through M5, and ionotropic nicotinic pentamers.
- **The striatal balance, which explains the side effect.** In the dorsal striatum, nigral dopamine exerts tonic inhibition over large aspiny cholinergic interneurons. D2 blockade removes that brake, causing

excessive local acetylcholine release, and unopposed cholinergic excitation over basal ganglia output precipitates acute dystonia, cogwheel rigidity, resting tremor and akathisia, which anticholinergic agents relieve by restoring equilibrium.

- **What chronic blockade does instead.** Compensatory receptor upregulation, manifesting as tardive dyskinesia.

The hypothalamic-pituitary-adrenal axis, and the feedback failure. Neural stress inputs converge on the paraventricular nucleus, releasing corticotropin-releasing factor and arginine vasopressin into the hypophyseal portal plexus. The factor acts both as a central limbic neurotransmitter and as a hypophyseotropic neurohormone.

- **The cascade.** It stimulates the anterior pituitary to secrete adrenocorticotropic hormone, which drives adrenal cortisol.
- **The two limbic controllers.** The amygdaloid complex activates the paraventricular nucleus through excitatory output. The hippocampus, dense in glucocorticoid and mineralocorticoid receptors, exerts tonic inhibitory feedback.
- **What happens in melancholic depression.** Sustained stress produces excessive central factor release, adrenal hypertrophy and hypercortisolaemia, and persistent glucocorticoid exposure downregulates hippocampal glucocorticoid receptors, impairing negative feedback.
- **The test that demonstrated it.** The overnight dexamethasone suppression test, at a dexamethasone dose given in the library's textbook source as 1 mg, where non-suppression of morning plasma cortisol demonstrates feedback insensitivity.
- **Verify before the exam.** The diagnostic post-suppression threshold is not carried in the sources, so do not quote a cut-off.

Neuroplasticity and the neurotrophic hypothesis. Activity-dependent plasticity depends on retrograde signalling between postsynaptic densities and presynaptic active zones.

- **The retrograde step.** Postsynaptic calcium influx through N-methyl-D-aspartate receptors recruits calcium and calmodulin-dependent protein kinase II and activates retrograde messengers, including nitric oxide, that alter presynaptic vesicle release machinery.
- **What the neurotrophin does.** Brain-derived neurotrophic factor, signalling through tropomyosin receptor kinase B, supports neuronal survival, dendritic arborisation and synaptic maintenance in hippocampus and prefrontal cortex.
- **What stress does to it.** Sustained stress and elevated glucocorticoids suppress hippocampal transcription of the factor, causing CA3 dendritic retraction and reduced adult neurogenesis in the subgranular zone of the dentate gyrus.
- **What treatment does.** Antidepressants, electroconvulsive therapy and physical exercise raise phosphorylation of the cyclic AMP response element-binding protein, upregulating synthesis and promoting dendritic remodelling.
- **Verify before the exam.** The common functional Val66Met polymorphism impairs activity-dependent secretion. Its population frequency and baseline serum reference ranges are not carried in the sources.

The default mode network, in three lines. Its core nodes are the medial prefrontal cortex, the posterior cingulate cortex and precuneus, and the bilateral inferior parietal lobules, with subsystem connections to hippocampus and parahippocampal cortex.

- **When it is active.** During passive rest, autobiographical recall, future planning, self-referential introspection and theory-of-mind processing. It deactivates under external task demand, anti-correlated with the central executive and dorsal attention networks.
- **In major depressive disorder.** It is hyperconnected and fails to deactivate during cognitive tasks, which correlates with rumination, self-blame and intrusive negative self-focus.

- **In schizophrenia.** Anterior nodes are hyperconnected and communication with the salience network is aberrant, impairing the boundary between internally generated thought and external auditory perception.

GENOMIC FACTOR	DISORDERS	BIOLOGY AND CELL TYPE
Bipolar disorder and schizophrenia	Schizophrenia, bipolar disorder	**Enrichment in excitatory cortical neurons; approximately 70% of the schizophrenia genetic signal is shared with bipolar disorder, which is the single most asked number here**
Internalising conditions	Major depressive disorder, anxiety disorders, post-traumatic stress disorder	**Variants act on non-neuronal oligodendrocytes, implicating myelin and white-matter connectivity rather than excitatory neurons**
Compulsive disorders	Obsessive-compulsive disorder, anorexia nervosa, Tourette disorder	**Shared corticostriatal regulatory circuitry**
Neurodevelopmental conditions	Autism spectrum disorder, attention-deficit hyperactivity disorder	**Early embryonic and fetal neurodevelopmental pathways**
Substance use disorders	Alcohol, nicotine and illicit substance dependence	**Shared mesolimbic reward and hedonic pathways**

The cross-disorder analysis drew on more than 1 million individuals with at least 1 of 14 psychiatric disorders and 5 million undiagnosed controls, identifying 238 shared variants organised into 5 genomic factors. More than half of those diagnosed receive a second or third diagnosis across a lifetime, and approximately 41% meet criteria for 4 or more disorders.

FOUR TRAPS IN ONE BLOCK

Autoreceptor polarity: postsynaptic D2 activation drives motor and limbic responses, while presynaptic D2 autoreceptor activation inhibits adenylate cyclase, suppresses calcium entry and reduces synthesis and release. **Serotonin syndrome against neuroleptic malignant syndrome:** serotonin syndrome comes on within minutes to hours with neuromuscular hyperactivity, hyperreflexia, clonus, tremor and shivering; the malignant syndrome comes on insidiously over days with lead-pipe rigidity and hyporeflexia, from dopamine D2 antagonism. **Receptor classes:** 5-HT3 is ionotropic and nicotinic receptors are ionotropic, while GABA-B and metabotropic glutamate receptors signal through G proteins, so the monoamine-equals-metabotropic rule fails. **Genetic specificity:** claiming distinct architectures for schizophrenia and bipolar disorder is wrong, because roughly 70% of the signal is shared.

HOLD THIS

Four dopamine pathways, 5 receptor subtypes, 70% genetic overlap between schizophrenia and bipolar disorder. Do not offer a chemical imbalance: offer receptor change, second messengers, neurotrophic signalling and shared polygenic risk.

WHAT EARNS THE MARKS

- **Ending each pathway on its clinical cost**: prolactin, extrapyramidal signs, negative symptoms
- **Getting autoreceptor polarity right**: presynaptic D2 activation reduces synthesis and release
- **Explaining the anticholinergic rescue mechanically**: D2 blockade releases the brake on striatal interneurons
- **Replacing the imbalance story**: receptor change, second messengers, neurotrophins, shared polygenic risk

SEE ALSO 05 Schizophrenia beyond D2 07 Rapid-acting antidepressants 20 Drug spotters

19 · Prevention and positive psychiatry

PREVENTION · TOPIC 19

What is positive psychiatry, and how would you classify prevention in mental health?

VIVA TABLE: PUBLIC HEALTH AND PREVENTION, AND THE PROMOTION QUESTION IN THE GENERAL VIVA

THE SHAPE OF THE ANSWER

1	Define positive psychiatry	Promotion of wellbeing through positive psychosocial factors
2	Name the three constructs	Resilience, wisdom, meaning and purpose, each measurable
3	Answer when, who, how	Three frameworks, three different questions
4	Keep the frameworks apart	Risk without symptoms is selective, not secondary
5	Land it in a district	School, clinic, rehabilitation, with the Indian lineage

Positive psychiatry has a definition, so give it verbatim in substance. It is the science and practice of psychiatry that focuses on the study and promotion of mental health and wellbeing through enhancement of positive psychosocial factors, conceptualised and popularised by Dilip Jeste.

- **The premise, with its number.** Approximately 20% of the population experiences a diagnosable mental disorder at any given time, yet mental health exists across the whole population, and positive attributes can be identified and enhanced in clinical and non-clinical groups alike.
- **What it adds to the encounter.** Strengths-based enquiry: identifying personal strengths, competencies and sources of pleasure; evaluating positive social networks and supportive relationships; and examining the protective factors that maintained wellness or prevented recurrence over a period such as the preceding 5 years.

DOMAIN	TRADITIONAL PSYCHIATRY	POSITIVE PSYCHIATRY
Target population	Those with a manifest disorder	Those in distress or at elevated risk of mental or physical disorder
Assessment focus	Psychopathology, symptom checklists, diagnostic criteria	**Positive attributes, character strengths, psychological assets**
Research focus	Risk factors, pathophysiology, vulnerability	**Protective factors, positive neuroplasticity, coping**
Primary goal	Symptom reduction, distress relief, relapse prevention	**Functional recovery, wellbeing, successful ageing, post-traumatic growth**
Level of prevention	Predominantly tertiary	**Weighted to primary prevention and health promotion**

The three measurable constructs. Definition, then evidence, for each.

- **Resilience.** The capacity to adapt, recover and maintain functioning under significant adversity, trauma or severe stress, held as both trait and dynamic process.
- **Its evidence.** Higher resilience correlates with emotional stability, social connectedness and fewer depressive episodes. Structured group interventions using relaxation, cognitive reframing and shared lived experience raise perceived resilience in older people with chronic physical conditions.
- **Wisdom.** A multi-component trait of prosocial attitude and behaviour, empathy, altruism and compassion, alongside self-reflection, affective regulation, tolerance of uncertainty and diverse viewpoints, and rational decision-making.
- **Its evidence.** Greater wisdom across adult life is linked to subjective wellbeing, life satisfaction and better physical and psychological outcomes. Structured 8-session group programmes targeting emotional regulation and prosocial behaviour yield gains in self-compassion and wellbeing.
- **Meaning and purpose in life.** The appraisal that one's existence and daily activity carry coherence, value and direction. Strong purpose is a recognised protective factor against suicidal ideation and self-harm.
- **Its evidence.** Structured life review, delivered individually or in groups over 2 to 8 sessions of 30 to 90 minutes each, produces reductions in depressive symptoms and improved existential wellbeing in older people and in advanced medical illness.

LEVEL	TIMING AND OBJECTIVE	PSYCHIATRIC EXAMPLES
Primary prevention	Pre-pathogenesis, before biological onset, to eliminate causal risk factors, boost protective factors and reduce incidence	**Thiamine and vitamin B12 supplementation to eradicate deficiency dementias; universal classroom behaviour programmes such as the good behaviour game in the first and second grades; parenting skills training; school and community substance education**

LEVEL	TIMING AND OBJECTIVE	PSYCHIATRIC EXAMPLES
Secondary prevention	Latent or subclinical phase, early identification and rapid intervention to arrest progression, shorten duration and lower prevalence	**Routine screening for depressive symptoms and suicide risk in primary care; screening for hazardous alcohol consumption before a use disorder develops; youth access hubs for those aged 12 to 25 that curtail the duration of untreated illness; crisis telephone helplines**
Tertiary prevention	Established clinical phase, to prevent complications, arrest disability, reduce relapse and optimise quality of life	**Maintenance pharmacotherapy in bipolar disorder and schizophrenia; supported employment through individual placement and support, where competitive employment rates run more than 2 times higher than traditional prevocational training; family psychoeducation; postvention bereavement groups**

The Leavell and Clark levels of 1953 answer the question "when". The Institute of Medicine framework, adopted in 1994 and updated in 2009, answers the question "who".

The "who" framework, with its three tiers. One block per tier.

- **Universal.** Delivered to an entire unselected population regardless of individual vulnerability: school-wide social and emotional learning curricula, public awareness campaigns, dietary nutrient fortification such as folate, and public safety messaging.
- **Selective.** Targets asymptomatic subgroups with elevated statistical risk on biological, psychological or social grounds: group psychotherapy for asymptomatic offspring of parents with major depressive disorder, support services for trauma survivors, victims of hate-motivated abuse or marginalised populations facing chronic bullying, and targeted coping interventions for people with serious long-term physical illness.
- **Indicated.** Targets high-risk individuals who already show detectable subclinical signs, mild subthreshold symptoms or biological markers without meeting full criteria: specialised monitoring for young people meeting clinical high risk criteria for psychosis, acknowledging the substantial false-positive rate; cognitive behavioural therapy for subthreshold anxiety; and lifestyle and pharmacological modification for metabolic abnormality secondary to second-generation antipsychotic therapy.

RULE Secondary prevention requires a latent, subclinical or early disease process detected by screening. Intervening in a wholly asymptomatic individual purely because of high epidemiological risk is selective prevention under the "who" framework, not secondary prevention under the "when" framework. Secondary sources conflate the two, and the examiner is testing whether you do.

The "how" framework, which completes the answer. Comprehensive prevention requires an upstream population-level frame built on the social determinants of health: concentrated poverty, substandard housing, food insecurity, discrimination, educational disparity, adverse built environment and violence exposure.

Those structural determinants act on the individual through three pathogenic mechanisms:

1. **Restricted choices:** deprivation forces reliance on inexpensive nutrient-poor high-calorie food that raises vulnerability to metabolic syndrome and depression.
2. **Persistent neurobiological stress:** chronic adversity produces enduring activation of autonomic and neuroendocrine pathways.
3. **Biological interaction:** adverse environments act on the genome through gene-by-environment interaction and epigenetic modification.

- **What actionable prevention therefore means.** Changing public policy, legislation, judicial decision and municipal zoning, and changing social norms against discrimination and social exclusion.
- **The shared limitation.** Neither the "when" nor the "who" framework resolves systemic disparity in access: well-resourced populations receive extensive preventive services while disadvantaged groups meet substantial barriers.

The interventions with evidence. Six worth naming.

- **The good behaviour game.** Initiated in the first and second grades, it rewards prosocial classroom interaction, and longitudinal follow-up shows reduced adult rates of substance dependence, antisocial personality disorder and suicidal ideation.
- **Mindfulness.** It enhances functional neurocircuitry for emotional regulation and empathy while mitigating autonomic and neuroendocrine stress reactivity.
- **Yoga.** It promotes resilience and self-regulation, with measurable improvement in quality of life as an adjunct in schizophrenia.
- **Compassion-focused meditation.** It reduces subjective anxiety and dampens physiological reactivity to social evaluative stress.
- **Youth early access hubs.** Low-stigma entry points that shorten the duration of untreated illness.
- **Supported employment.** Individual placement and support more than doubles competitive employment against prevocational training.

PREVENTION AND PROMOTION IN A DISTRICT PROGRAMME

The lineage runs from the Bhore Committee in 1946, the Mudaliar Committee in 1962 and the Srivastava Committee community health volunteer scheme in 1974.

From there to the Raipur Rani project from 1975 to 1981 covering 100 villages under N.N. Wig, and the family-inclusive camp models begun in Amritsar in 1950 by Vidya Sagar. The national programme was drafted in 1981 and approved in August 1982.

Primary, at district level: school life-skills education, college counselling, workplace stress seminars and community awareness camps with local self-government institutions, teachers and traditional faith healers.

Secondary: early identification clinics, 10-bed district inpatient facilities, 4 monthly satellite clinics at community and primary health centres, structured 9 days of training for primary care medical officers, and suicide prevention helplines.

Tertiary: psychosocial rehabilitation, vocational reintegration and partnership with self-help groups, caregiver forums and non-governmental organisations. The Indian community trial evidence to name spans the task-shifting and collaborative care studies and the community care trial for schizophrenia.

VERIFY BEFORE THE EXAM Indian examiners commonly ask for the level that precedes primary prevention in the Leavell and Clark sequence and for its definition. The library sources for this volume do not define or mention that term, so it is not asserted here. Confirm the definition and its psychiatric examples from a preventive medicine or public health text before the viva.

TRAP Two misplacements. Treating tertiary prevention as passive terminal care, when in psychiatry it is active: relapse-prevention pharmacotherapy, family psychoeducation, cognitive adaptation and supported employment. And classifying a clinical high risk programme as selective prevention, when attenuated symptoms are already manifest, which makes it indicated prevention under the "who" framework or secondary prevention under the "when" framework.

HOLD THIS

When, who, how: Leavell and Clark levels, the Institute of Medicine tiers, and the social determinants. Screening finds latent disease; high risk without symptoms is selective, not secondary.

WHAT EARNS THE MARKS

- **Naming which question each framework answers**: when, who, and how
- **Classifying a high-risk clinic correctly**: indicated, because attenuated symptoms are already present
- **Making tertiary prevention active**: maintenance treatment, psychoeducation, supported employment
- **Adding the determinants layer**: policy, legislation, zoning and social norms, not only clinic work

SEE ALSO 06 Clinical high risk and early intervention 12 The epidemiology numbers 17 Psychiatric emergencies

Spotters

A spotter is scored on the first sentence. Identify the object, then say what it is for, then give the one parameter or precaution that shows you have handled it. Guessing a number here is worse than naming the object and saying which figure you would confirm from the protocol.

20 · Drug spotters: the ten on the tray

SPOTTERS · TOPIC 20

What is this drug, and what would you check before you gave it?

SPOTTER TABLE: THE DRUG TRAY

THE SHAPE OF THE ANSWER

1 **Inspect out loud**	Generic name, strength, dosage form, batch and expiry
2 **Read the schedule**	Schedule H, H1 or X under the Drugs and Cosmetics Rules
3 **Indication in one line**	What it is for, not a paragraph of pharmacology
4 **The one precaution**	The parameter or route detail that proves you have given it
5 **Declare the gaps**	Name the figure you would confirm from the protocol

Pick the strip up and inspect it out loud. Announce the generic name, the trade formulation, the active strength per unit, and the dosage form: uncoated tablet, film-coated tablet, mouth-

dissolving tablet, sustained-release matrix, oily depot ampoule or aqueous injection. Then give the batch, manufacturing and expiry details.

- **Then read the label.** Check the statutory schedule under the Drugs and Cosmetics Rules: Schedule H, Schedule H1 or Schedule X. That sequence alone earns the first mark before any pharmacology.

SPOTTER	PRESENTATION	INDICATION IN ONE LINE	THE DISCRIMINATOR THE EXAMINER LISTENS FOR
Lithium carbonate	Round white uncoated or film-coated tablet in a strip, or a sustained-release matrix tablet	Acute mania, maintenance prophylaxis in bipolar disorder, augmentation in treatment-resistant unipolar depression, and established anti-suicidal effect	**Narrow therapeutic index, so therapeutic drug monitoring is mandatory on a 12-hour post-dose trough. The British Association for Psychopharmacology target for acute mania is 0.8 to 1.0 mmol/L**
Clozapine	Uncoated, scored, pale-yellow tablet in blister strips	Treatment-resistant schizophrenia, and schizophrenia with persistent recurrent suicidal behaviour	**Neutropenia in approximately 3% and agranulocytosis in 0.4%; an interruption beyond 48 hours requires full retitration; smoking cessation reduces clearance enough to need reductions of up to 70%**
Olanzapine	Film-coated tablet, orally disintegrating tablet, or a vial for intramuscular use	Acute and maintenance treatment in schizophrenia, acute manic or mixed episodes in bipolar I disorder, rapid tranquillisation of agitation	**The dominant burden is metabolic: baseline and periodic weight, body mass index, waist circumference, fasting glucose and fasting lipid profile. Sedation, anticholinergic effects and orthostatic hypotension are common**
Haloperidol decanoate and fluphenazine decanoate	Small glass ampoule of pale-yellow viscous oily solution in a sesame or vegetable oil vehicle	Maintenance in schizophrenia with frequent relapse from poor adherence	**Deep intramuscular gluteal injection by Z-track technique only; intravenous administration is prohibited because of the risk of pulmonary oil microembolism. A short-acting test dose precedes the first depot dose to detect acute severe dystonia or hypersensitivity**

SPOTTER	PRESENTATION	INDICATION IN ONE LINE	THE DISCRIMINATOR THE EXAMINER LISTENS FOR
Disulfiram	Uncoated or dispersible round white tablet in a foil strip	Aversive deterrent in alcohol dependence, for highly motivated individuals or under strict family or clinical supervision	**Irreversible inhibition of hepatic aldehyde dehydrogenase, so ethanol produces acetaldehyde accumulation and the reaction: flushing, throbbing headache, sweating, nausea, persistent vomiting, dyspnoea, tachycardia, hypotension, chest pain and possible cardiovascular collapse. Contraindicated in severe coronary disease, heart failure, idiopathic seizure disorder, cirrhosis, active psychosis, cognitive impairment and pregnancy**
Naltrexone	Film-coated oral tablet in a blister pack	Alcohol use disorder, where it blocks opioid-mediated dopamine release in the nucleus accumbens and reduces craving, heavy drinking and alcohol-induced euphoria; and relapse prevention in opioid use disorder	**A pure competitive antagonist at mu, kappa and delta receptors, so it precipitates immediate severe withdrawal if any opioid remains. An opioid-free interval must be verified by negative urine toxicology or a negative naloxone challenge, and baseline and periodic liver function tests are required**
Buprenorphine with naloxone	Orange, round, biconvex sublingual tablet; the 2 mg with 0.5 mg strength is 6 mm and debossed "A" on one side and "14" on the reverse, and the 8 mg with 2 mg strength is 11 mm and debossed "AN 415" on one side and plain on the reverse	Maintenance treatment of opioid dependence within a complete plan of counselling and psychosocial support	**The two moieties are combined in a fixed 4:1 ratio of free bases. Sublingual naloxone is clinically inactive through near-complete first-pass metabolism; its only purpose is to precipitate severe withdrawal if the tablet is crushed and injected**

SPOTTER	PRESENTATION	INDICATION IN ONE LINE	THE DISCRIMINATOR THE EXAMINER LISTENS FOR
Methylphenidate	Plain immediate-release tablet, or an osmotic-release extended tablet, in a blister pack	Attention-deficit hyperactivity disorder in children and adults, and narcolepsy	**Schedule X under the Drugs and Cosmetics Rules and the narcotic and psychotropic substances legislation: prescription in duplicate with one copy retained by the chemist, a dedicated licence, stock under lock and key, and inspection logs. Monitor resting blood pressure, pulse, height and weight on growth percentile charts, and screen for tics, insomnia, appetite suppression and diversion**
Thiamine hydrochloride	Glass ampoule of clear aqueous solution for intramuscular or intravenous use, or an oral tablet	Prophylaxis and treatment of Wernicke encephalopathy, Korsakoff syndrome, alcohol withdrawal and nutritional beriberi	**Parenteral thiamine precedes or accompanies any intravenous glucose. Thiamine pyrophosphate is the cofactor for pyruvate dehydrogenase and transketolase, so dextrose in a deficient person exhausts residual stores during glycolysis and the citric acid cycle, driving intracellular acidosis and irreversible neuronal necrosis in the mammillary bodies, thalamus and periaqueductal grey**

The tray, with the one precaution or parameter that separates a candidate who has given the drug from one who has read about it.

The buprenorphine and naloxone detail, because this spotter carries the most askable pharmacology. Take administration, then induction, then the maintenance range.

- **How it is taken.** The tablet is placed under the tongue until fully dissolved and is taken whole. Cutting, chewing or swallowing is not permitted, because swallowing sharply reduces bioavailability.
- **During dissolution.** The patient does not eat or drink until it dissolves, and if more than two tablets are needed they are placed all at once or two at a time.
- **Afterwards, and the dental reason.** The patient takes a sip of water, swishes gently around the teeth and gums, swallows, and waits at least 1 hour before brushing, to reduce the risk of dental caries, decay, fracture and tooth loss.

- **Induction.** Buprenorphine monotherapy is used when objective and clear withdrawal signs are evident, transitioning afterwards to the combination tablet. Doses move in increments of 2 mg with 0.5 mg or 4 mg with 1 mg.
- **Maintenance.** The range runs 4 mg with 1 mg to 24 mg with 6 mg daily, with a recommended target of 16 mg with 4 mg as a single daily dose. Doses above 24 mg with 6 mg confer no demonstrated advantage.

HEPATIC STATUS	BUPRENORPHINE CHANGE	NALOXONE CHANGE	CLINICAL DIRECTIVE
Moderate impairment	Peak concentration up 8%, exposure up 64%, half-life up 35%	Peak concentration up 170%, exposure up 218%, half-life up 165%	**Not recommended for induction; caution in maintenance**
Severe impairment	Peak concentration up 72%, exposure up 181%, half-life up 57%	Peak concentration up 1030%, exposure up 1302%, half-life up 122%	**Avoid the combination entirely: naloxone escapes first-pass clearance even sublingually and can precipitate withdrawal or antagonise buprenorphine**

Naloxone clearance depends on hepatic extraction, which is why the combination is a hepatic question and not simply an abuse-deterrence question.

Pharmacokinetics, if the examiner keeps going. Buprenorphine has a median time to peak of 1.50 hours, plasma protein binding of approximately 96% to alpha and beta globulin, and an elimination half-life of 24 to 42 hours.

It undergoes CYP3A4-mediated dealkylation to active norbuprenorphine and glucuronidation, with recovery in urine at 30% and faeces at 69% over 11 days. Naloxone has a median time to peak of 0.75 hours, protein binding of approximately 45% to albumin, and a half-life of 2 to 12 hours.

Safety, in four blocks. In a 4-week trial at 16 mg daily, the common adverse events were headache at 36.4%, withdrawal syndrome at 25.2%, pain at 22.4%, nausea at 15%, insomnia at 14%, sweating at 14%, constipation at 12.1% and abdominal pain at 11.2%.

Buprenorphine products can prolong the QTc interval by 15 msec or less, can cause adrenal insufficiency after more than 1 month of use, and can cause neonatal opioid withdrawal syndrome in pregnancy. In lactation an exclusively breastfed infant receives less than 1% of the maternal daily dose.

Overdose gives pinpoint pupils, sedation, hypotension, hypoglycaemia and respiratory depression, and deaths have occurred in opioid-naive individuals at a 2 mg sublingual dose.

Management is assisted ventilation and a patent airway. Reversal needs higher than usual naloxone doses repeated, because of high mu-receptor affinity and long duration of action. Co-prescription of take-home emergency naloxone at initiation and renewal should be strongly considered.

Concomitant benzodiazepines, sedatives or alcohol carry warnings for profound sedation, respiratory depression, coma and death. CYP3A4 inhibitors raise concentrations and inducers lower them, and the combination is not recommended with monoamine oxidase inhibitors or within 14 days of stopping one.

ESSENTIAL MEDICINES STATUS, AS CANDIDATE RECORDS CARRY IT

Across the 2011, 2015 and 2022 editions of the National List of Essential Medicines, lithium and haloperidol are listed in all three; clozapine and fluphenazine appear in the 2015 and 2022 editions; olanzapine was in the 2011 edition and not in the later two; and buprenorphine and the buprenorphine with naloxone combination appear in the 2022 edition.

These entries come from candidate records rather than a primary notification, so present them as such. The listing status of disulfiram, naltrexone, methylphenidate and thiamine is not carried in the library sources at all, so do not assert it.

VERIFY BEFORE THE EXAM Three items examiners ask reliably at this table have no answer in the library sources, and none is supplied from memory here. The clozapine haematological monitoring cadence, that is the weekly, fortnightly and monthly intervals, together with the neutrophil and leucocyte cut-offs that require interruption or permanent discontinuation. The lithium maintenance target and the concentration at which toxicity begins; the acute mania target above is sourced, the others are not. And whether the Indian public opioid substitution programme predominantly dispenses buprenorphine alone or the combination tablet, which means the dosing and kinetic detail above should be attributed to the package insert rather than presented as Indian programme practice. Close all three from an authoritative protocol and an Indian programme document before the practical.

TRAP Four bedside errors. Advising the patient to swallow the sublingual tablet with water, which destroys efficacy through first-pass metabolism. Saying sublingual naloxone reduces craving, when it is inactive by that route. Advising immediate tooth brushing after dissolution instead of swishing water, swallowing, and waiting. And offering to give an oily depot intravenously in an emergency.

HOLD THIS

Name, strength, form, schedule, then the one precaution. Thiamine before glucose; Z-track for the depot; naloxone in the sublingual tablet is there for the syringe, not for the tongue.

WHAT EARNS THE MARKS

- **Opening with the inspection sequence**: name, strength, form, schedule, before any pharmacology
- **Giving the route rule for a depot**: deep intramuscular by Z-track, and not intravenous
- **Explaining why thiamine precedes glucose**: the cofactor is exhausted during glycolysis
- **Naming the purpose of sublingual naloxone**: deterrence if injected, inactive on the tongue

SEE ALSO 13 The underused treatments 18 Neurobiology at the table 22 Neuromodulation and instruments

21 · The electroconvulsive therapy machine and the mouth gag

SPOTTERS · TOPIC 21

Identify this machine. What waveform does it deliver, and what would you check before treatment?

SPOTTER TABLE: INSTRUMENTS, WITH THE CONSENT AND STATUTE FOLLOW-UP

THE SHAPE OF THE ANSWER

1 **Name the console**	Stimulus generator, panel controls, monitoring ports
2 **Give the waveform**	Brief pulse square wave against the old sine wave
3 **Two impedance phases**	Static before, dynamic during, and dynamic runs lower
4 **Check the machine too**	Self-test and the safety monitor alarm on a load resistor
5 **Then the mouth gag**	Material, timing, position on the molars, what it protects

Name the console, the waveform and the impedance phase, in that order. An electroconvulsive therapy machine is a stimulus generator that passes controlled current across the cranium to induce a therapeutic generalised seizure.

- **What a modern console carries.** A power switch, a stimulus delivery button, parameter selector controls, digital display screens and monitoring cable ports for electroencephalogram, electrocardiogram and electromyogram.
- **The widely used integrated instrument.** Its front panel carries a power switch, an impedance test button, a printer button, a treatment button, two alphanumeric displays and five indicator lights, with a 2-pin stimulus jack and a 9-pin monitoring jack below the impedance test button.

The waveform answer. Sine wave devices deliver continuous bidirectional sinusoidal current through the whole electrical cycle, so energy is delivered during the relative and absolute refractory periods of neuronal membranes. That produces greater tissue heating and more marked retrograde and anterograde amnesia.

Modern brief pulse machines produce bidirectional rectangular square pulses, specified on the reference instrument as bipolar brief pulse square wave with a selectable pulse width of 0.25 to 1.5 ms in 0.25 ms increments.

- **Ultra-brief stimuli.** The same device is supplied with either a 0.25 ms or a 0.30 ms pulse width, one or the other and not both.
- **Why the square pulse wins.** Rectangular pulses match the neuronal chronaxie more efficiently, initiating axonal depolarisation while substantially reducing cognitive side effects.
- **The remaining parameters.** Frequency runs 10 to 70 Hz in 10 Hz increments, rising to 140 Hz for the 0.25 ms pulse, with stimulus duration of 0.14 to 8.0 seconds in increments of equal charge and a constant current of 0.9 A isolated from line current.

Historical ECT apparatus

Companion to original page 747

Real photograph of historical ECT apparatus displayed at Glenside Hospital Museum. This is an equipment-history spotter, not a modern operating console or an electrode-placement demonstration. Its controls must not be used as guidance for current treatment or stimulus dosing.

Identify the subject and explain its historical significance.

Rodw. ECT machine 01.JPG. CC BY-SA 4.0. https://commons.wikimedia.org/wiki/File:ECT_machine_01.JPG Licence: https://creativecommons.org/licenses/by-sa/4.0. Unmodified.

CHECK	HOW IT IS MEASURED	ACCEPTABLE	WHAT AN ABNORMAL READING MEANS
Static baseline impedance	Across the stimulus electrodes before the shock, with a non-stimulating test current at 800 Hz, reading 0 to 3000 ohms within plus or minus 100 ohms	Above 100 ohms and below 3000 ohms before the treatment dose	**A reading of 3000 ohms or more, shown as a flashing value, means poor electrode contact, inadequate skin preparation, dried or insufficient gel, interposed hair, or a dislodged or broken cable, and risks superficial scalp burns. A reading below 100 ohms suggests a short circuit, usually in the stimulus cable**
Dynamic impedance	During actual stimulus passage, spanning 0 to 500 ohms on the reference device and printed on the treatment report	Lower than static impedance; a worked manufacturer example records 1440 ohms falling to 260 ohms during delivery	**A significant deviation indicates a faulty lead or sudden movement**
Power-on self-test	Automatic circuit self-test at power-up, printing a confirmation report before baseline electroencephalographic collection	A printed self-test confirmation	**The machine is checked, not only the patient, and saying so is what separates a candidate who has run a list from one who has watched one**
Safety monitor alarm test	Stimulus cable banana plugs connected to a 200 ohm, 10 watt load resistor, or into the designated jacks of the testing device, with the treatment button held through a full stimulus cycle	The safety monitor light and a continuous alarm tone must appear	**If they do not, the unit is withdrawn from patient use until cleared by biomedical personnel. The manufacturer schedules this annually or as hospital regulations require; whether a per-session dummy-load check is mandated in Indian practice is not carried in the sources**

Two impedance phases and two machine checks. Static is before, dynamic is during, and dynamic runs lower.

Dosing interface and monitoring. On the reference instrument the energy dial is calibrated to total charge in millicoulombs with parametric control, with a standard maximum output of 504 mC across 220 ohms, that is 99.4 to 99.8 J, and 1008 mC, about 199 J, where a double-dose option is available.

Monitoring runs 4 recording channels, two for electroencephalogram, one for electromyogram and one for electrocardiogram, with 8 selectable gain positions per channel.

- **What the end-of-treatment report prints.** Electroencephalographic and electromyographic seizure duration, a postictal suppression index expressed as a percentage, an average seizure energy index, maximum sustained power and coherence with times to peak, spectral analysis, and peak heart rate.
- **The duration figures the manual gives.** A typical minimum motor seizure of 20 seconds, with restimulation at an appropriately higher dose to be considered below that. Pharmacological termination with a short-acting intravenous agent is to be considered beyond about 100 seconds, and the product literature discusses seizures beyond 120 to 180 seconds on the electroencephalogram as prolonged.

VERIFY BEFORE THE EXAM Three things about this spotter are not in the library sources and should not be improvised. The section numbers of the Act that bar unmodified treatment and that regulate the procedure in minors: state the requirement, general anaesthesia and muscle relaxation, and prior approval of the review board with guardian consent for a minor, and cite a section only after reading the Act. The minimum adequate electroencephalographic seizure duration, as distinct from the 20 second motor figure above. And Indian commercial device brand names and their charge maxima, since the one retrieved Indian source was paywalled; name only the machine you have actually handled in your own unit.

The mouth gag, in five lines. Material, timing, position, purpose, removal.

- **Material.** A firm, flexible, non-conductive device of medical-grade silicone, vulcanised rubber or ethylene vinyl acetate, curved to conform to the dental arch, often with a central opening or lateral air channels that allow pharyngeal suctioning and maintain airway patency.
- **Timing.** Placed immediately after anaesthetic induction and full muscle relaxation, verified by loss of fasciculations after succinylcholine, and just before stimulus electrode placement.
- **Position.** Between the posterior premolars and molars bilaterally.
- **What it protects.** It prevents tongue laceration and severe lip biting, protects against tooth fracture or dislodgement from masseter and temporalis spasm driven by direct stimulation of trigeminal motor branches, prevents temporomandibular joint dislocation, and protects the airway by preventing posterior prolapse of the tongue.
- **Removal.** Retained until convulsions cease and spontaneous respiratory effort resumes, then replaced by an oropharyngeal airway if needed.

TRAP Placing the bite block between the incisors. Incisors are single-rooted and mechanically fragile, so direct bite forces subluxate or fracture them, creating dental trauma and an aspiration hazard. The block belongs on the molars, and saying so unprompted is the mark the examiner is waiting for.

HOLD THIS

Bipolar brief pulse square wave, 0.25 to 1.5 ms, constant current 0.9 A; static impedance above 100 and below 3000 ohms. Block on the molars, not the incisors.

WHAT EARNS THE MARKS

- **Giving the waveform with its parameter range**: brief pulse square wave, 0.25 to 1.5 ms
- **Separating the two impedance phases**: static before the shock, dynamic during, dynamic lower
- **Checking the machine as well as the patient**: self-test report and the alarm test on a load resistor
- **Placing the bite block on the molars**: said before the examiner asks

SEE ALSO 13 The underused treatments 22 Neuromodulation and instruments 17 Psychiatric emergencies

22 · Neuromodulation, recording and testing instruments

SPOTTERS · TOPIC 22

Identify this coil. How deep does it reach, and what would you treat with it?

SPOTTER TABLE: INSTRUMENTS, AND THE NEUROMODULATION FOLLOW-UP

THE SHAPE OF THE ANSWER

1	Physics before geometry	Induction, field strength, and decay with distance
2	Depth against focality	Figure-of-eight focal, helmet deep and diffuse
3	Direct current is different	Continuous low current, anodal up, cathodal down
4	The recording tray	Electrodes, landmarks, the placement system, indications
5	The testing devices	Biofeedback, breathalyser type, and why a chart is here

For a coil, give the physics before the geometry. Repetitive transcranial magnetic stimulation works by electromagnetic induction: the coil is driven by pulsed currents of several hundred amperes for approximately 50 to 150 microseconds, producing transient magnetic fields of approximately 1 tesla, which induce electric fields in the brain.

- **The one fact that generates the trade-off.** The magnetic field crosses scalp and skull with far less attenuation than an applied current would suffer, but the induced field still falls with distance from the coil, so superficial cortex is stimulated most strongly.

COIL	GEOMETRY	FIELD AND DEPTH	CLINICAL USE
Figure-of-eight, and its butterfly variant	Two adjacent round loops side by side with opposite current direction; the butterfly variant is bent at the centre to conform to the head and gain depth. A representative clinical casing is 3.5 by 2 inches	**Highly focal: currents summate constructively at the intersection, roughly doubling stimulating ability at the centre, with motor cortex stimulation achieved within a 5 mm resolution and suprathreshold field to depths of up to 1.5 cm**	Focal cortical targeting: left dorsolateral prefrontal cortex in depression, supplementary motor area in obsessive-compulsive disorder

COIL	GEOMETRY	FIELD AND DEPTH	CLINICAL USE
Circular	A single continuous round loop of copper winding; a representative round coil is 3.5 inches in diameter	**Non-focal, so a targeted area is difficult to stimulate locally. The depth in centimetres is not carried in the sources, described only as too shallow for mood-regulating regions**	Motor threshold determination, functional motor mapping, and the lower-limb motor cortex on the medial hemispheric walls
Deep coils, the helmet designs	Complex multi-loop windings inside a rigid helmet, in several geometries based on figure-eight coils	**A broad non-homogeneous field designed to minimise surface charge accumulation and slow field decay. At an output of 120% of hand motor threshold, one design induces suprathreshold field at lateral and medial frontal regions to depths of up to 4 to 5 cm, and another to 2 to 3 cm medial prefrontally and up to 5 to 6 cm at lateral frontal regions**	Treatment-resistant depression, obsessive-compulsive disorder and smoking cessation, with different helmet variants for each

Benchmarking across 50 coil designs found figure-eight coils most suitable for focused stimulation and helmet coils for wide stimulation, with the butterfly intermediate on both, which is the systematic trade-off stated as a finding rather than an opinion.

VERIFY BEFORE THE EXAM The frequency cut-offs that divide high-frequency from low-frequency stimulation, and the resting motor threshold definition, that is the minimum motor evoked potential amplitude and the proportion of consecutive trials required, are not carried in the library sources. What is sourced is the direction, high-frequency stimulation raises cortical excitability and is applied over the left dorsolateral prefrontal cortex in depression while low-frequency stimulation suppresses it and is applied over the right dorsolateral prefrontal cortex or the supplementary motor area, that motor thresholds are read from abductor pollicis brevis, and that treatment intensity is expressed as a percentage of that threshold. Take the cut-offs and the threshold criteria from a stimulation protocol before the viva.

The direct current stimulator. It is a compact battery-powered constant-current unit delivering uniform low-intensity direct current, typically 1 mA or 2 mA with experimental escalation up to 4 mA, through cutaneous electrodes.

- **Anodal stimulation.** Subthreshold depolarisation of resting membrane potentials, raising cortical excitability by opening sodium-dependent channels, augmenting intracellular calcium and fostering long-term potentiation.

- **Cathodal stimulation.** Hyperpolarises membranes, reducing spontaneous firing and dampening excitability.
- **Where it stands.** It has been evaluated in over 20 neurological and psychiatric disorders including major depressive disorder, post-stroke motor recovery, aphasia and anxiety, with no approved indication to date.

MONTAGE	ELECTRODES AND PLACEMENT	PEAK TARGET FIELD AT 2 MA	VOLUME STIMULATED
Conventional motor cortex to supraorbital	7 by 5 cm pads, anode at C3 and cathode at the right supraorbital site, mean separation 6.51 cm	0.273 V/m	**1.51 times 10 to the fifth cubic millimetres: low focality with high off-target spread**
Conventional bilateral motor cortex	7 by 5 cm pads, anode at C3 and cathode at C4, mean separation 5.39 cm	0.271 V/m	**1.70 times 10 to the fifth cubic millimetres: diffuse cortical stimulation**
High-definition four-by-one	0.5 cm diameter circular electrodes, central anode at C3 with four cathodes at FC3, C1, CP3 and C5, mean separation 2.93 cm	0.306 V/m	**1.07 times 10 to the fourth cubic millimetres: the highest focality of the conventional set**
Anterior-posterior pad surround	7 by 5 cm pads, anode at CP3 and cathode at FC3, mean separation 2.12 cm	0.363 V/m, which is 33.9% higher than conventional	**2.75 times 10 to the fourth cubic millimetres**
Optimised anterior-posterior pad surround	1 by 1 cm pads, anode at CP3 and cathode at FC3 adjusted closer, mean separation 4.12 cm	0.553 V/m, which delivers 204.1% of the conventional on-target field	**2.14 times 10 to the fourth cubic millimetres, stimulating merely 12.6% of the off-target volume of a conventional pad montage**

The optimised montage produces a field equivalent to 4.08 mA using only 2 mA at the scalp, and a conventional 2 mA equivalent can be achieved with 0.98 mA.

The electroencephalography tray. It shows fabric caps with pre-installed colour-coded electrode holders, individual silver and silver chloride cup or disc electrodes with flexible insulated leads, skin preparation abrasive paste, conductive chloride-based gel and blunt-tipped filling syringes.

- **The three landmarks.** The nasion, the depression just above the bridge of the nose. The inion, the bulge at the back of the skull. The preauricular points or tragi.
- **How the vertex is found.** Measure nasion to inion and mark the halfway point, measure tragus to tragus, and take the intersection as the vertex. The remaining positions are mapped from those measurements.
- **How positions are named.** Letters denote the underlying territory: frontopolar, frontal, central, temporal, parietal, occipital and auricular reference. Odd numbers are left-sided and even numbers right-sided, and the midline carries the letter z. The extended system adds intermediate positions for neuromodulation targeting.

- **The psychiatric indications.** Differentiating delirium from functional disorder, investigating episodic behavioural dyscontrol or suspected temporal lobe seizures, assessing metabolic or hepatic encephalopathy through triphasic waves, and identifying periodic sharp wave complexes in rapidly progressive dementia.
- **Verify before the exam.** The spacing percentages that give the system its name, and the inter-electrode impedance threshold below which a diagnostic recording is acceptable, are not carried in the library sources. Confirm both from an electroencephalography text, noting that high impedance introduces powerline noise, movement artefact and baseline drift.

INSTRUMENT	WHAT IT MEASURES	THE DISCRIMINATOR
Biofeedback unit	Converts covert physiological process into real-time visual display or acoustic tone, through transducers and cutaneous sensors, a high-gain differential preamplifier and filter, a microprocessor, and a feedback interface	**Electromyographic feedback from frontalis or trapezius for tension headache and chronic pain; electrodermal activity for panic and phobic anxiety; peripheral thermal feedback from distal phalanges for migraine and Raynaud phenomenon; heart rate variability training of respiratory sinus arrhythmia for post-traumatic stress disorder, panic and depression; and neurofeedback frequency band training for attention-deficit hyperactivity disorder and insomnia**
Breathalyser, fuel cell type	Platinum electrodes catalyse oxidation of ethanol to acetic acid, generating a current proportional to ethanol content in deep end-expiratory alveolar breath	**High specificity for ethanol and largely unaffected by acetone, ketones or aromatic hydrocarbons, with high calibration stability; the evidentiary and clinical instrument of choice**
Breathalyser, semiconductor type	Ethanol adsorption changes the electrical resistance of a heated tin dioxide bead	**Low specificity: it cross-reacts with acetone in diabetic ketoacidosis, with cigarette smoke and with breath mints, drifts, and needs frequent calibration. A screening device, not an evidentiary one**
Snellen chart	Rows of optotypes decreasing in size, each row calibrated so the optotype subtends a fixed visual angle at its designated distance	**Test each eye monocularly with an opaque occluder and no pressure on the globe, then binocularly, reading down until fewer than half the letters on a line are identified; acuity is recorded as the ratio of the viewing distance to the distance at which unimpaired vision reads that line**

Why the chart is on a psychiatry table. Baseline visual screening precedes psychotropics with ocular effects.

- **The phenothiazines.** They are associated with anterior lens and corneal granular deposits, and one of them with pigmentary retinopathy and visual field constriction above a daily dose threshold.
- **The anticholinergic burden.** Tricyclics and low-potency antipsychotics cause cycloplegia with blurred near vision and can trigger acute angle-closure glaucoma in a shallow anterior chamber.
- **Two more reasons.** Screening covers ethambutol-induced toxic optic neuropathy in a tuberculous meningitis or delirium presentation, and separates visual hallucination from primary sensory impairment in delirium and Lewy body dementia.
- **In functional neurological disorder.** Preserved acuity or intact optokinetic nystagmus against severe subjective visual complaint is the finding that matters.

VERIFY BEFORE THE EXAM The library sources for this volume carry no breath alcohol instrumentation or road traffic statute, and no visual acuity geometry. So the mandatory observation period before breath sampling, which exists to let residual mouth alcohol from regurgitation, vomitus or alcohol-containing mouthwash dissipate, the statutory section and the permitted blood alcohol concentration, the standard testing distance for the chart, the visual angles subtended by the optotype and by each stroke, the notation for normal acuity, and the dose threshold for phenothiazine retinopathy are all unsourced here. Read the limit off the Act and the geometry off a forensic medicine or ophthalmology text before the viva.

TRAP Misidentifying a direct current stimulator as a transcutaneous electrical nerve stimulation or cranial electrotherapy device: direct current stimulation delivers continuous non-oscillating current at 1 mA or 2 mA, while the others deliver pulsed alternating current. And assuming a conventional pad montage peaks beneath the electrodes, when modelling shows the peak field often sits midway between them, which is the whole reason the surround montages exist.

HOLD THIS

Figure-of-eight is focal to about 1.5 cm; the helmet designs reach 4 to 5 cm and lose focality. Fuel cell for evidence, semiconductor for screening.

WHAT EARNS THE MARKS

- **Explaining depth against focality from the physics**: the induced field falls with distance from the coil
- **Naming the current type**: continuous direct current, not pulsed alternating current
- **Finding the vertex by measurement**: nasion to inion and tragus to tragus, then the intersection
- **Choosing the breathalyser by purpose**: fuel cell for evidence, semiconductor for screening only

SEE ALSO 21 The electroconvulsive therapy machine 09 Inflammation and precision psychiatry 23 Scale and test material spotters

23 · Scale and test material spotters

SPOTTERS · TOPIC 23

What is this form or test material, and what does it measure?

SPOTTER TABLE: SCALES AND PSYCHOLOGICAL TEST MATERIAL

THE SHAPE OF THE ANSWER

1 **Read the layout first**	Title, item groupings, scoring grid, publisher format
2 **Then the four facts**	Construct, rater, informant, target population
3 **Name the near neighbour**	And the feature that separates the two
4 **Describe administration**	Phases, timing, and what is recorded
5 **Declare the band**	Structure now, cut-off confirmed from the manual

Identify the form by its layout before you name it. Read the document title, the publisher layout, the item groupings and the scoring grid.

- **Then give four facts.** What construct it measures, who rates it, who provides the information, and the target population.
- **The definition, if asked.** A rating scale is a standardised metric of symptom severity, treatment response and disease trajectory in clinical and research settings. The examiner wants the structure before the interpretation.

FORM	CONSTRUCT AND RATER	STRUCTURE THAT IS SOURCED	THE NEAR NEIGHBOUR IT IS CONFUSED WITH
Hamilton Rating Scale for Depression	Depressive symptom severity, clinician-rated interview	**The standard classical version contains 17 items, probing depressed mood, guilt, suicide, initial, middle and delayed insomnia, work and activities, psychomotor retardation, agitation, psychic and somatic anxiety, gastrointestinal and general somatic symptoms, genital symptoms, hypochondriasis, weight loss and insight**	The anxiety scale of the same family, which is focused on psychic and somatic anxiety; the self-administered depression inventory, which emphasises subjective cognitive distortion; and the shorter self-report questionnaires, one of which omits the suicide item
Positive and Negative Syndrome Scale	Schizophrenia and psychotic syndromes, clinician-rated interview and observation	**A multi-page booklet bearing three subscales, positive, negative and general psychopathology, encompassing 30 symptom constructs, each rated on a 7-point Likert severity scale**	The shorter 18-item psychiatric rating scale for rapid multidimensional assessment, and the standalone scales for the assessment of positive and of negative symptoms
Mini-Mental State Examination	Global cognitive screening, clinician-administered task battery	**A single sheet with clear task subdivisions, orientation, registration, attention and calculation, recall, and language and praxis, with a maximum score of 30 points**	The Montreal cognitive assessment, which adds clock drawing, trail-making and abstraction; and the Addenbrooke examination

FORM	CONSTRUCT AND RATER	STRUCTURE THAT IS SOURCED	THE NEAR NEIGHBOUR IT IS CONFUSED WITH
Abnormal Involuntary Movement Scale	Abnormal involuntary movements, predominantly tardive dyskinesia from long-term neuroleptic exposure, clinician observation and physical examination	**A movement examination sheet arranged anatomically by body region, moving through orofacial movements, then extremities, then trunk, then global judgements of severity, incapacitation and patient awareness, then dental status and denture fit**	The akathisia rating scale, which assesses subjective inner restlessness and objective motor agitation, and the extrapyramidal symptom rating scale
Yale-Brown Obsessive Compulsive Scale	Obsessive-compulsive symptom severity, clinician-rated semi-structured interview	**A comprehensive symptom checklist probing past and current aggressive, contamination, sexual, hoarding, religious, symmetry and somatic obsessions alongside cleaning, checking, repeating, counting, ordering and hoarding compulsions, followed by a core severity scale that rates time spent, interference with functioning, distress, resistance and degree of control, separately for obsessions and for compulsions**	Any generic anxiety severity measure; the symptom checklist plus the paired obsession and compulsion structure is what identifies this form

The five forms, each described by what is actually printed on the page.

VERIFY BEFORE THE EXAM The numeric cut-offs for these scales are not carried in the library sources and are not printed here: the depression scale's remission and severe-depression thresholds and its per-item anchor range, the syndrome scale's total score range, the cognitive examination's impairment cut-off and its mild and moderate bands, the involuntary movement scale's item count and per-item anchor range, and the obsessive-compulsive scale's item count, maximum total and severity bands. Examiners ask for every one of them, so close this set from the published scale manuals before the practical, and at the table describe the structure and say you will confirm the band.

One longitudinal figure worth carrying. Prospective follow-up in obsessive-compulsive disorder documents that 10 to 15% of patients experience unremitting deterioration, while full

remission occurs in 10 to 15% up to 22% of patients, partial remission in 53%, and 48% relapse after remission.

- **Why it is worth holding.** It is the kind of course statistic that turns a scale question into a prognosis question.

TEST MATERIAL	WHAT IS ON THE TABLE	WHAT IT MEASURES	THE DISCRIMINATOR
Bender Visual Motor Gestalt cards	A boxed set of 9 separate white cards, each 3 by 5 inches, each showing one distinct black geometric line drawing; archival reference sheets measure 22.5 by 29 cm	Visual-motor maturity and neurological impairment, administered individually from 3 years and older, untimed and typically taking 10 to 20 minutes	**Developed in 1938 by Lauretta Bender on Gestalt principles and popularised by Max Hutt in 1947. Score for accuracy and spatial organisation, looking for perseveration, repeating an element beyond normal limits as in continuous rows of dots, common in frontal lesions; rotation of 45 to 180 degrees from the presented axis; gross distortion of angles, failure of closure or destruction of the Gestalt; and fragmentation with loss of symmetry**
Seguin form board	A polished wooden board with recessed geometric cut-outs and matching wooden blocks, laid flat before the child with the blocks in a standardised arrangement above it	Speed of processing, visual-spatial perception, eye-hand coordination and non-verbal intelligence, for young children and for individuals with speech or hearing deficit, communication disorder or suspected intellectual developmental disorder	**A direct performance test administered to the child across consecutive timed trials, each timed from the first touch to the last piece seated, with the best time referenced against Indian normative tables to derive a mental age. The number of recesses, the number of trials and the performance quotient formula are not carried in the sources, so describe the procedure and confirm the counts**

TEST MATERIAL	WHAT IS ON THE TABLE	WHAT IT MEASURES	THE DISCRIMINATOR
Rorschach inkblot plates	A boxed set of 10 heavy cardboard plates carrying bilaterally symmetrical unstructured inkblots on an off-white ground	Projective assessment of personality, reality testing and defensive style	**Two phases. Free association: plates are handed over one at a time in fixed orientation with the question "what might this be", recording exact verbal responses, response latency, time per card and card rotations. Inquiry: after all plates, every response is reviewed to establish where the percept was seen and what qualities of the blot produced it. Scoring covers location, determinants, form quality and content. The monochrome, bicoloured and polychrome plate distribution is not carried in the sources**
Thematic Apperception Test cards	Monochrome picture cards depicting ambiguous interpersonal scenes, figures of varying ages and classical evocative engravings, alongside a blank card, with identification numbers and sex and age coding letters on the reverse	Projective assessment of interpersonal dynamics, motives and conflicts	**The instruction is a dramatic narrative: what led up to the scene, what is happening now, what the characters think and feel, and how it ends. Interpretation follows the system of needs and press: the hero with whom the subject identifies, the hero's internal needs, the environmental press acting on the hero, the thema, and the resolution. The exact card count is inconsistently sourced in the library, so describe the set rather than asserting a number**

TEST MATERIAL	WHAT IS ON THE TABLE	WHAT IT MEASURES	THE DISCRIMINATOR
Vineland Social Maturity Scale	An interview protocol or record booklet detailing behavioural and developmental milestones arranged hierarchically across sequential age bands	Adaptive behaviour, social competence and personal independence, providing objective criteria for intellectual developmental disorder, autism spectrum disorder and developmental delay	**An indirect semi-structured interview with a primary caregiver who knows the individual well, augmented by direct observation, across the domains of self-help general, self-help eating, self-help dressing, self-direction, occupation, communication, locomotion and socialisation. Total points convert to a social age, from which a social quotient is derived. The item count, the age span, the part-credit scoring rule and the quotient formula are not carried in the sources**

Performance and projective materials. The examiner separates the direct performance test from the informant interview, and the abstract blot from the figurative scene.

THE INDIAN ADAPTATIONS, AND THE ONE THAT IS MISSING

Two adaptations are named in the library. The Indian adaptation of the apperception test by Uma Chowdhary, which replaces Western domestic scenery, hairstyles and attire with Indian interpersonal situations including joint-family dynamics and rural and urban settings. And the Indian adaptation of the social maturity scale by Malin, on which Indian psychiatric and clinical psychology departments standardly rely.

Examiners at this station also ask for the Indian adaptation of the cognitive screening examination. That adaptation and its Indian validation data are not carried in the sources, so it is not named here. Confirm it before the viva.

On certification, Indian disability certification rests on standardised intelligence testing together with documented significant deficit in adaptive functioning, which the maturity scale can document as a supporting measure. The social quotient is not itself the certifying figure, and the certifying cut-off values are not carried in the sources.

TRAP Two identification errors. Mistaking the Gestalt cards for block design or object assembly material: the Gestalt set is 9 cards of single black line figures, not coloured designs or puzzles. And mistaking a Rorschach plate for an apperception card: the plates are abstract and bilaterally symmetrical with no figurative humans, and the cards are realistic ambiguous human scenes.

The interpretive trap. Treating an abnormal Gestalt reproduction as definitive evidence of localised brain damage, or a low cognitive screening score as diagnostic of dementia. Clinical guidance is that

these tools screen, and belong inside a broader neuropsychological battery and a comprehensive evaluation.

HOLD THIS

Depression scale 17 items clinician-rated; syndrome scale 30 constructs on a 7-point Likert scale; cognitive examination out of 30; Gestalt set 9 cards of 3 by 5 inches. Describe the structure, confirm the band.

WHAT EARNS THE MARKS

- **Describing what is printed on the page**: subscales, item groupings, scoring grid
- **Naming rater and informant separately**: clinician-rated interview against caregiver interview
- **Giving the two Rorschach phases**: free association, then inquiry into location and determinants
- **Refusing to over-read a screen**: these instruments screen inside a fuller battery

SEE ALSO 16 Medical education and pedagogy 24 Image spotters 25 Historical figure spotters

24 · Image spotters

SPOTTERS · TOPIC 24

Describe this image and give me your impression.

SPOTTER TABLE: HISTOPATHOLOGY, IMAGING, TRACES AND CLINICAL PHOTOGRAPHS

THE SHAPE OF THE ANSWER

1	Use one format every time	Modality, plane or stain, then findings, then impression
2	Histology by the inclusion	Protein, appearance, halo or no halo, stain and site
3	Stage it if asked	Topographical stages, then the ascending sequence
4	Read structure and trace	Atrophy pattern, sulcal pattern, waveform and its lag
5	Close on the discriminator	The neighbour it is not, and why

For every image, use one format: modality, plane or stain, primary positive finding, secondary findings, clinical impression. A candidate who describes before diagnosing survives a wrong first guess; a candidate who diagnoses first has nothing left to say.

NEUROPATHOLOGY	PROTEIN AND ULTRASTRUCTURE	LIGHT MICROSCOPY	STAIN AND SITE
Senile and neuritic plaques	Amyloid-beta, extracellular fibrils	Amorphous spherical pink parenchymal deposits	**Silver impregnation and thioflavin S, in neocortex, association areas and the default mode network. Diffuse plaques lack cytoskeletal alteration; neuritic plaques carry an amyloid core encircled by dystrophic phospho-tau-immunoreactive neurites and reactive glial processes, which is the clinically significant subset**
Neurofibrillary tangles	Hyperphosphorylated tau in paired helical filaments	Basophilic or amphophilic inclusion, flame-shaped in neocortical and hippocampal pyramidal neurons and globose in subcortical and pigmented nuclei	**Silver stains and phospho-tau antibodies, in transentorhinal cortex, hippocampus and association neocortex. After the neuron dies, extracellular tombstone or ghost tangles persist**
Brainstem Lewy bodies	Alpha-synuclein, fibrillar and phosphorylated	Round intracytoplasmic eosinophilic inclusion with a dense central core and a distinct pale peripheral halo	**Weakly argyrophilic, so alpha-synuclein immunohistochemistry is the diagnostic standard. Found in pigmented dopaminergic neurons of the substantia nigra pars compacta, noradrenergic neurons of the locus coeruleus, and the vagal dorsal motor nucleus**
Cortical Lewy bodies	Alpha-synuclein, less densely packed	Ill-defined, pale, non-haloed round inclusion	**Immunohistochemistry is required rather than optional, in anterior cingulate gyrus, entorhinal cortex and temporal isocortex. Lewy neurites are thread-like aggregates within axons and dendrites**

NEUROPATHOLOGY	PROTEIN AND ULTRASTRUCTURE	LIGHT MICROSCOPY	STAIN AND SITE
Pick bodies	Three-repeat tau	Round or oval, well-demarcated, moderately basophilic, smooth contour, and no peripheral halo	**Intensely argyrophilic on silver stains and positive for three-repeat tau, which separates them from the paired three-repeat and four-repeat tau of Alzheimer tangles. Found in dentate granule neurons of the hippocampus and the frontotemporal neocortex**

The halo is the single most useful feature on this table: present in brainstem Lewy bodies, absent in cortical Lewy bodies and in Pick bodies.

The staging systems, named and ordered. For Lewy body disease, Kosaka and McKeith categorised 3 topographical stages: brainstem predominant, limbic or transitional, and diffuse neocortical.

Braak refined this into 6 ascending stages. Lesions begin in the dorsal motor nucleus of the vagus and the olfactory bulb, then ascend through the pontine tegmentum into the midbrain and substantia nigra pars compacta.

They then spread rostrally to basal forebrain, amygdala, transentorhinal cortex and the allocortical CA2 plexus, invade temporal and sensory association neocortices, and finally involve the entire neocortex including primary sensory and motor fields.

- **The recognised departure.** The amygdala-predominant variant, where inclusions concentrate in the amygdala with minimal lower brainstem involvement, seen with advanced Alzheimer change and driven by cross-seeding between hyperphosphorylated tau and alpha-synuclein.
- **The Alzheimer consensus score, three components.** Amyloid phase, tracking progression from neocortex down to limbic structures, basal ganglia, brainstem and cerebellum. Tangle stage, tracking phospho-tau spread from transentorhinal cortex to limbic hippocampus to isocortex. Neuritic plaque density, graded semi-quantitatively as none, sparse, moderate or frequent.
- **What the combined score means.** It establishes 3 categorical levels, low, intermediate and high, and only intermediate and high levels are considered sufficient to explain cognitive impairment or dementia.

The molecular images. Four, each with its finding.

- **Amyloid tracers.** Retention in neocortical grey matter, the default mode network and striatum, with loss of normal grey and white differentiation.
- **Tau tracers.** Paired helical filament uptake in transentorhinal, inferior temporal and parieto-occipital cortices, closely matching clinical symptoms.
- **Dopamine transporter imaging.** Reduced striatal uptake, initially asymmetric putaminal loss, reflecting nigrostriatal degeneration.
- **Cardiac sympathetic scintigraphy.** Reduced postganglionic uptake in the Lewy body spectrum.

The biomarker frame. The biological definition of Alzheimer disease runs on a matrix of three axes.

- **Amyloid.** Decreased cerebrospinal fluid amyloid-beta 42 or a reduced ratio to amyloid-beta 40, or a positive amyloid scan.
- **Tau.** Divided into diagnostic markers, raised cerebrospinal fluid or plasma phospho-tau species, and staging markers.
- **Neurodegeneration.** Raised neurofilament light chain, raised total tau, or structural atrophy.
- **The seed amplification assay, mechanically.** Misfolded alpha-synuclein in cerebrospinal fluid, skin biopsy or olfactory swab recruits added recombinant monomer in a reaction plate, accelerating fibril formation detected by fluorescence across cycles of quaking and incubation.

QUESTION	THE FIGURE	THE DISCRIMINATOR
Assay performance in confirmed Lewy body disease	Reported sensitivity begins at 91.6% and specificity at 94.3%	**Quote it as a floor rather than a point estimate, because the reported range runs upward from there**
Sensitivity by disease burden	At least 95.0% in neocortical and limbic disease at the higher Braak stages, falling to 35% to 60.9% when pathology is restricted to the brainstem, and rising within that band from 37% at the earliest stages to 73% at the third	**Assay sensitivity tracks anatomical burden, which is why a negative result early does not exclude disease**
Sensitivity in the amygdala-predominant variant	14.3% to 50.0% in ante-mortem lumbar fluid, and 60.0% to 63.6% in post-mortem ventricular fluid	**The variant most often co-occurring with Alzheimer change is also the one the assay misses**
Co-occurrence of the two pathologies	Biomarker-confirmed co-occurrence affects approximately 20% to 25% of cognitively impaired elderly individuals and approximately 8% of asymptomatic individuals	**Mixed pathology is the expectation, not the exception**
Alpha-synuclein in Alzheimer brains at autopsy	33% to 66% of sporadic cases, with comparable rates in early-onset below 65 years and late-onset above 65 years; 27% to 85% of autosomal dominant cases; and 14% to 50% of Down syndrome-associated cases	**The spread of these ranges is itself the answer to a question about mixed pathology**
Alzheimer change in clinically diagnosed Lewy body dementia	48% to 91% show concurrent change at autopsy, with approximately 50% meeting criteria for intermediate or high levels; in vivo approximately 50% sit within the Alzheimer continuum, 32% amyloid-positive and tau-negative and 15% positive for both, and a total tau to amyloid-beta 42 ratio above 0.52, or above 0.3, denotes concomitant pathology	**Severe concurrent Alzheimer change reduces the diagnostic sensitivity for the classical core features to 46%, obscuring parkinsonism and hallucinations and producing misclassification as pure Alzheimer disease**

Mixed pathology accelerates decline in global cognition, attention and executive function, and memory, and in Alzheimer disease the addition of Lewy pathology increases motor impairment and raises the incidence of delusions, hallucinations and sleep disturbance.

The structural slices, described as the examiner wants them. Four patterns.

- **Medial temporal lobe atrophy.** Scored on coronal T1-weighted slices by inspecting three features: the width of the choroid fissure, the width of the temporal horn of the lateral ventricle, and the height or volume of the hippocampus.

- **Its progression.** From a normal appearance, through isolated widening of the choroid fissure, to temporal horn enlargement with loss of hippocampal height, to a severely enlarged horn with a shrunken hippocampal remnant. Asymmetric or marked bilateral atrophy supports Alzheimer disease.
- **Frontotemporal lobar degeneration.** Circumscribed severe knife-blade gyral atrophy in the frontal and anterior temporal poles with marked widening of the frontal horns. The semantic variant shows asymmetric left-greater-than-right anterior temporal atrophy; the behavioural variant shows bilateral frontal and anterior insular volume loss.
- **Normal pressure hydrocephalus.** Ventriculomegaly, a disproportionately enlarged subarachnoid space with enlarged Sylvian fissures and basal cisterns alongside effacement and crowding of parasagittal sulci at the high vertex, and an acute callosal angle on coronal slices.
- **Wilson disease.** The face of the giant panda on axial T2 at the midbrain: high tegmental signal surrounding normal low-signal red nuclei as the eyes, with preserved substantia nigra as the ears. A miniature panda appears in the pons where tegmental hyperintensity spares the central tegmental tracts.
- **Verify before the exam.** The diagnostic grades, the ventricular index value and the callosal angle threshold are not carried in the library sources.

The traces. Three, each with the feature that names it.

- **Periodic sharp wave complexes in Creutzfeldt-Jakob disease.** Repetitive biphasic or triphasic sharp waves or mixed spike-wave discharges recurring rhythmically, synchronous and generalised or periodically lateralised, superimposed on a disorganised slow background.
- **Its imaging correlate.** Carried in the library's textbook source: high intensity along the cortical ribbon on diffusion-weighted imaging, with hyperintensity of the basal ganglia, particularly caudate and putamen.
- **Triphasic waves of hepatic and metabolic encephalopathy.** High-amplitude waves with three components, a small initial negative deflection, a prominent positive wave and a slow negative wave, recurring rhythmically, bilaterally synchronous and frontally predominant, with an anterior-to-posterior phase lag. They diminish after metabolic correction or a benzodiazepine challenge.
- **Generalised spike-and-wave discharges of absence seizures.** Sudden bilateral synchronous symmetric complexes at 3 Hz on the library's electroencephalography text, with maximum amplitude frontally, on a normal interictal background, with abrupt onset and offset and provocation by hyperventilation.

The clinical photographs. Three, described before they are named.

- **Kayser-Fleischer ring.** A golden-brown, greenish-brown or bronze band of granular pigmentation at the corneal limbus, from copper granules deposited in Descemet membrane of the peripheral endothelium. It develops first at the superior pole, then the inferior, before merging laterally into a complete circle.
- **Its mechanism.** Impaired biliary copper excretion with overflow into tissues including the basal ganglia and cornea.
- **Tardive dyskinesia.** Involuntary, repetitive, purposeless stereotypic or choreiform movement predominantly of tongue, mouth and lower face: tongue protrusion described as a fly-catcher or darting tongue, chewing, lip smacking, puckering, pouting, grimacing and lateral deviation of the mandible.
- **Its two named signs.** A bolus-like protrusion inside the cheek named the bonbon sign, with axial and limb manifestations of choreic toe flicking, pelvic rocking and trunk writhing.
- **Catatonic posturing, the five signs.** Catalepsy, the spontaneous active maintenance of a posture against gravity for a prolonged period. Waxy flexibility, initial resistance to passive movement followed by smooth compliant moulding like bending a wax candle. The psychological pillow, the head held above the bed surface for hours with sustained sternocleidomastoid contraction.
- **And the two resistance signs.** Gegenhalten, automatic resistance that increases in proportion to the examiner's force. Mitgehen, the limb moving to light fingertip pressure despite instruction to resist.

VERIFY BEFORE THE EXAM

A long list of classic spotter numbers is not carried in the library sources for this volume, and none is supplied here from memory: the Lewy body size range in micrometres; the amyloid phase and tangle stage numbering of the consensus score; the atrophy scale grades; the ventricular index threshold and the callosal angle cut-off in normal pressure hydrocephalus; the discharge frequency and interval of periodic sharp wave complexes; the amplitude and frequency of triphasic waves and the phase lag in milliseconds; the serum ceruloplasmin threshold and the urinary copper excretion threshold in Wilson disease, and the gene and chromosome; the involuntary movement scale item count and anchor range; and the catatonia rating scale item count, its screening subset, the lorazepam challenge dose and the time to objective response. Close them from a neuroimaging text, an electroencephalography text and the published rating scales before the practical.

FOUR DISCRIMINATORS IN ONE BLOCK

Lewy against Pick: Lewy bodies have an eosinophilic dense core with a clear halo and are alpha-synuclein positive; Pick bodies are smooth, rounded, non-haloed, strongly argyrophilic and three-repeat tau positive. **Kayser-Fleischer ring against corneal arcus:** arcus is extracellular lipid that leaves a clear zone of cornea between the opacity and the limbus, while the copper ring extends directly to the limbus with no intervening clear zone. **Periodic sharp waves against triphasic waves:** triphasic waves show an anterior-to-posterior phase lag and diminish with metabolic treatment or benzodiazepines, while periodic complexes are stereotypic, sharper, and persist despite metabolic stabilisation. **Hydrocephalus ex vacuo against normal pressure hydrocephalus:** in ex vacuo dilatation the cortical sulci are symmetrically widened over the convexities, while in normal pressure hydrocephalus ventriculomegaly coexists with tight effaced high-convexity sulci and dilated Sylvian fissures.

One more discriminator. Tardive dyskinesia is objective involuntary movement the patient may be unaware of, while akathisia is driven by a distressing subjective inner restlessness that compels voluntary adjustment.

HOLD THIS

Core with a halo is brainstem Lewy; no halo and argyrophilic is Pick. Mixed pathology sits in 20% to 25% of cognitively impaired elderly people, and it drops the sensitivity for the classical Lewy features to 46%.

WHAT EARNS THE MARKS

- **Describing before diagnosing**: modality, plane or stain, findings, then impression
- **Using the halo as the histology discriminator**: present in brainstem Lewy bodies alone
- **Reading the sulci in hydrocephalus**: tight high-convexity sulci against symmetrically widened ones
- **Naming the phase lag on triphasic waves**: anterior to posterior, and reversible with treatment

SEE ALSO 18 Neurobiology at the table 23 Scale and test material spotters 25 Historical figure spotters

25 · Historical figure and classic spotters

SPOTTERS · TOPIC 25

Who is this, and what is he known for?

SPOTTER TABLE: PORTRAITS, MONOGRAPHS AND JOURNALS

THE SHAPE OF THE ANSWER

1 **Face, idea, book**	In that order, and one idea only per figure
2 **Separate the three nosologists**	Course, splitting, form of conscious experience
3 **The prizes**	Three awarded in this territory, and why the others were not
4 **Monograph to concept**	Each classic reduces to one line
5 **The Indian faces**	Name the contribution, and keep the two Ranchi names apart

Identify the face, then the single idea, then the book. Kraepelin, 1856 to 1926, is shown with round wire-rimmed spectacles, a prominent grey moustache and formal academic dress.

- **His single idea.** He established clinical course and longitudinal outcome as the primary criteria of psychiatric nosology, separating dementia praecox, early onset with progressive cognitive and volitional decline, from manic-depressive psychosis, episodic and periodic with preserved functioning between episodes.
- **His treatise.** The multi-volume Psychiatrie, editions spanning 1909 to 1915, with a ninth edition in 4 volumes prepared for publication in 1927 and later called the bible of modern psychiatry.
- **His institutions.** He founded the experimental journal Psychologische Arbeiten and established the research institute in Munich, which instituted the Golden Kraepelin Medal in 1928.
- **In social psychiatry.** He championed temperance and advocated replacing mechanical restraint and isolation with continuous warm baths.

The Nobel question, which is asked because the answer is conceptual. Kraepelin was nominated at least 8 times between 1909 and 1926, by Robert Gaupp in 1909, Eugen Bleuler in 1917, Oswald Bumke in 1923, Wilhelm Weygandt in 1925 and 1926, and by Ernst Meyer and Giovanni Mingazzini.

- **Why he was not awarded it.** The 1895 will required a discrete tangible discovery, and his sponsors conceded that his achievement was a lifetime systematisation and clinical synthesis rather than an isolated experimental breakthrough.
- **His own view.** He expected that any recognition would honour his experimental work on the work and fatigue curve rather than his classification, and in 1910, invited to nominate a candidate, replied that no researcher in his discipline could compete for such an award.

Philippe Pinel: public-domain portrait

Companion to original page 767

Public-domain print of Philippe Pinel, made by Lambert under Ambroise Tardieu and held by the Rijksmuseum. Use the portrait to discuss humane psychiatric care and its historical context; a portrait does not substantiate every story associated with its subject.

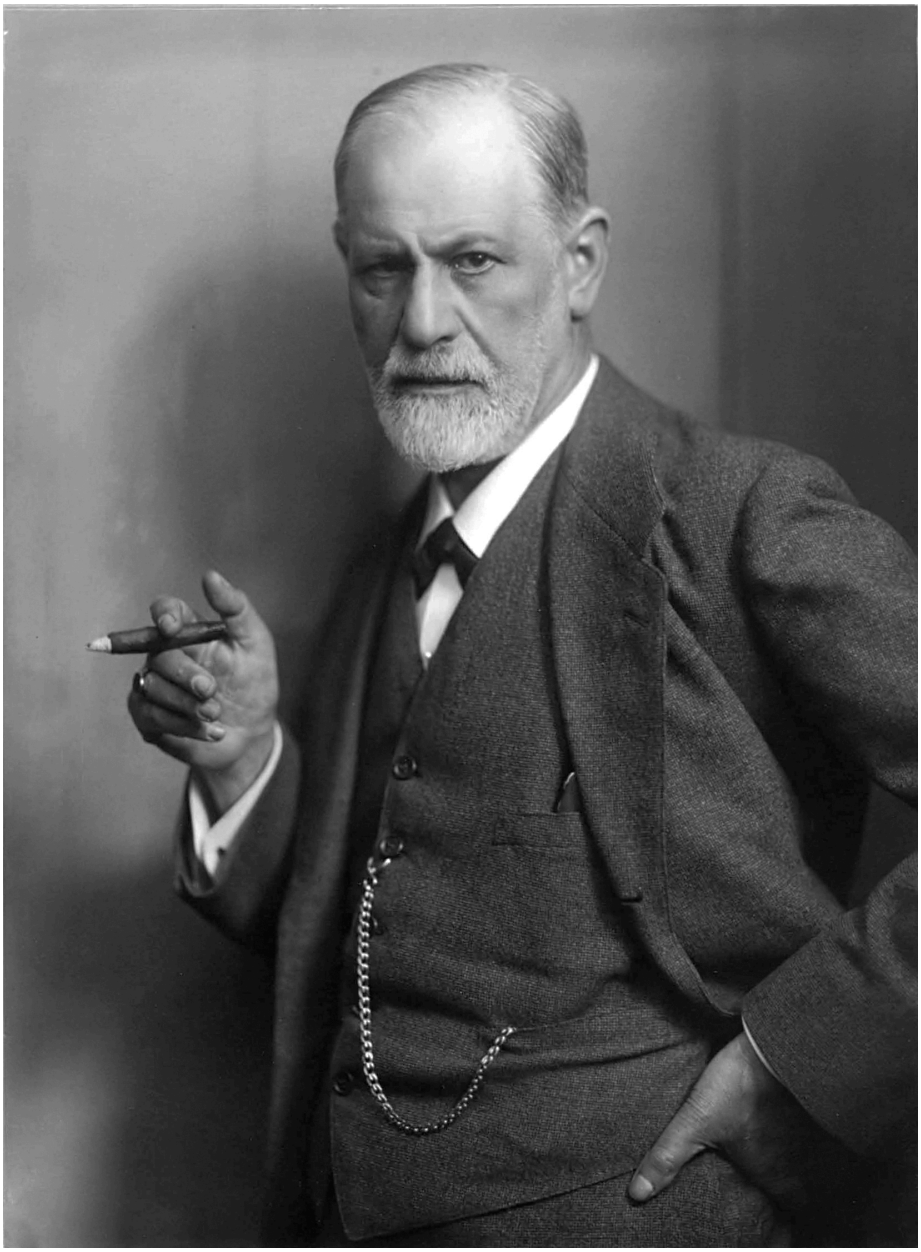
Identify the subject and explain its historical significance.

Lambert (l'Aîné et le Jeune), under Ambroise Tardieu; digital image Rijksmuseum. Portret van Philippe Pinel Pinel (titel op object), RP-P-1910-5377.jpg. CC0. https://commons.wikimedia.org/wiki/File:Portret_van_Philippe_Pinel_Pinel_(titel_op_object),_RP-P-1910-5377.jpg Licence: http://creativecommons.org/publicdomain/zero/1.0/deed.en. Unmodified.

FIGURE	SPOTTER FEATURES	THE IDEA	THE BOOK OR THE LINE
Sigmund Freud	Trimmed grey beard, three-piece suit, cigar; 1856 to 1939	Founded psychoanalysis: unconscious process, repression, transference, infantile sexuality and the tripartite structural theory	**The Interpretation of Dreams in 1899 and Three Essays on the Theory of Sexuality in 1905; his daughter Anna published The Ego and the Mechanisms of Defence in 1936. He is said to have called Kraepelin the super-Pope of psychiatry, and was repeatedly passed over for the Nobel Prize because the committee insisted on laboratory verification**
Eugen Bleuler	Bald crown, trimmed white goatee, wire pince-nez	As director of the Burgholzli clinic he refused Kraepelin's claim that dementia praecox led inevitably to dementia or occurred only in youth, shifting diagnosis from long-term outcome to fundamental psychological features: loosening of associations, affective blunting, autism and ambivalence, with delusions and hallucinations as accessory symptoms	**Dementia Praecox or the Group of Schizophrenias in 1911, which introduced the term schizophrenia, the splitting of psychic functions. In his 1917 nomination of Kraepelin he used the folk metaphor of climbing a glass mountain, where investigators slid back 2 steps for every 1 step taken until Kraepelin cut stable steps into it. Kurt Kolle proposed Bleuler himself in 1958**

Sigmund Freud: public-domain portrait

Companion to original page 768



Public-domain portrait of Sigmund Freud by Max Halberstadt. Use the portrait as a recognition cue, then discuss psychoanalysis and its historical context. The sitter's signature is part of the historical source.

Identify the subject and explain its historical significance.

Max Halberstadt. Sigmund Freud, by Max Halberstadt (cropped).jpg. Public domain. https://commons.wikimedia.org/wiki/File:Sigmund_Freud,_by_Max_Halberstadt_(cropped).jpg Licence: https://commons.wikimedia.org/wiki/File:Sigmund_Freud,_by_Max_Halberstadt_(cropped).jpg#Licensing. Unmodified.

FIGURE	SPOTTER FEATURES	THE IDEA	THE BOOK OR THE LINE
Karl Jaspers	Tall, ascetic, angular face, high forehead, severe gaze; 1883 to 1969	Introduced phenomenological psychopathology: evaluate the patient's subjective conscious experience as reported, setting theoretical presupposition aside	**Three distinctions to give in order. Form against content: form is the phenomenology of the experience, whether it presents as an auditory hallucination or a delusion, and content is the personal and cultural theme inside it. Understanding against explaining: understanding reaches meaningful psychological connection through empathy, explaining reaches somatic causes through natural science. And primary delusions, phenomenologically irreducible and not understandable, arising from delusional mood or delusional perception, against secondary delusion-like ideas that arise understandably from affect or life events**

Three faces and three ideas. Kraepelin classifies by course, Bleuler by cross-sectional psychological splitting, Jaspers by the form of conscious experience.

PIONEER	TREATMENT INTRODUCED	CONTEXT	NOBEL STATUS
Julius Wagner-Jauregg	Malarial inoculation, pyrotherapy, for dementia paralytica	Introduced during the First World War, with published outcomes	**Awarded the prize in 1927**
Antonio Egas Moniz	Prefrontal leucotomy	Published results in 20 patients in 1936, following frontal injections by Maurice Ducoste in the 1920s which he did not acknowledge	**Awarded the prize in 1949**
Manfred Sakel	Insulin coma therapy for schizophrenia	Introduced in the 1930s	**Nominated repeatedly and not awarded**

PIONEER	TREATMENT INTRODUCED	CONTEXT	NOBEL STATUS
Ugo Cerletti and Lucio Bini	Electroconvulsive therapy	Introduced in the late 1930s, using electric current to provoke a seizure	**Nominated repeatedly and not awarded**
Eric Kandel	Molecular and cellular basis of memory storage in neurons	The third and only other prize in this territory	**Awarded the prize in 2000**

These physical treatments were classified as remedia heroica, a nineteenth-century designation for perilous treatments whose use required professional boldness in the face of possible mortality, and the committee rewarded dramatic intrusive therapy over descriptive nosology. Others nominated and not awarded include Kraepelin, Freud, Jaspers, Bleuler, Sakel, Cerletti, the developer of the electroencephalogram, Alzheimer, Bonhoeffer, von Economo, Lombroso and Kretschmer.

The psychopharmacology pioneers. Three names, with the gaps declared.

- **John Cade.** The Australian psychiatrist discovered the calming and antimanic effect of lithium salts in guinea pigs and then in psychiatric patients. The year of the discovery and the target acute serum level are not carried in the library sources for this chapter, and the acute mania level is available in the underused-treatments material above.
- **Jean Delay and Pierre Deniker.** At Sainte-Anne Hospital in Paris they identified the antipsychotic and neuroleptic profile of chlorpromazine. The year of the report and the initial daily dosage are not carried in the sources.
- **One more worth naming.** The French demonstration of the psychotropic properties of a valproate derivative in 1966.

MONOGRAPH	AUTHOR AND YEAR	THE CONCEPT IT INTRODUCED
The Anatomy of Melancholy	Robert Burton, 1621	**A comprehensive Jacobean medical, literary and philosophical examination of depressive states**
Nosographie Philosophique	Philippe Pinel, 1799	**The foundation of moral treatment, and systematic clinical observation unburdened by theological superstition**
Rhapsodieen	Johann Christian Reil, 1803	**Advocated psychological therapies, and coined the medical term psychiatry**
Lehrbuch der Storungen des Seelenlebens	Johann Christian Heinroth, 1818	**An early somatopsychic German text, which coined the term psychosomatic**
A Treatise on Insanity	James Cowles Prichard, 1835	**Introduced moral insanity, severe conduct pathology without intellectual impairment**
Des Maladies Mentales	Jean-Etienne Esquirol, 1838	**Distinguished illusions from hallucinations, and elaborated lypemania and monomania**

MONOGRAPH	AUTHOR AND YEAR	THE CONCEPT IT INTRODUCED
Traite des Maladies Mentales	Benedict Morel, 1860	**Formulated hereditary degeneration theory and coined demence precoce**
Pathologie und Therapie der psychischen Krankheiten	Wilhelm Griesinger, 1871	**Articulated the somatogenic principle that mental diseases are brain diseases**
Die Katatonie	Karl Kahlbaum, 1874	**Defined catatonia as a distinct syndrome of motor tension, mutism, posturing and stupor**
Die psychopathischen Personlichkeiten	Kurt Schneider, 1923	**Systematised personality disorders into descriptive typologies; he later formulated the first-rank symptoms, in a year not carried in the sources**
Psychodiagnostik	Hermann Rorschach, 1932	**The projective diagnostic technique using standardised perceptual inkblots**

The classic monographs, each reducible to one concept, which is how the spotter is scored.

The journals. Two historical, one Kraepelin's own, then the modern set.

- **The Zoist.** Subtitled a journal of cerebral physiology and mesmerism and their application to human welfare, it ran to 13 volumes, with volumes 1 to 8 appearing between 1843 and 1856, and provided an influential British platform for cerebral localisation, phrenology and mesmerism.
- **Psychologische Arbeiten.** Founded and edited by Kraepelin to publish empirical work from his laboratory on pharmacological agents, motor fatigue curves and reaction times.
- **Allgemeine Zeitschrift fur Psychiatrie.** It published key European nosological debates, including Weygandt's 1927 obituary tribute to Kraepelin. Its founding year is not carried in the sources.
- **The modern organs to name.** The American and British journals of psychiatry, Biological Psychiatry, Schizophrenia Research, Acta Psychiatrica Scandinavica and The Lancet, alongside the Indian Journal of Psychiatry as the official journal of the Indian Psychiatric Society.

THE INDIAN FACES, AND THE RANCHI QUESTION

Vidya Sagar is celebrated as a father figure of Indian psychiatry for clinical deinstitutionalisation and family stay-in open wards at Amritsar. Girindra Sekhar Bose was the academic psychoanalyst who founded and first led the Indian psychoanalytical society in Kolkata and corresponded with Freud, formulating the theory of opposite wishes.

Narendra Nath Wig pioneered social and community psychiatry, initiating the Raipur Rani primary care model and conceptualising Dhat syndrome. Jaswant Singh Neki, psychiatrist and poet, conceptualised the Guru-Chela paradigm suited to Indian relational dependency.

Robert Brockelesby Davis modernised the Ranchi institute as Medical Superintendent and established early child clinics. Erna Martha Hoch pioneered Indian child psychiatry, teaching at Lady Hardinge Medical College and at AIIMS and developing services in Srinagar.

Thara Rangaswamy co-founded the schizophrenia research foundation and led community-based rehabilitation and longitudinal outcome work. Jitendra Kumar Trivedi led academic work in clinical psychopharmacology and geriatric psychiatry and presided over the Indian Psychiatric Society.

Keep the two Ranchi names apart. Owen Berkeley-Hill, 1879 to 1944, is the British-era figure documented for his work at the Ranchi mental hospital and an original member of the Indian psychoanalytical society founded in 1922. Davis is the later superintendent credited with modernising the institute.

If asked who founded the Ranchi hospital or in which year, say that the founding year, the original name of the institution and any single-founder attribution are not carried in the sources, and give Berkeley-Hill's documented association instead of a date.

TRAP Two photograph confusions and one priority error. Kraepelin has a thick moustache, neatly parted hair and round spectacles; Bleuler has a bald crown, a pointed white beard and pince-nez. Freud is identified by beard, three-piece suit and cigar; Jaspers is clean-shaven or thinly trimmed, tall, gaunt and austere. And do not confuse Sakel's insulin coma therapy with Meduna's chemical convulsion therapy using pentylenetetrazol, or either with Cerletti and Bini's electrical induction; the year of the chemical method is not carried in the sources.

HOLD THIS

Kraepelin classified by course, Bleuler renamed by splitting, Jaspers separated form from content. Three prizes in this territory: 1927, 1949 and 2000.

WHAT EARNS THE MARKS

- **Giving one idea per face**: course, splitting, or the form of conscious experience
- **Explaining the Nobel omission conceptually**: the will required a discrete tangible discovery
- **Reducing each monograph to one concept**: that is how the spotter is scored
- **Keeping the two Ranchi figures apart**: and declining to invent a founding year

SEE ALSO 13 History and the Indian lineage 24 Image spotters 01 The ICD-11 architecture

The whole note in one table	
ICD-11	Approved May 2019, statistics from January 2022; 21 groupings in place of a decimal cap of 10; essential features not symptom counts; catatonia promoted, schizophrenia subtypes out, 6 dimensional qualifiers; personality dimensional in 3 severity levels and 5 trait domains; depressive episode 5 of 10 for 2 weeks.
DSM-5-TR	March 2022, a text revision; prolonged grief moved from Section III to Section II; 12 months in adults and 6 in youth, 1 of 2 core and 3 of 8 accessory over the last 1 month; ICD-11 asks 1 of 10 after 6 months, which is why its prevalence runs higher at 5.4% to 6.8% against 4.7%.
INDIAN GUIDELINES	Draft series: consultation-liaison December 2021, emergencies and brain stimulation October 2022, wellbeing October 2023, cognition November 2024. Cite in four beats: problem, Indian guideline, operational recommendation, legal and contextual justification.
NON-DOPAMINERGIC ANTIPSYCHOTICS	Xanomeline with trospium: central M1 and M4 agonism, peripheral blockade, first approved non-dopaminergic mechanism, late 2024. Ulotaront: full trace amine receptor 1 agonist with 5-HT1A, no D2 binding, PANSS difference of 7.5 points against placebo.
CLINICAL HIGH RISK	Three clusters; 73% do not transition within 2 years and nearly half of those remit; first line is cognitive behavioural therapy, family support and monitoring; critical period 2 to 5 years, and gains attenuate on transfer back to generic care at 2 years.
RAPID-ACTING ANTIDEPRESSANTS	Ketamine 0.5 mg/kg over 40 minutes, response within 72 hours; esketamine 28 mg intranasal on site with 2 hours of observation; dextromethorphan 45 mg with bupropion 105 mg, benefit at 1 to 2 weeks; brexanolone 60 hours intravenous against zuranolone 50 mg for 14 days.
INFLAMMATION AND LITHIUM	Hedges g 0.40 for anhedonia and 0.35 for severity with no categorical advantage; lithium at 0.8 to 1.0 mmol/L for acute mania, trough range 0.6 to 1.2 mM, up to 12 months for full prophylaxis, and only 5.90% monotherapy in one national registry.
ARTIFICIAL INTELLIGENCE	Standalone accuracy 48.1% to 62.0%; psychosis transition balanced accuracy 85.5%; 46% of genomic studies carry leakage-prone feature selection; models fail on external cohorts; teleconsultation cannot open a supported admission or prescribe Schedule X.
LANDMARK TRIALS	74% discontinued before 18 months; perphenazine matched the second-generation agents at 300 to 500 dollars per month less; clozapine median 10.5 months in the efficacy pathway; 21 antidepressants all beat placebo; 32 antipsychotics differ mainly in tolerability.
INDIAN EPIDEMIOLOGY	Current 10.56%, lifetime 13.67%, treatment gap 84.5%; common mental disorders 10.0%, severe

The whole note in one table	
	0.8%, psychosis 0.4%, alcohol 4.6% with an 86.3% gap, tobacco 13.1%.
UNDERUSED TREATMENTS	Electroconvulsive therapy: Cerletti and Bini 1938, relapse up to 85% without maintenance. Clozapine: 2 trials at 600 mg chlorpromazine equivalent for 6 weeks with 80% adherence, 41% response, 82% within 2.8 years against 31% later, agranulocytosis 0.4%.
ETHICS	Four principles; four boxes in order; capacity stringency rises with decision gravity; Madrid 1996 with seven general and twelve specific guidelines; the 2020 Code in four sections adding family caregivers; consent inside a power differential is not consent.
BIOSTATISTICS	Alpha 0.05, beta up to 0.2, power 80%; design decides causality and tests only measure association; concealment before entry, blinding after allocation; observational and trial estimates agree at a pooled ratio of 1.08.
PEDAGOGY	Knows, knows how, shows how, does; written papers correlate with the apex at 0.3 to 0.5; reliability needs 6 to 14 observations; Pendleton is learner-first in four steps within 5 minutes, and beats the sandwich.
EMERGENCIES AND DISASTER	Eight-step ladder with a show of force of 4 to 6 staff before restraint; 22% of conflict-affected populations carry a disorder, 13% mild and 9% moderate to severe; psychological first aid in five principles and eight actions; routine debriefing is avoided.
NEUROBIOLOGY	Four dopamine pathways and 5 receptor subtypes; permissive hypothesis; excitotoxicity through calcium; striatal dopamine and acetylcholine balance explains both acute extrapyramidal effects and tardive dyskinesia; 70% genetic overlap between schizophrenia and bipolar disorder.
PREVENTION	When, who and how: Leavell and Clark levels, the Institute of Medicine tiers of universal, selective and indicated, and the social determinants acting through restricted choice, chronic stress and gene-environment interaction. Screening finds latent disease; high risk without symptoms is selective.
DRUG SPOTTERS	Name, strength, form, schedule, then the one precaution. Thiamine before glucose; depot by Z-track and not intravenously; sublingual naloxone is inactive and exists for the syringe; methylphenidate is Schedule X with duplicate prescription.
INSTRUMENT SPOTTERS	Bipolar brief pulse square wave, 0.25 to 1.5 ms at 0.9 A; static impedance above 100 and below 3000 ohms and dynamic lower; bite block on the molars; figure-of-eight focal to 1.5 cm against helmet coils at 4 to 5 cm; fuel cell for evidence, semiconductor for screening.
SCALE AND IMAGE SPOTTERS	Depression scale 17 items, syndrome scale 30 constructs on a 7-point Likert, cognitive examination out of 30, Gestalt set 9 cards of 3 by 5 inches. Dense core with a halo is brainstem Lewy, no

The whole note in one table	
	halo and argyrophilic is Pick; mixed pathology in 20% to 25% drops Lewy core-feature sensitivity to 46%.

HOLD THIS

At this table, the candidate who names the object, states the one discriminator, and says which figure will be confirmed outscores the candidate who guesses a number. Breadth is held as pairs: instrument and change, trial and endpoint, drug and precaution, image and its near neighbour.

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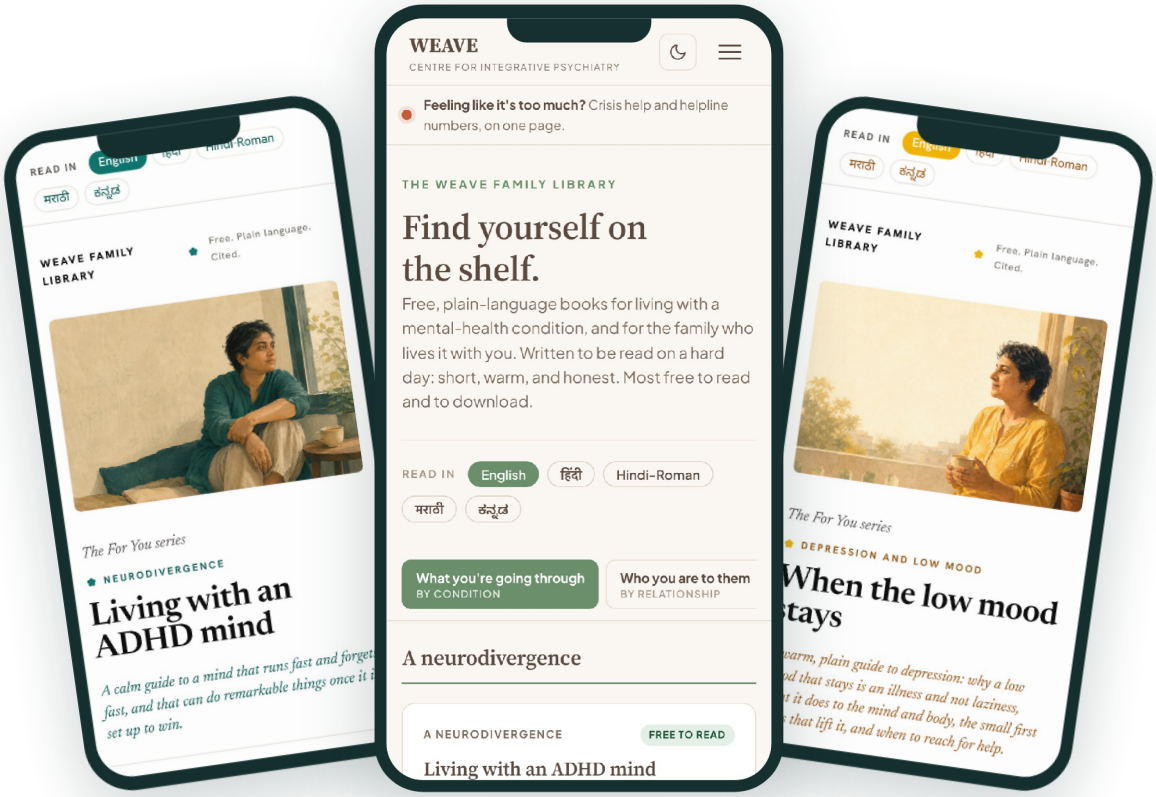

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These notes started as messages in a room full of residents helping each other through. That room is still open. Bring your doubts, your corrections, and the questions the textbooks do not answer.

Join the study group

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