WEAVE 100

MD Psychiatry theory. DNB revision fits too.

The Complete Set. Papers I to IV

Four hundred ten-mark questions, solved as answer maps

Eight examining boards. Seventy-eight sittings, counted once and read four ways. This is where the marks have actually gone across all four papers of the MD psychiatry written examination: the basic sciences and the method behind them; the psychoses, mood and the adult disorders; child, forensic and the special populations; and the organic paper, its drugs and its community. Fifty-four areas, each weighted by what was asked, then four hundred questions drawn in that proportion and solved as one-page answer maps. Every question in this book was set by a board. None was invented.

Dr. Wilfred Dsouza · Dr. Niharika Reddy

WEAVE

A NOTE ON WHO MADE THIS

Weave, Centre for Integrative Psychiatry

Weave is a psychiatry practice and a publishing house for the things clinicians and families actually need to read. Two consultant psychiatrists run it, online across India and in person in Mumbai.

WHAT WEAVE DOES	Integrative psychiatry, online and in Mumbai, and a free library for residents, clinicians and families.
WHO RUNS IT	Dr. Niharika Reddy and Dr. Wilfred D'souza, Consultant Psychiatrists.
WHAT THAT MEANS HERE	This book is free because the practice pays for it. There is nothing to buy on any page of it.

WEAVE 100

The Complete Set. Papers I to IV

Four hundred ten-mark questions, solved as answer maps

THE WEAVE 100 SERIES

TITLE

Weave 100: The Complete Set, Papers I to IV

The four books of Weave 100 in one volume. One hundred solved ten-mark questions for each paper of the MD psychiatry written examination, four hundred in all.

WRITTEN BY

Dr. Wilfred Dsouza and Dr. Niharika Reddy

PUBLISHED BY

Weave, Centre for Integrative Psychiatry · weave.clinic

EDITION

Omnibus edition, first compiled 2026. Free to read, share and print for study.

First edition (R1), August 29, 2026.

Assembled from the four Weave 100 R1 volumes.

SOURCES

Question records were read from the published papers and archives of the boards named on the back cover. Board names appear throughout because the count is only meaningful with them. No board has endorsed this book and none was asked to.

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, thresholds and statutory detail against the current references and your local protocols before any clinical use.

A WORD BEFORE THE QUESTIONS

If this book helps you

Weave 100 is free, and it stays free. It was built at Weave, a psychiatry and psychotherapy practice in Mumbai, by the two of us on the cover, as our own revision for the same exams you are sitting.

If these answer maps help you, the simplest way to support the work is to follow it: **@weave.clinic** on Instagram carries everything Weave publishes, and **@doc.wilfred.md** and **@doc.niharika.md** are the clinicians behind it. A follow costs nothing and it keeps books like this one free.

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THE PRACTICE AND THE LIBRARY

Scan any of the four, or type the address. Every code on this page is drawn as vector, not photographed.

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02 **Functional neuroanatomy and neuroimaging** I-14 to I-25 · 12 slots · 13.5% 29

03 **Personality theory and psychodynamics** I-26 to I-35 · 10 slots · 10.5% 43

04 **Learning, memory and general psychology** I-36 to I-44 · 9 slots · 9.9% 55

05 **Psychometry and assessment** I-45 to I-53 · 9 slots · 9.1% 66

06 **Genetics in psychiatry** I-54 to I-62 · 9 slots · 9.0% 77

07 **Social, cross-cultural and evolutionary psychology** I-63 to I-70 · 8 slots · 7.8% 88

08 **Biostatistics and epidemiology** I-71 to I-78 · 8 slots · 7.6% 98

09 **Research methodology** I-79 to I-84 · 6 slots · 5.3% 108

10 **Developmental psychology and attachment** I-85 to I-90 · 6 slots · 5.1% 116

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02 **Substance use: opioids, other drugs and treatment** II-10 to II-18 · 9 slots · 9.4% 155

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04 **Schizophrenia: aetiology, phenomenology and course** II-28 to II-35 · 8 slots · 8.4% 177

05 **Trauma, dissociative and somatic symptom disorders** II-36 to II-43 · 8 slots · 8.4% 187

06 Schizophrenia: treatment and antipsychotics II-44 to II-51 · 8 slots · 8.3% 197

07 Mood disorders: pharmacotherapy II-52 to II-58 · 7 slots · 7.5% 207

08 Mood disorders: syndromes and nosology II-59 to II-65 · 7 slots · 7.5% 216

09 Psychotherapies II-66 to II-72 · 7 slots · 6.9% 225

10 Personality disorders II-73 to II-79 · 7 slots · 6.6% 234

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05 Mental health legislation and disability law III-41 to III-49 · 9 slots · 9.6% 325

06 ADHD and disruptive behaviour disorders III-50 to III-58 · 9 slots · 9.5% 336

07 Geriatric, consultation-liaison and transcultural psychiatry III-59 to III-66 · 8 slots · 8.7% 347

08 ECT and neurostimulation III-67 to III-74 · 8 slots · 7.9% 357

09 Emergency psychiatry III-75 to III-81 · 7 slots · 6.5% 367

10 Perinatal and women's mental health III-82 to III-88 · 7 slots · 6.3% 376

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02 **Dementias: types and differential** IV-12 to IV-21 · 10 slots · 11.4% 422

03 **Consultation-liaison and systemic medicine psychiatry** IV-22 to IV-30 · 9 slots · 10.1% 434

04 **Clinical neurology, headache and sleep neurology** IV-31 to IV-38 · 8 slots · 8.0% 445

05 **Stroke, TBI and focal brain syndromes** IV-39 to IV-46 · 8 slots · 7.9% 455

06 **Movement and degenerative disorders in psychiatry** IV-47 to IV-53 · 7 slots · 6.7% 465

07 **Endocrine and metabolic psychiatry** IV-54 to IV-59 · 6 slots · 6.2% 474

08 **Recent advances and landmark studies** IV-60 to IV-65 · 6 slots · 5.9% 482

09 **Drug emergencies, interactions, TRD and neurostimulation** IV-66 to IV-71 · 6 slots · 5.6% 490

10 **Psychopharmacology: principles and drug classes** IV-72 to IV-76 · 5 slots · 5.0% 498

11 **Epilepsy and psychiatry** IV-77 to IV-81 · 5 slots · 4.9% 505

12 **Nutritional, toxic and metabolic encephalopathies** IV-82 to IV-86 · 5 slots · 4.2% 512

13 **Neuro-infective and autoimmune neuropsychiatry** IV-87 to IV-91 · 5 slots · 4.0% 519

14 **Delirium and amnestic syndromes** IV-92 to IV-96 · 5 slots · 3.9% 526

15 **Cognitive assessment and dementia management** IV-97 to IV-100 · 4 slots · 2.8% 533

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HOW TO USE THIS BOOK

Four parts, one question a page

The four Weave 100 books in one block, in paper order. Each part opens on a divider, then a three-page weightage chapter, then its areas in weight order, and closes on its own ten-set appendix. Page numbers run straight through, so the contents, the index and every cross-reference point at one number that means one page. Every other page inside a part is one question and its answer map, and nothing continues onto a second page. Five blocks, always in the same order.

1	**Question band**	The slot number, the question as a board set it, and three chips: the mark split, the area, and where the question has been seen.
2	**Skeleton**	Three to five rows. The mark column sums to ten, so you can see the allocation before you read a word of content.
3	**Body**	Two to four components, chosen for the question: a comparison table, a flowchart, a plate, typed lines, at most one pitfall box.
4	**What earns the marks**	Three or four things an examiner ticks. Each one names something that is on the page above it.
5	**Links**	The related slots in this book and, where the map names one, the Weave Sprint day that teaches the underlying topic in full.

The colour code

Structure	The shape of the answer: headers, rules, table heads, the question band. When a line is indigo it is telling you how the answer is organised.
Key number	A figure to carry exactly: a mark split, a threshold, a count, a cut-off. Amber means memorise the number, not just the shape of it.
Trap	The mistake the examiner is waiting for. When a line is red it is warning you off an error that looks right until it costs you the mark.

Two ways to work. Read an area straight through, banner first, to see how the boards have carved it up. Or use the ten sets that close each part, forty in all: each is ten questions and one hundred marks from that paper, drawn one from each rank decile, so a set is a paper-shaped sitting rather than ten easy questions or ten hard ones.

The chips are traceable. A recurrence chip names the boards and counts against a stated denominator. Three of seventy-eight sittings means three out of the seventy-eight this book counted, with the boards named so you can check. It is the same seventy-eight behind all four parts, because one sitting sets all four papers.

PART ONE OF FOUR

PAPER I

Brain, Mind and Method

The basic sciences of psychiatry: neurochemistry and neurophysiology, functional neuroanatomy and neuroimaging, personality theory and psychodynamics, learning and memory, psychometry and statistics, and the method behind all of it.

12	100	78	8	14.5%
AREAS, IN WEIGHT ORDER	ANSWER MAPS	SITTINGS COUNTED	EXAMINING BOARDS	HEAVIEST AREA SHARE

ANCHOR 11 · HIGH 18 · RECURRENT 3 · SINGLE 68

Heaviest area in this paper: Neurochemistry and neurophysiology. The weightage chapter overleaf shows how every area in Paper I was weighted, and what the weight could not promise.

THE WEIGHTAGE CHAPTER, PAGE ONE OF THREE

Where the marks have gone

Nothing in this book is a prediction. It is a count of 78 sittings from 8 examining boards, and a statement of where those boards have put their marks in Paper I.

Twelve areas, weighted. The university boards are pooled by how much each is trusted, DNB is held out and given a fixed thirty percent, and the resulting weight becomes a share of one hundred and then a whole number of slots. The ledger of which boards were counted, how their papers reached us, and how far each one is trusted, is on the page opposite.

AREA, IN WEIGHT ORDER	UNIV POOL	DNB	WEIGHT	SHARE	SLOTS
01 Neurochemistry and neurophysiology	0.455	0.532	0.479	14.52%	13
02 Functional neuroanatomy and neuroimaging	0.456	0.422	0.446	13.53%	12
03 Personality theory and psychodynamics	0.321	0.407	0.347	10.52%	10
04 Learning, memory and general psychology	0.335	0.304	0.326	9.89%	9
05 Psychometry and assessment	0.318	0.254	0.299	9.07%	9
06 Genetics in psychiatry	0.252	0.396	0.295	8.96%	9
07 Social, cross-cultural and evolutionary psychology	0.326	0.098	0.257	7.81%	8
08 Biostatistics and epidemiology	0.232	0.295	0.251	7.61%	8
09 Research methodology	0.115	0.311	0.174	5.27%	6
10 Developmental psychology and attachment	0.192	0.110	0.168	5.09%	6
11 Nosology, classification and descriptive psychopathology	0.051	0.422	0.162	4.92%	6
12 Neuroendocrinology and basic psychopharmacology	0.077	0.127	0.092	2.79%	4

Univ pool is the trust-weighted verdict of the seven university boards; DNB is the national paper, held out. Weight = 0.70 times the pool plus 0.30 times DNB. Share is that weight as a fraction of Paper I. Slots are the share turned into whole questions by largest remainder, with a floor of two and a cap of twenty; Paper I reached neither.

THE WEIGHTAGE CHAPTER, PAGE TWO OF THREE

How the hundred were picked

BOARD	SITTINGS COUNTED	HOW THE PAPERS REACHED US	TRUST
RGUHS	12	Hand transcription and public mirrors of the printed papers	0.806
KUHS	9	Official university portal PDFs	0.600
TN-MGRMU	35	Public archive of the printed papers, exact sitting dates recovered	0.480
KNRUHS	3	Official university portal PDFs	0.300
MUHS	2	Two watermark-confirmed papers, read by OCR. A third-party recurrence compilation was tested against them, matched none, and was removed from the sitting count	0.267
NTRUHS	2	Mirror text of the printed papers	0.160
WBUHS	2	Public archive of the printed papers	0.180
DNB	10	Official NBE material; 32 of 40 files verified by checksum against the board's own published path	fixed 0.30

Trust is how far a board sets the university pool: its standing, its number of sittings, and the quality of its evidence. DNB sits outside the pool. A third-party compilation claiming thirteen further MUHS sittings was tested against the only confirmed MUHS papers inside its own window, matched none of them, and was removed from this ledger; its topic distribution still contributes to area mass at reduced quality.

1 Count the sittings

78 sittings from 8 boards, each weighted so that it halves roughly every six years and is reduced again when the paper reached us as a mirror, an archive or a transcription rather than an official file.

2 Weight the areas

Each board reads each area twice: breadth, how often it examines there, and mark share, how much of its marks land there. The two are averaged, the boards are pooled by trust, and DNB is held out at a fixed thirty percent.

3 Turn weight into slots

The twelve weights become shares of one hundred, then whole slots by largest remainder, with a floor of two and a cap of twenty so no area disappears and none swallows the book.

4 Score every eligible question

Four components: weighted recurrence (45 per cent), recency (30), how many boards have asked it (15), and how well it is shaped as a ten-mark question (10). Normalised across all four papers, so one score means one thing everywhere.

5 Fill the slots

The highest scorers in each area, up to that area's slot count, near-duplicates merged first. All 100 were filled from questions actually set: 11 anchor, 18 high, 3 recurrent, 68 single-appearance.

THE WEIGHTAGE CHAPTER, PAGE THREE OF THREE

What this book cannot promise

A frequency count is a strong prior, not a prophecy. Here is exactly what that sentence costs, stated before you rely on it.

It does not predict a paper. Nothing here says what your board will set. It says where the marks have gone across 78 sittings, which is a different claim and a smaller one. A board that changes its examiner, its syllabus or its habits can leave every number in this book behind in one sitting.

Most questions were seen once. Of the 403 selectable Paper I questions in the record, 342 appeared in a single sitting. That is not a defect in the counting, it is what happens when eight boards set different papers: the same TOPIC returns constantly while the same QUESTION rarely does. So the selection is driven by AREA weight, where the signal is dense, and each map prints a band rather than a frequency claim about its own question.

The band on a map is a description, not a forecast. Anchor means this question has come back across boards and years. Single means it was set once in the record we hold. A single-appearance question is here because its AREA is heavy, and it is a fair worked example of that area, which is the only thing it is offered as.

Nothing here is invented. All 100 slots in this book are questions a board actually set. Where a paper in this series cannot fill its slots from observed questions, the shortfall is authored to syllabus and labelled as such on the map. Paper I has no such slots.

The record is uneven, and openly so. Two of the eight boards set papers in a legacy structure that does not map onto the modern four, so their sittings are counted against all of their papers rather than one. Two more contribute only two sittings each. The trust column on the first page of this chapter is where that unevenness is priced in, and it is the number to argue with if you want to argue with the weighting.

No board has endorsed this. Board names appear on every page because a count that hides its sources is not a count. None of them was asked, and none of them is responsible for anything in this book.

AREA 01 OF 12 · P1-A02

Neurochemistry and neurophysiology

13	14.5%	I-01	I-13
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 HIGH 2 SINGLE 9

REVISION ORDER

The memory block first and together: four slots share one answer and two of them carry the anchor weight in this area. Sleep next, as a set of three, and the classification slot last of the three because it is the only one that turns clinical. The dopamine pathways after that, short and set often. The three transmitter slots, the electrophysiology slot and the markers slot are independent and each stands alone.

WHAT IS IN THIS AREA

Memory in four slots (the neurobiology with its clinical relevance, the cortical contribution with memory impairment across illnesses, the types of memory with rehabilitation in chronic illness, and synaptic plasticity). Sleep in three (the neurobiology of sleep and wakefulness, polysomnography with the stages and their EEG findings, and the classification with restless legs and a benzodiazepine taper). Single transmitters in three: nitric oxide, glutamate and the endocannabinoids. The dopamine pathways, the electrophysiological abnormalities of four disorders, and the biological-markers slot.

I - 01

Describe the Neuro-biology of memory and its relevance in clinical practice. [10]

10 NEUROCHEMISTRY AND NEUROPHYSIOLOGY

KUHS, MUHS, RGUHS, TN-MGRMU - 6 APPEARANCES, 6 OF 78 SESSIONS

SKELETON

2	The four stages	Encoding, consolidation, storage, retrieval, each a separate failure
3	Systems and their anatomy	One structure per system, not one memory centre
3	Cellular basis	Long term potentiation, and what makes a trace permanent
2	Clinical relevance	The bedside uses that follow from the anatomy

THE LINE WORTH WRITING FIRST

Memory is not one faculty in one place. It is several parallel systems, each with its own anatomy, its own timescale and its own way of failing. An answer that names the systems separately has already earned more than one that describes memory in general.

SYSTEM	ANATOMY	WHAT IT HOLDS	WHERE IT FAILS
Working	Dorsolateral prefrontal cortex	Seconds of actively held material	Schizophrenia, ADHD, delirium
Episodic	Hippocampus, medial temporal lobe	Events with a time and a place	Alzheimer disease, Korsakoff, ECT
Semantic	Anterolateral temporal cortex	Facts stripped of their context	Semantic dementia
Procedural	Striatum and cerebellum	Skills and habits	Parkinson disease; spared in amnesia

The last row is the one that separates the bands: procedural learning survives dense amnesia, which is what proves the systems are separate.

- **Cellular basis.** Long term potentiation at glutamatergic synapses. Strong activity relieves the magnesium block on the NMDA receptor, calcium enters, kinases move more AMPA receptors into the membrane, and the synapse is stronger.
- **Making it last.** Early potentiation needs no new protein. Late potentiation does: calcium signalling reaches CREB, new proteins are made, spines change shape. That gene step is why consolidation takes hours and why it can be blocked.
- **Clinical relevance.** Sparing of procedural learning guides rehabilitation; ECT produces retrograde and anterograde deficits that mostly recover; benzodiazepines and alcohol block encoding rather than erase storage, which is what blackout means.

WHAT EARNS THE MARKS

- **Four stages named before any anatomy.** It lets you say which stage a drug or a lesion damages.
- **Each system with its own structure,** and procedural memory identified as the spared one.
- **Relevance made concrete.** Blackout, ECT and rehabilitation are the three the examiner expects.

SEE ALSO Slot I - 10 types of memory and rehabilitation Slot I - 13 synaptic plasticity SPRINT Day 1, Functional Neuroanatomy and Neurophysiology

I - 02

Describe the role of cortex in memory formation. Discuss about memory impairment in various psychiatric illnesses. [5+5]

5 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	**Which cortex, for what**	Prefrontal for control, sensory cortex for the trace
3	**Systems consolidation**	How a trace moves from hippocampus to neocortex
3	**Impairment by illness**	Which stage fails, and whether it persists
2	**What it changes**	Assessment and rehabilitation that follow

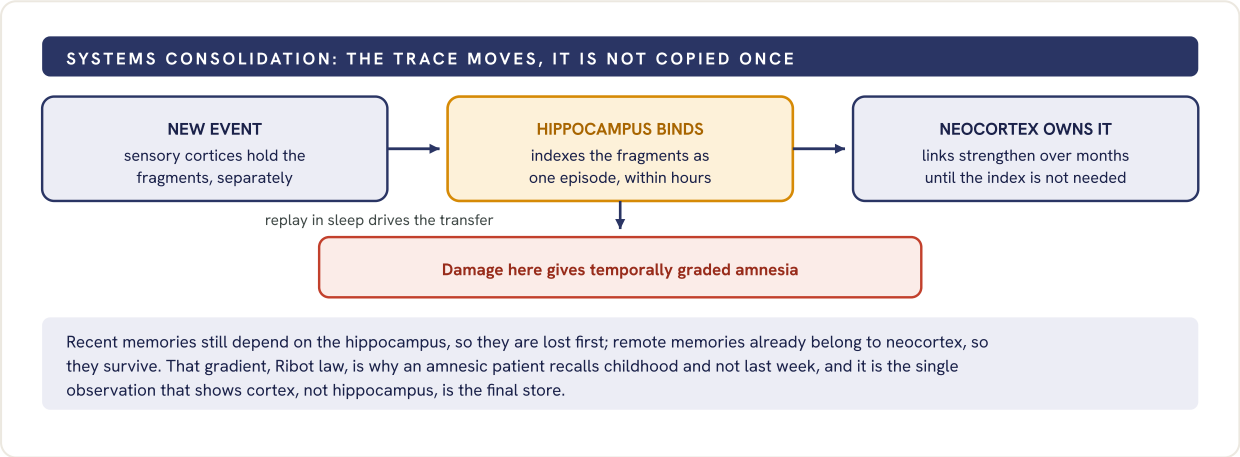


Fig 002.1 Consolidation drawn as a handover: cortex holds the fragments throughout, the hippocampus indexes them, and the index is gradually made unnecessary.

- **Which cortex does what.** Dorsolateral prefrontal cortex runs encoding strategy and retrieval search; ventrolateral prefrontal cortex selects among competitors; the sensory cortices are where the content itself is finally stored.
- **Impairment by illness.** Schizophrenia: verbal declarative and working memory deficits present from onset and largely independent of symptoms. Depression: effortful encoding fails with attention and motivation, plus overgeneral autobiographical recall. Bipolar disorder: deficits persist into euthymia.
- **The reversible causes, kept separate.** Delirium, benzodiazepines and alcohol, ECT, thiamine deficiency, hypothyroidism and dissociative amnesia. Naming which of these are treatable before describing the untreatable is the marked move.

WHAT EARNS THE MARKS

- **Cortex named as the final store,** with the hippocampus as a temporary index.
- **The temporal gradient, and what it proves.** It is the evidence, not just a clinical sign.
- **Deficits described by stage,** encoding against retrieval, rather than as poor memory.
- **Reversible causes listed separately** from the disorders themselves.

SEE ALSO Slot I - 01 neurobiology of memory Slot I - 23 hippocampal formation **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 03

Describe in brief neurobiology of sleep and wakefulness. [10]

10 NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB, KUHS, RGUHS, WBUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

3	**Ascending arousal system**	Two branches out of the brainstem, and their transmitters
2	**Sleep promoting system**	The preoptic switch, GABA and galanin
2	**Flip-flop switch**	Mutual inhibition, and what orexin does to it
2	**REM sleep**	The cholinergic and aminergic reciprocal model
1	**Drug correlates**	Each sedating drug named to the system it acts on

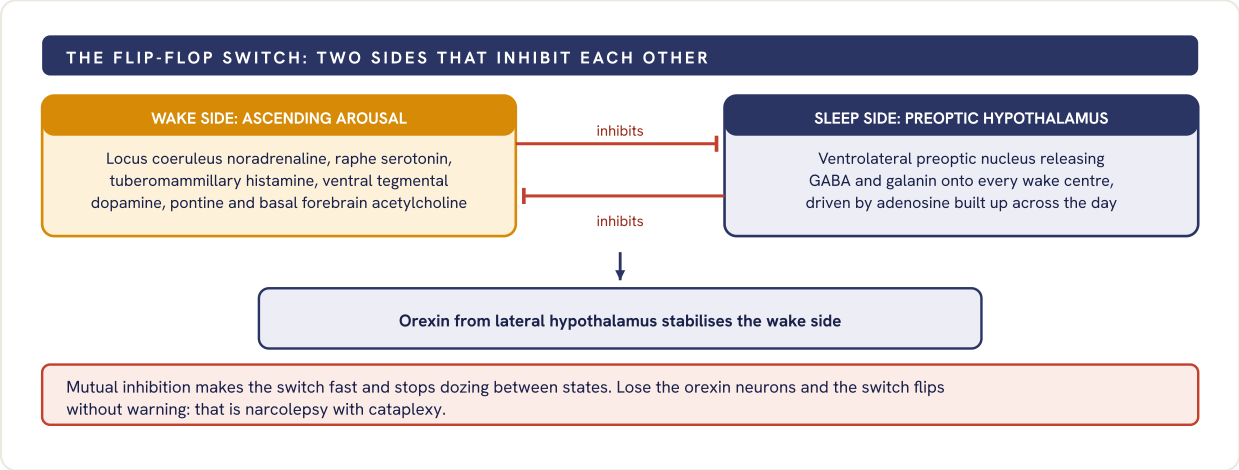


Fig 003.1 Wake and sleep drawn as two mutually inhibiting populations with orexin as the stabiliser, which is why the state changes abruptly rather than by degrees.

- **REM sleep.** The reciprocal interaction model: cholinergic pontine neurons fire to generate REM while noradrenergic and serotonergic neurons fall silent. Loss of that aminergic tone with active cholinergic drive gives the vivid dreams and the muscle atonia.
- **Two clocks, not one.** A homeostatic drive from accumulating adenosine, and a circadian drive from the suprachiasmatic nucleus signalling through melatonin. Sleep happens where the two align.

DRUG	WHAT IT ACTS ON	THE EFFECT THIS PREDICTS
Benzodiazepines, Z drugs	GABA-A receptor, the sleep side	Faster sleep onset, less slow wave sleep
Sedating antihistamines	Histamine, a wake transmitter	Sedation, and daytime hangover
Caffeine	Adenosine receptor blockade	Removes the homeostatic sleep signal
Melatonin agonists	MT1 and MT2, the circadian arm	Shifts timing rather than sedating

WHAT EARNS THE MARKS

- **Both sides of the switch, with transmitters attached.** A list of nuclei without transmitters is half the answer.
- **Mutual inhibition explained as a mechanism,** and orexin given its stabilising role.
- **Drugs mapped back onto the diagram,** which is what makes this a pharmacology answer too.

SEE ALSO Slot I - 09 polysomnography and sleep stages Slot I - 11 classification of sleep disorders **SPRINT** Day 1, Functional Neuroanatomy and Neurophysiology

I - 04

Describe the synthesis, mechanism of action and neuropathological role of nitric oxide. [3+3+4]

3 + 3 + 4 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Synthesis**	One substrate, three enzymes, and what triggers them
3	**Mechanism of action**	Why it breaks every rule of a classical transmitter
4	**Neuropathological role**	The same molecule as signal and as poison

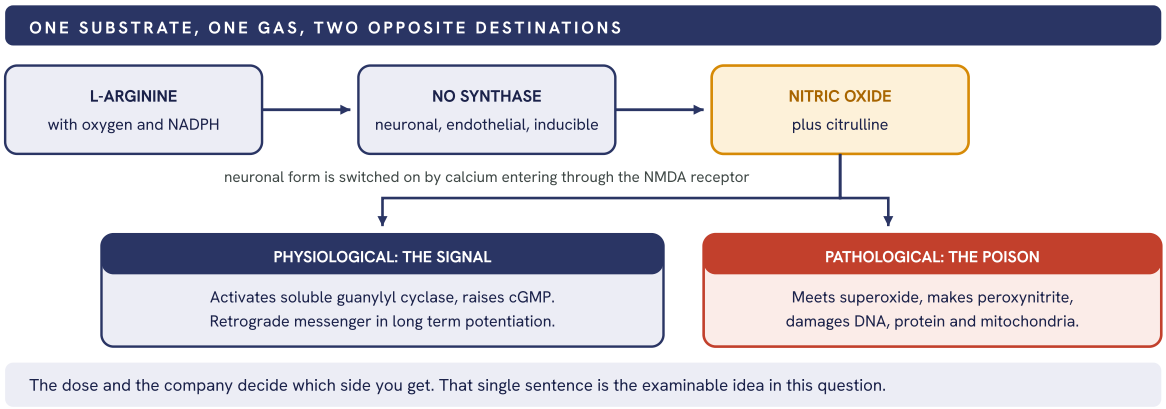


Fig 004.1 Nitric oxide drawn once and used twice: the same synthetic step feeds a signalling arm and an excitotoxic arm, and only the local conditions choose between them.

- **Why it is unusual.** A gas: not stored, not exocytosed, no membrane receptor. Made on demand, it crosses membranes freely and acts within microns for seconds, signalling backwards from the postsynaptic cell to the terminal that fed it.
- **Where it turns pathological.** Excess glutamate opens NMDA receptors, calcium floods in, neuronal NO synthase runs hard, and peroxynitrite forms. This is the final common path of excitotoxicity in stroke, neurodegeneration and, as an inducible form in microglia, in neuroinflammation.

THE PSYCHIATRIC HOOK

Nitric oxide signalling is downstream of the NMDA receptor, which is the same receptor implicated in the glutamate model of schizophrenia and targeted by ketamine. It is also the mediator of penile erection, which is why phosphodiesterase inhibitors treat one of the commonest antidepressant side effects.

WHAT EARNS THE MARKS

- **The synthetic step written out,** arginine with oxygen and NADPH to nitric oxide and citrulline, and all three synthase isoforms named.
- **The four ways it breaks transmitter rules.** Not stored, not exocytosed, no receptor, membrane permeable.
- **Signal and poison as one mechanism,** separated by amount and by the presence of superoxide.

SEE ALSO Slot I - 06 glutamate receptors and pathways Slot I - 13 synaptic plasticity

I - 05

Describe the electrophysiological abnormalities in the following conditions. a) Schizophrenia. b) Catatonia. c) Obsessive compulsive disorder. d) Delirium. [3+2+2+3]

3 + 2 + 2 + 3 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Schizophrenia	Evoked potentials and gating, not the resting record
2	Catatonia	Mostly to exclude something, and what that something is
2	Obsessive compulsive disorder	The error signal, and frontal rhythm
3	Delirium	The one diagnosis where EEG genuinely decides

CONDITION	RESTING EEG	EVOKED AND EVENT RELATED	WHAT IT IS USED FOR
Schizophrenia	Non-specific; no diagnostic pattern	Reduced P300 amplitude with delayed latency, P50 gating failure, reduced mismatch negativity, weak gamma response	Endophenotypes for research, not diagnosis
Catatonia	Normal, or diffuse non-specific slowing	Not diagnostically useful	To exclude non-convulsive status epilepticus
Obsessive compulsive disorder	Increased frontal beta in some series	Enhanced error related negativity, present before onset and in unaffected relatives	A candidate trait marker
Delirium	Diffuse slowing, loss of alpha, more theta and delta; triphasic waves in hepatic causes	Slowing tracks severity and settles as the patient clears	Separates delirium from a functional psychosis

Amber cells are the discriminators. Only one row on this page describes a test that changes what you do next, and saying which one is itself a mark.

- **What each measure means.** P50 gating asks whether the brain suppresses a repeated click; mismatch negativity, whether it notices an odd tone unattended; P300, whether it registered a rare target. All three are weak in schizophrenia and attention independent, which is why they survive as endophenotypes.
- **Delirium, in practice.** Slowing appears early and is graded, so a normal record in a confused patient argues against delirium and toward a functional cause.
- **The catatonia trap.** Catatonia and non-convulsive status epilepticus look alike at the bedside, and a benzodiazepine improves both. The EEG is what separates them, so it is ordered for exclusion rather than for confirmation.

WHAT EARNS THE MARKS

- **Resting EEG kept apart from evoked potentials.** Merging them is the commonest error on this question.
- **Each finding named with its purpose,** research marker or clinical decision.
- **Non-convulsive status epilepticus named under catatonia,** which is the safety point the question is testing.
- **Delirium identified as the one diagnostic use,** with the direction of the change stated.

SEE ALSO Slot I - 08 biological markers Slot I - 09 sleep stages and EEG SPRINT

Day 1, Functional Neuroanatomy and Neurophysiology

I - 06

Describe the synthesis, receptors and major pathways of glutamate in brain. [3+3+4]

3 + 3 + 4 NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Synthesis and disposal**	The glutamine cycle, and why the astrocyte is essential
3	**Receptors**	Three ionotropic, three metabotropic groups, with their jobs
4	**Pathways and relevance**	Four named tracts, then the psychiatric use

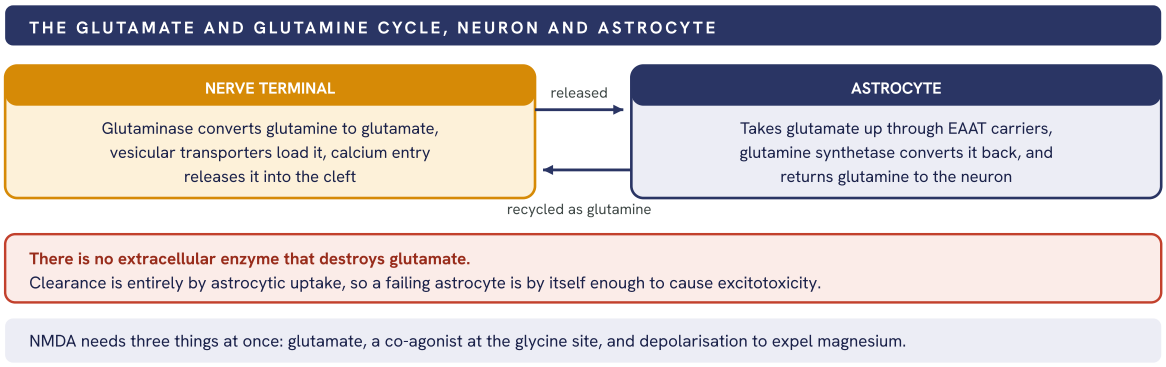


Fig 006.1 Synthesis and clearance as one loop between neuron and astrocyte, with the two facts that follow: no enzymatic breakdown, and a receptor that fires only on coincidence.

RECEPTOR	TYPE	WHAT IT DOES	DRUG THAT ACTS HERE
AMPA	Ionotropic, sodium	Fast excitation, moment to moment	Perampanel in epilepsy
NMDA	Ionotropic, calcium	Coincidence detection, plasticity	**Ketamine, memantine, alcohol**
Kainate	Ionotropic	Modulates release, some fast excitation	Topiramate in part
mGluR groups I to III	Metabotropic, G protein	Tunes gain rather than carrying the signal	Investigational only

- **Major pathways.** Corticocortical association fibres, the corticostriatal projection, reciprocal corticothalamic and thalamocortical fibres, and inside the temporal lobe the perforant path and Schaffer collaterals.
- **Why psychiatry cares.** Ketamine and phencyclidine block the NMDA receptor and reproduce positive, negative and cognitive symptoms in healthy volunteers, the strongest argument for the glutamate model of schizophrenia. The same receptor carries ketamine's rapid antidepressant effect.

WHAT EARNS THE MARKS

- **The astrocyte named as the only clearance route.** It is the fact the whole excitotoxicity story rests on.
- **Ionotropic and metabotropic separated,** with all three ionotropic receptors named.
- **The three NMDA requirements listed together.** Glutamate, co-agonist, depolarisation.
- **Named pathways, not just named receptors,** since the question asks for both.

SEE ALSO Slot I - 04 nitric oxide Slot I - 13 synaptic plasticity

I - 07

Describe the biosynthesis, transmission and implications in psychiatric diagnosis of endocannabinoids. [2+3+5]

2 + 3 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Biosynthesis	Two ligands, made on demand from membrane lipid
3 Transmission	Backwards across the synapse, then broken down locally
5 Psychiatric implications	Cannabis and psychosis first, then the honest limits

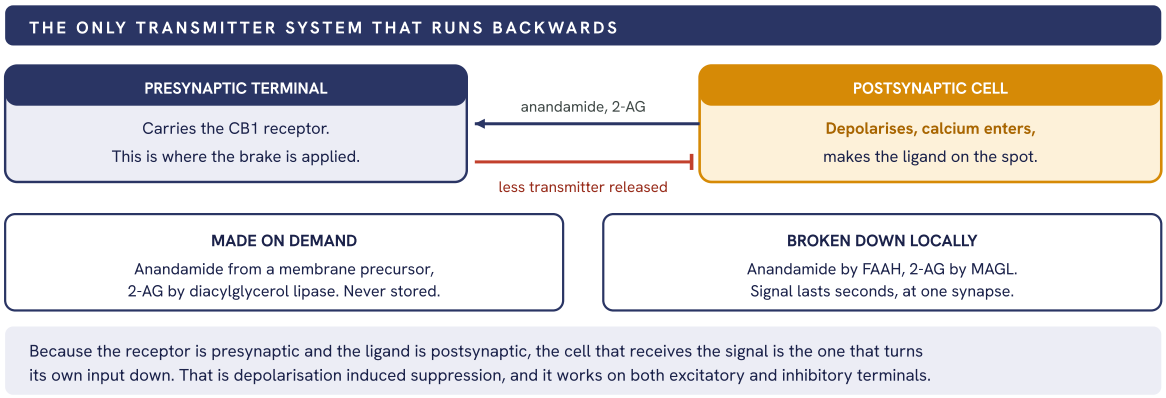


Fig 007.1 The endocannabinoid synapse: ligand made postsynaptically on demand, receptor sitting presynaptically, and a brake applied by the receiving cell onto its own input.

- **The two ligands and the two receptors.** Anandamide and 2-arachidonoylglycerol are the ligands, both derived from arachidonic acid in membrane phospholipid. CB1 is the brain receptor, dense in hippocampus, basal ganglia, cerebellum and cortex and sparse in the brainstem, which is why cannabis is not respiratory depressant. CB2 sits mainly on immune cells and microglia.
- **Cannabis and psychosis.** Tetrahydrocannabinol is a partial CB1 agonist and reproduces positive and cognitive symptoms in volunteers. Prospective cohorts show a dose dependent rise in later psychosis, larger for early and for high potency use. Cannabidiol is not psychotomimetic and has been studied as an antipsychotic.
- **Where the evidence stops.** No endocannabinoid measure is a diagnostic test. Raised anandamide in cerebrospinal fluid and altered CB1 binding are reported in schizophrenia but are not specific. Causation in the cannabis association is supported by dose response and by genetic studies, but reverse causation and confounding are not fully excluded.

WHAT EARNS THE MARKS

- **Retrograde signalling stated as the defining property,** with the receptor placed presynaptically.
- **Made on demand, not stored,** and each ligand paired with the enzyme that ends it.
- **The sparse brainstem CB1 density explained,** since it is why overdose does not kill.
- **Diagnosis answered honestly.** No test exists, and saying so with the reason earns the top of this band.

SEE ALSO Slot I - 06 glutamate receptors Slot I - 08 biological markers

I - 08

Discuss the biological markers of any two major psychiatric illnesses. [5+5]

5 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Define and classify	State against trait, and the four properties a marker needs
3	Schizophrenia	Electrophysiological, imaging, ocular, biochemical
3	Depression	Endocrine, sleep, inflammatory
2	The verdict	Why none is yet a diagnostic test

DEFINE IT BEFORE YOU LIST ANYTHING

A biological marker is an objectively measured biological characteristic that indicates a normal process, a disease process, or a response to treatment. A state marker changes with the episode; a trait marker persists in remission and in unaffected relatives, which is what makes it an endophenotype.

DOMAIN	SCHIZOPHRENIA	DEPRESSION	STATE OR TRAIT
Electrophysiology	P50 gating failure, reduced P300 and mismatch negativity	Shortened REM latency, increased REM density	**Mostly trait**
Endocrine	Blunted growth hormone response to dopamine agonists	Dexamethasone non-suppression, raised cortisol	State
Imaging	Enlarged ventricles, reduced temporal grey matter	Reduced hippocampal volume with recurrence	**Trait for the first, cumulative for the second**
Ocular	Smooth pursuit eye movement abnormality	Not established	**Trait, present in relatives**
Inflammatory	Raised inflammatory markers in a subgroup	Raised interleukin 6 and C reactive protein	State
Neurotrophic	Reduced BDNF reported inconsistently	Low serum BDNF that rises with treatment	State

Amber cells are the trait markers. They are the ones that survive recovery and appear in well relatives, which is what makes them useful for genetics rather than for diagnosis.

- **The four properties a marker must have.** Sensitivity, specificity, reliability across labs, and independence from treatment. Almost every marker above fails at least one, most often specificity.
- **The verdict.** No biological marker is currently diagnostic in psychiatry. They separate groups, not individuals, and the distributions of ill and well people overlap. The working uses today are research stratification, endophenotypes for genetics, and monitoring rather than diagnosis.

WHAT EARNS THE MARKS

- **Two illnesses chosen and named at the top,** since the question allows a choice and expects you to make it.
- **State separated from trait,** applied to each marker rather than stated once.
- **Markers grouped by domain,** which keeps ten items readable as two answers.
- **The honest closing verdict.** Claiming a diagnostic test loses the last marks.

SEE ALSO Slot I - 05 electrophysiological abnormalities Slot I - 99 personalised medicine

I - 09

What is polysomnography? Briefly describe various stages of sleep along with their EEG findings. Discuss the neurobiology of sleep and wakefulness. [2+3+5]

2 + 3 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Polysomnography	The three channels that stage sleep, then the rest
3 The stages	Each with its signature waveform and its share of the night
5 Neurobiology	The switch, the two drives, and the REM mechanism

STAGE	EEG SIGNATURE	EYES AND MUSCLE TONE	SHARE OF THE NIGHT
N1	Posterior alpha of wake drops out, low voltage mixed frequency, vertex sharp waves	Slow rolling eye movements	Roughly a twentieth
N2	Sleep spindles and K complexes	Eyes still, tone reduced	About half
N3	Delta, under 4 hertz, high amplitude	Eyes still, tone low	About a fifth, front loaded
REM	Low voltage mixed frequency, saw-tooth waves	Rapid eye movements with atonia	About a fifth, back loaded

- **What polysomnography is.** A multi-channel overnight recording. Three channels stage it: EEG, electro-oculogram, chin electromyogram; the rest measure breathing, oxygen, heart, legs and position.
- **Neurobiology.** Wake is held by the ascending arousal system: noradrenaline, serotonin, histamine, dopamine, acetylcholine. Sleep is imposed by the ventrolateral preoptic nucleus using GABA and galanin. The two inhibit each other; orexin stabilises wake, and adenosine with the suprachiasmatic clock sets timing.
- **REM, and what shortens its latency.** Pontine cholinergic neurons generate REM while aminergic firing falls silent, giving dreams with atonia. Short REM latency, increased REM density and reduced slow wave sleep is the classic depression profile. Sleep onset REM points to narcolepsy.



Fig 009.1 The architecture of one night, with the parasomnias placed in the half of the night whose sleep stage produces them.

WHAT EARNS THE MARKS

- **Three staging channels named,** EEG, eye movement and chin tone, before the rest of the montage.
- **Spindles and K complexes tied to N2,** delta to N3, sawtooth to REM.
- **Both drives named,** homeostatic adenosine and the circadian clock, not sleep pressure alone.

SEE ALSO Slot I - 03 neurobiology of sleep and wakefulness Slot I - 11 classification of sleep disorders SPRINT Day 1, Functional Neuroanatomy and Neurophysiology

I - 10

What are the various type of the memory? Describe neurobiology of the memory. Describe rehabilitation of memory problems in chronic mental illness. [2+3+5]

2 + 3 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The classification	Name the axis, then divide: awareness first, duration second
3 Neurobiology	Anatomy per branch, then the shared cellular mechanism
5 Rehabilitation	Restore, compensate, adapt, and what the evidence supports

BRANCH	SUBTYPE	WHAT IT HOLDS	ANATOMY
Declarative	Episodic	Events, with their time and place	**Hippocampus, medial temporal**
Declarative	Semantic	Facts, stripped of context	Anterolateral temporal cortex
Non-declarative	Procedural	Skills and habits	**Striatum, cerebellum**
Non-declarative	Priming	Easier processing after exposure	Neocortex
Non-declarative	Conditioning	Learned associations, including fear	Amygdala, cerebellum
By duration	Sensory, short term, working, long term	The second axis, cutting across the first	Prefrontal for working memory

The axis is awareness. Duration is a second, independent axis, so working memory is not a stage on the way to long term memory.

- **The shared mechanism.** Whatever the branch, the cellular event is the same: long term potentiation, made permanent by new protein synthesis and structural change at the spine.
- **Rehabilitation, three strategies.** Restorative training of the impaired function; compensatory training that routes around it; and environmental adaptation that removes the demand. In chronic mental illness the compensatory and adaptive routes carry most of the benefit.

WHAT THE EVIDENCE SUPPORTS IN CHRONIC ILLNESS

Errorless learning and spaced retrieval, leaning on intact procedural memory. External aids: alarms, pill boxes, written routines. Cognitive remediation improves cognition in schizophrenia. Treat the reversible contributors first: sedative load, anticholinergic burden, depression, poor sleep, thyroid and vitamin B12.

WHAT EARNS THE MARKS

- **The axis of division stated before the tree.** A list with no axis reads as recall.
- **Duration named as a second, independent axis,** not a deeper level of the first.
- **Errorless learning justified by the anatomy:** intact procedural carrying damaged declarative.
- **Reversible contributors named first.** Anticholinergic burden is most often missed.

SEE ALSO Slot I - 01 neurobiology of memory Slot I - 02 memory impairment in psychiatric illness **SPRINT** Day 1, Functional Neuroanatomy and Neurophysiology

I - 11

Classify sleep disorders. How would you manage a case of restless leg syndrome? How do you manage benzodiazepine dependence in a chronically mentally ill person? [2+4+4]

2 + 4 + 4 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Classification**	Seven categories, named, with one example each
4	**Restless legs**	Confirm, correct the cause, then treat in order
4	**Benzodiazepine taper**	Assess, switch, taper slowly, support throughout

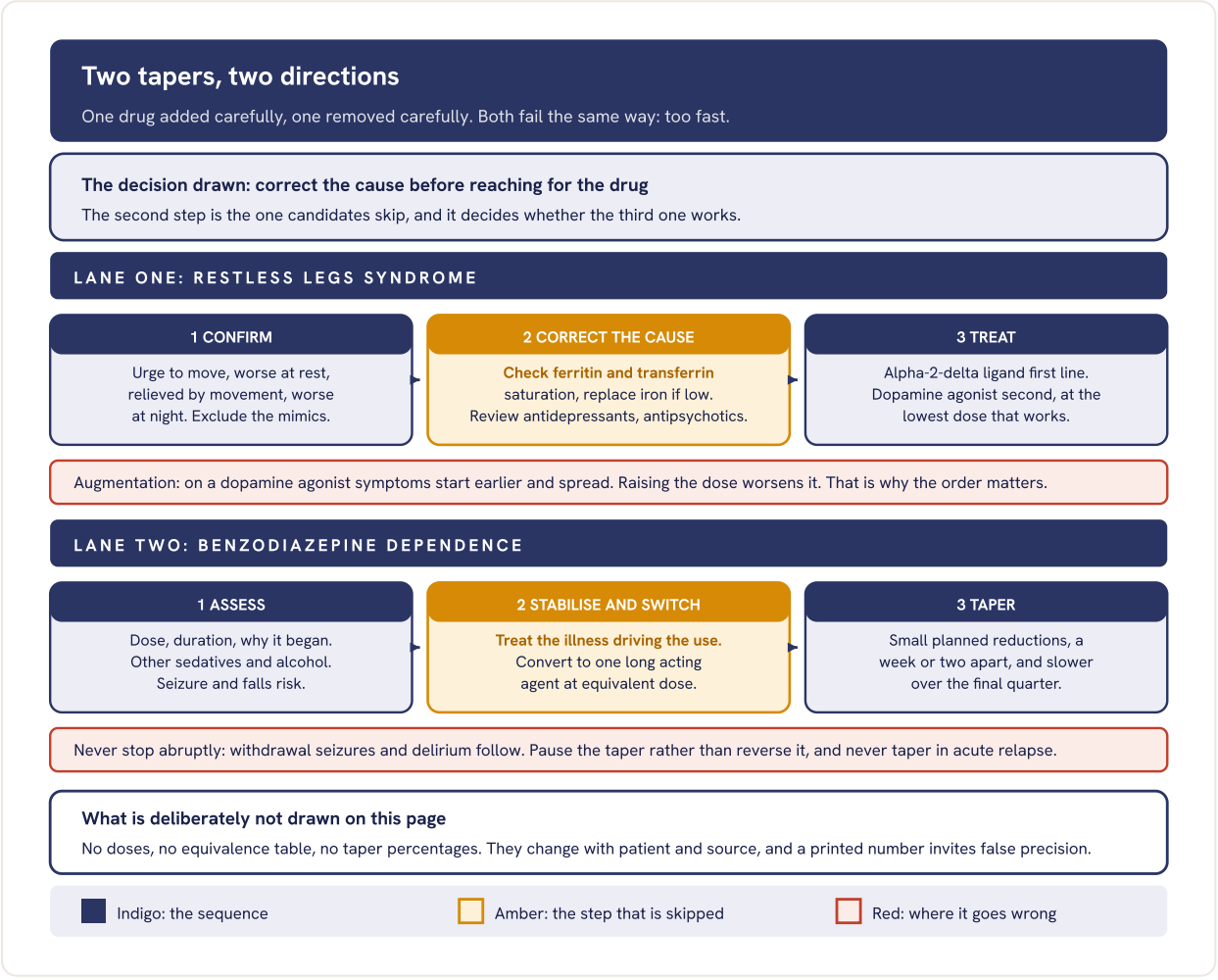


Fig 011.1 Both managements as three steps of the same shape, each with its failure mode below.

CATEGORY	ONE EXAMPLE EACH
Insomnia disorders	Chronic insomnia disorder
Sleep related breathing disorders	Obstructive sleep apnoea
Central disorders of hypersomnolence	Narcolepsy
Circadian rhythm sleep-wake disorders	Delayed sleep-wake phase disorder
Parasomnias	Sleepwalking, REM sleep behaviour disorder
Sleep related movement disorders	**Restless legs syndrome**

Six of the seven categories; the seventh is a residual other. Filing restless legs under movement disorders, not insomnia, is the classification mark.

- **The one that catches people out.** Antipsychotics and antidepressants can cause or worsen restless legs, and akathisia is the differential. Akathisia is generalised inner restlessness at any hour; restless legs is in the legs, at rest, in the evening, and eased by walking.

WHAT EARNS THE MARKS

- **Named categories,** with restless legs filed under movement disorders.
- **Iron studies before any drug,** and augmentation as the reason for the order.
- **The illness treated and a long acting agent substituted before tapering.**
- **Akathisia offered as the differential.** It is this question's discriminator.

SEE ALSO Slot I - 03 neurobiology of sleep Slot I - 09 polysomnography **SPRINT** Day 17, Eating and sleep-wake disorders

I - 12

Name various dopamine pathways of brain. Describe their significance in Psychiatry in detail. [3+7]

3 + 7 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB, NTRUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	The four pathways	Origin and destination for each, named precisely
3	The blockade problem	One drug, four pathways, four different consequences
2	Receptors and synthesis	D1 and D2 families, and where the transmitter comes from
2	Beyond blockade	Salience, addiction, and partial agonism

PATHWAY	FROM, TO	WHAT IT NORMALLY DOES	WHAT BLOCKADE PRODUCES
Mesolimbic	Ventral tegmental area to nucleus accumbens	Assigns salience and reward value	**Positive symptoms settle; this is the therapeutic pathway**
Mesocortical	Ventral tegmental area to prefrontal cortex	Working memory, motivation, drive	**Negative and cognitive symptoms worsen**
Nigrostriatal	Substantia nigra pars compacta to dorsal striatum	Selects and releases movement	**Parkinsonism, dystonia, akathisia, tardive dyskinesia**
Tuberoinfundibular	Arcuate nucleus to anterior pituitary	Tonically inhibits prolactin release	**Hyperprolactinaemia, galactorrhoea, amenorrhoea, sexual dysfunction**

Every amber cell is produced by the same molecule at the same receptor. An antipsychotic cannot choose its pathway, which is why the side effect profile is predictable from this table alone.

- **Synthesis and receptors.** Tyrosine to DOPA by tyrosine hydroxylase, the rate limiting step, then to dopamine by DOPA decarboxylase. Receptors fall into a D1 family that stimulates adenylyl cyclase and a D2 family that inhibits it. Antipsychotic potency tracks D2 affinity.
- **Salience, not pleasure.** Mesolimbic dopamine marks what matters rather than what feels good. Aberrant salience gives ordinary events undue significance, which is a mechanism for how delusions form, and explains why blockade dampens conviction before it removes the belief.
- **Where the simple model fails.** Blockade is immediate but the antipsychotic response takes weeks, so receptor occupancy cannot be the whole story. Clozapine works best with relatively low D2 occupancy, and partial agonists such as aripiprazole stabilise rather than block, which is why they raise prolactin less.

WHAT EARNS THE MARKS

- **Four pathways with both ends named.** Mesolimbic and mesocortical share an origin, and saying so shows the pathways are understood.
- **One drug, four consequences,** laid out as a single table rather than as a side effect list.
- **Salience rather than pleasure** as the account of mesolimbic function.
- **The delay problem raised honestly.** It is the strongest evidence that blockade alone is insufficient.

SEE ALSO Slot I - 21 basal ganglia pathways Slot I - 97 pineal gland and prolactin rhythm

I - 13

Define synaptic plasticity. How is it important in psychiatry? [2+8]

2 + 8 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Definition and forms**	One sentence, then short term against long term, functional against structural
3 **The mechanism**	Induction, expression, and what makes it last
3 **Disorders**	Where plasticity is excessive, deficient, or misdirected
2 **Treatments**	Every effective treatment as a plasticity intervention

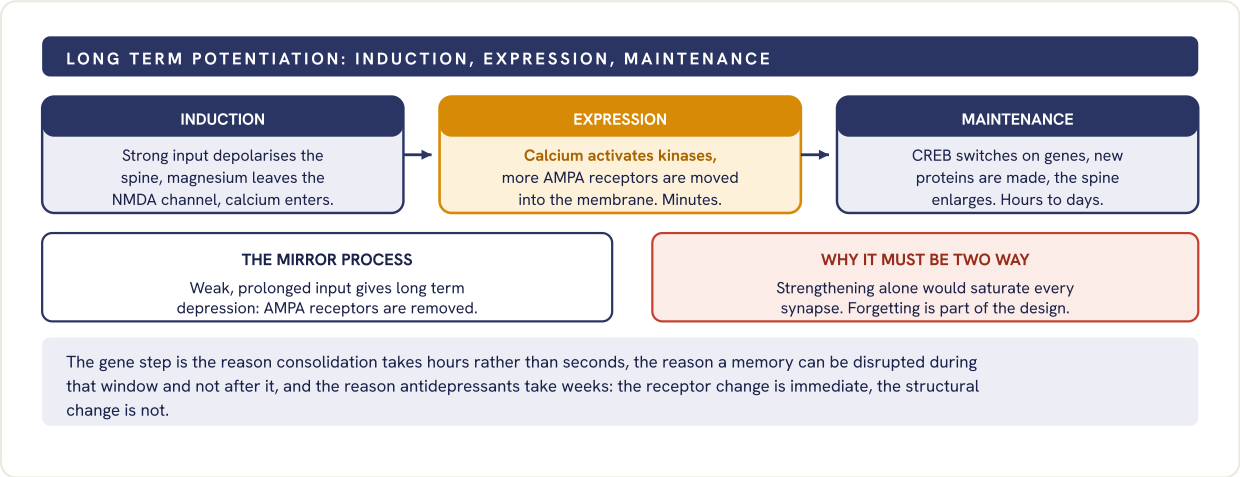


Fig 013.1 Potentiation in three stages with its mirror process alongside, and the gene step marked as the source of every clinically important delay.

- **The definition.** The capacity of a synapse to change the strength of its transmission in response to activity. It is functional when receptor number or release probability changes, and structural when spines and connections are added or lost.
- **Where it goes wrong.** Excessive synaptic pruning in adolescence, with complement C4 implicated, in schizophrenia. Stress and glucocorticoids retract dendrites in hippocampus and prefrontal cortex while the amygdala grows, in depression. Misdirected strengthening of reward circuits in addiction, and of fear circuits in post-traumatic stress.
- **Every treatment is a plasticity treatment.** Antidepressants raise BDNF signalling and restore spine density over weeks. Ketamine produces rapid synaptogenesis through mTOR. Electroconvulsive therapy and repetitive transcranial stimulation induce plasticity directly. Psychotherapy and exposure work by extinction learning, which is plasticity in prefrontal to amygdala pathways.

WHAT EARNS THE MARKS

- **A one sentence definition first,** since only two marks are on offer for it.
- **Potentiation and depression given as a pair.** Naming only strengthening misses why the system works.
- **The gene step used to explain a clinical delay.** That single link is the top band on this question.
- **Psychotherapy included among plasticity treatments,** not only the drugs.

SEE ALSO Slot I - 01 neurobiology of memory Slot I - 06 glutamate receptors

AREA 02 OF 12 · P1-A01

Functional neuroanatomy and neuroimaging

12	13.5%	I-14	I-25
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 5 · RECURRENT 1 · SINGLE 6

REVISION ORDER

The limbic group first and as one block: the Papez circuit, the limbic system, the amygdala and the two hippocampal slots overlap almost completely. The cortical group next, prefrontal, frontal, cerebral cortex and occipital, and the frontal slot is the long one. The caudate nucleus is asked twice and is one answer at two widths. Thalamus, corpus callosum and basal ganglia last, and all three are short.

WHAT IS IN THIS AREA

Twelve structures, close to one each: the corpus callosum, the Papez circuit with the caudate nucleus, the prefrontal cortex, the amygdala, the cerebral cortex with its cellular organisation and circuitry, the thalamus, the limbic system, the basal ganglia with the hippocampus, the frontal lobe with three injury syndromes and parietal atrophy, the hippocampal formation with its subfield volumes, the caudate nucleus on its own, and the occipital cortex. The registered name of this area also carries neuroimaging. Imaging is a limb of one slot here rather than the subject of any, and the area is described by what its slots actually ask.

I - 14

Discuss the role of Corpus callosum. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **What it is**	The largest commissure, and its five named segments
3 **Fibre topography**	Which cortex each segment joins, front to back
3 **What it does**	Transfer, lateralisation, bimanual and interhemispheric control
2 **When it fails**	Agenesis, callosotomy, and the disconnection signs

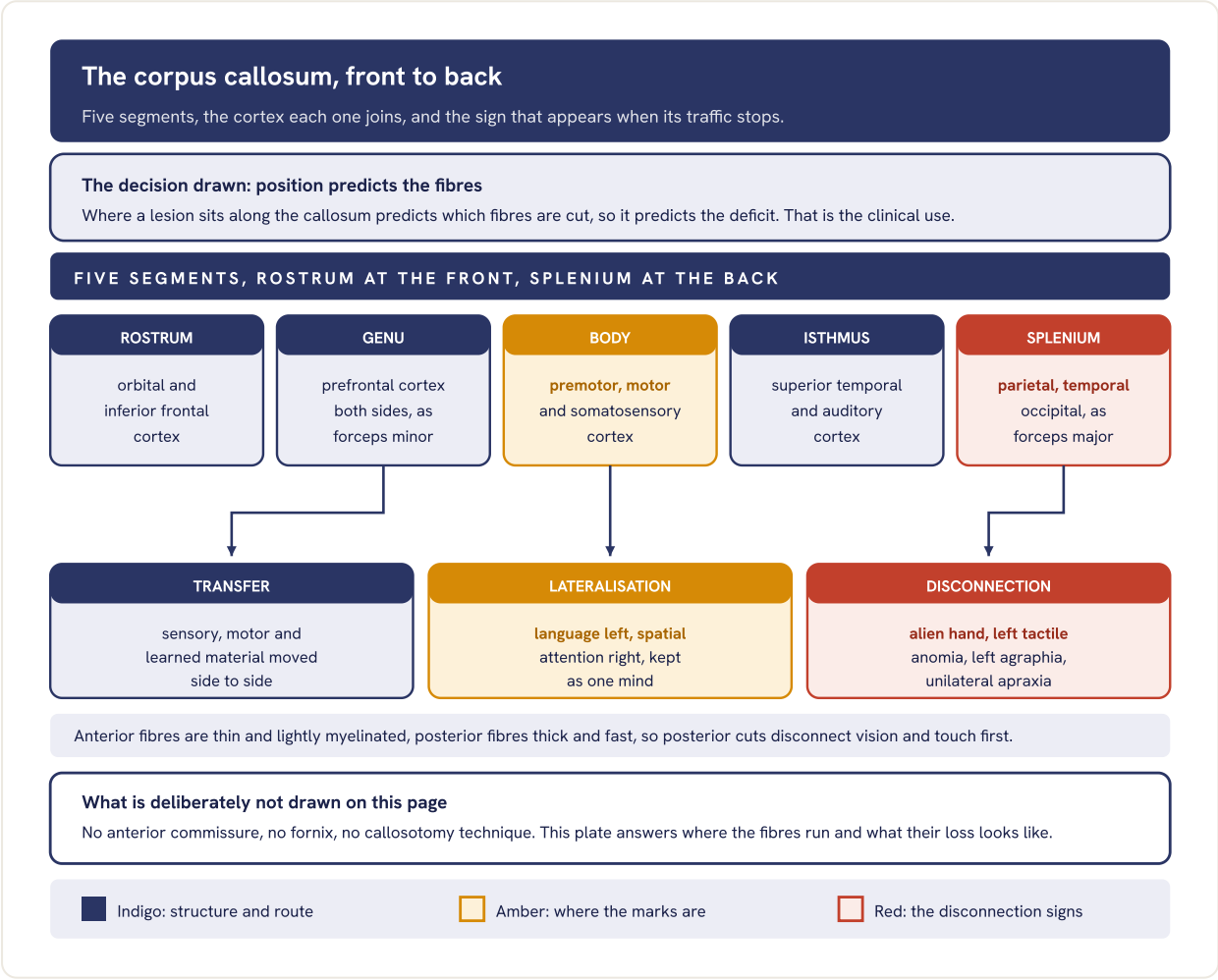


Fig 014.1 The callosum drawn as five segments with the cortex each joins, and the three consequences that follow from the traffic they carry.

THE DEFINITION

The largest white matter commissure of the brain, a sheet of myelinated axons joining homologous and heterologous cortical areas of the two hemispheres. It is topographically ordered: rostrum and genu in front carry frontal fibres, the splenium behind carries parieto-occipital fibres.

- **Transfer.** Sensory, motor and learned information crosses here, which is why a split-brain patient cannot name an object felt only by the left hand.

- **Integration with lateralisation.** Language sits left and spatial attention right; the callosum lets specialised hemispheres act as one, and coordinates bimanual movement.
- **Callosal syndromes.** Agenesis (often with other midline defects), surgical callosotomy for intractable epilepsy, and Marchiafava-Bignami disease from chronic alcohol use.

WHAT EARNS THE MARKS

- **The five segments named in order.** Rostrum, genu, body, isthmus, splenium, each with the cortex it joins.
- **Topography stated as a rule** before any sign is listed: front carries frontal, back carries posterior sensory.
- **Disconnection signs tied to a segment.** Left tactile anomia and left agraphia belong to anterior fibres, hemialexia to the splenium.
- **The three failure states,** agenesis, callosotomy and Marchiafava-Bignami, named as such.

SEE ALSO Slot I - 16 prefrontal cortex Slot I - 22 frontal and parietal lobe syndromes **SPRINT** Day 1, Functional Neuroanatomy and Neurophysiology

I - 15

Neuroanatomy of Papez circuit and a brief mention about the caudate nucleus. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	**The proposal**	Papez 1937, and what the loop was meant to explain
4	**The loop in order**	Six stations, hippocampus back to hippocampus
2	**What it explains**	Diencephalic amnesia sits on this ring
2	**Caudate, briefly**	Position, loop membership, two clinical uses

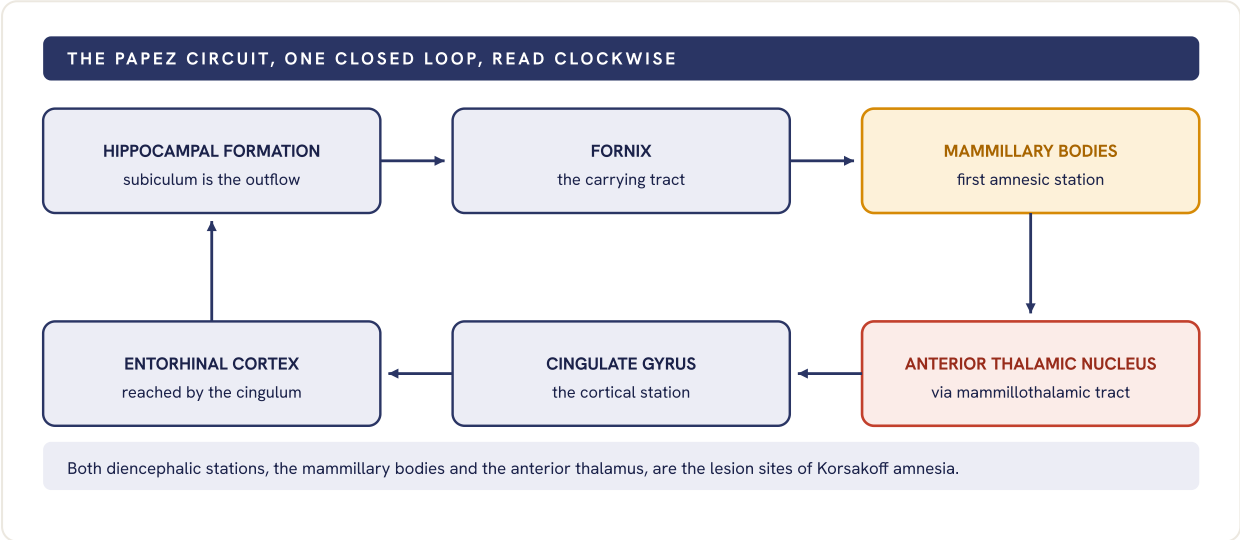


Fig 015.1 The circuit as a closed ring, with the two diencephalic stations that carry the amnesia marked out from the four that do not.

- **What Papez proposed.** In 1937 a cortical to hypothalamic loop offered as the substrate of emotional experience. MacLean later widened it into the limbic system.
- **Why the anatomy outlived the claim.** It is now taught as an episodic memory circuit: thiamine deficient Korsakoff amnesia, fornix section and anterior thalamic infarcts all sit on this ring.
- **Caudate nucleus.** A C-shaped mass with head, body and tail curving in the lateral ventricle wall, continuous with the putamen and forming the associative striatum.
- **Caudate, clinically.** Head atrophy with boxcar ventricles in Huntington disease, hypermetabolism in obsessive compulsive disorder that settles as the illness responds.

WHAT EARNS THE MARKS

- **All six stations, in order, with the two tracts named.** Fornix and mammillothalamic tract are the links most answers skip.
- **The circuit tied to amnesia, not only to emotion.** Saying where Korsakoff sits is the line that separates the bands.
- **One honest limit.** Emotion is not made by one ring; the amygdala and prefrontal circuits lie outside Papez.

SEE ALSO Slot I - 20 limbic system Slot I - 24 caudate nucleus SPRINT

Day 1, Functional Neuroanatomy and Neurophysiology

I - 16

Prefrontal cortex. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, MUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What defines it	Granular cortex in front of premotor, defined by its thalamic input
4	The three subdivisions	Each with its job and the syndrome its loss makes
2	Connections	Loops with striatum, thalamus, amygdala and hippocampus
2	Psychiatric relevance	Late maturation, hypofrontality, and what is testable

THE DEFINITION

The granular association cortex of the frontal lobe lying anterior to the premotor areas. It is defined anatomically by its reciprocal connection with the mediodorsal nucleus of the thalamus, and functionally as the seat of executive control: holding a goal, selecting an action, and suppressing the rest.

SUBDIVISION	WHERE IT SITS	WHAT IT DOES	WHAT ITS LOSS LOOKS LIKE
Dorsolateral	Middle frontal gyrus, lateral surface	Working memory, planning, set shifting, abstraction	Dysexecutive: perseveration, concrete thinking, poor planning
Orbitofrontal	Above the orbit, with gyrus rectus	Reward valuation, impulse control, social judgement	Disinhibited: tactless, impulsive, socially coarse
Ventromedial and anterior cingulate	Medial wall	Motivation, drive, error monitoring, autonomic tone	Apathetic: abulia, and akinetic mutism when severe

Three syndromes, not one frontal syndrome. Naming which subdivision produced the picture is what the examiner is checking.

- **Connections.** Five parallel cortico-striato-thalamo-cortical loops run through the caudate and thalamus, with heavy dopaminergic input from the ventral tegmental area and direct links to amygdala and hippocampus.
- **Development.** It myelinates last, into the third decade, which is the anatomical reason adolescent judgement and impulse control mature after appetite and emotion do.
- **Psychiatric relevance.** Reduced dorsolateral activation on working memory tasks in schizophrenia, subgenual cingulate overactivity in depression, and orbitofrontal loop overactivity in obsessive compulsive disorder.

WHAT EARNS THE MARKS

- **The mediodorsal thalamic connection as the definition.** It is what makes prefrontal cortex a region and not a direction.
- **Three syndromes, each tied to its subdivision.** A single undifferentiated frontal syndrome is a mid-band answer.
- **One bedside test per subdivision.** Verbal fluency and Wisconsin sorting for dorsolateral, go and no-go for orbitofrontal.

SEE ALSO Slot I - 22 frontal lobe syndromes Slot I - 19 thalamic nuclei **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 17

Write briefly about the functional neuroanatomy of amygdala. Discuss the psychiatric implications related with amygdala. [5+5]

5 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB, MUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Position and nuclei	Three nuclear groups, and which one is the gate
3	Input and output	Two roads in, four autonomic routes out
3	Disorders	Anxiety, trauma, mood, psychosis, and the direction of change
2	What treatment does	Prefrontal control as the mechanism of extinction

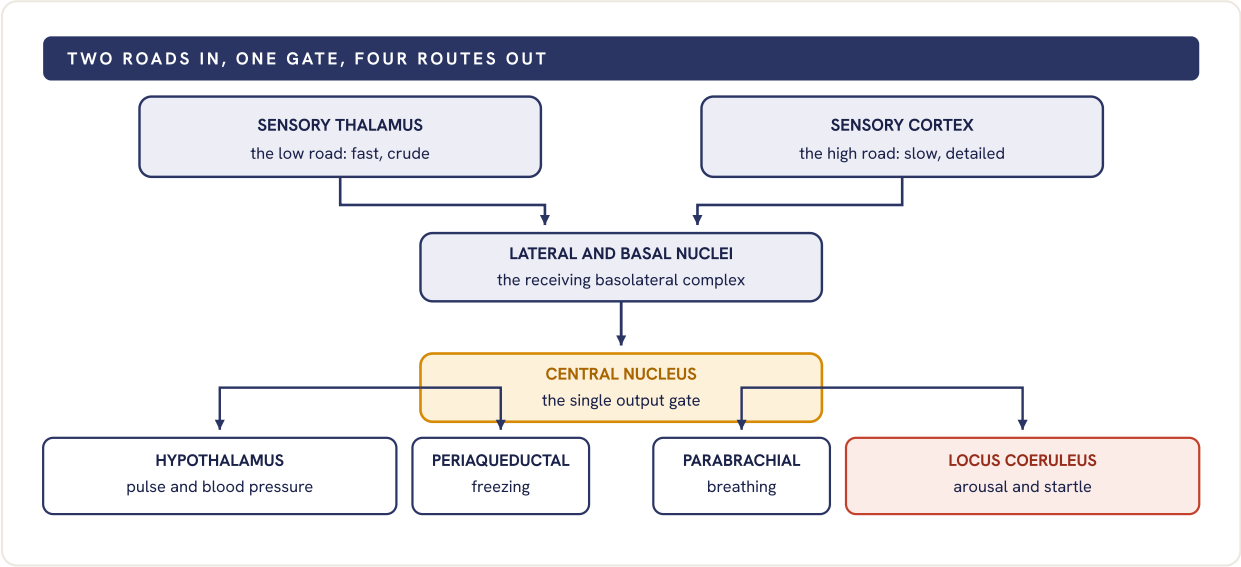


Fig 017.1 The fear pathway drawn as two afferent roads converging on the basolateral complex, one central output gate, and the four brainstem and hypothalamic routes that produce the bodily signs.

- **Position and nuclei.** An almond of grey matter in the medial temporal lobe, in front of the hippocampal head. Three groups: basolateral (input), centromedial (output), and cortical with the olfactory link.
- **What it computes.** Assigns emotional value to a stimulus, then drives the body to match. Fear conditioning is learned here, and the prefrontal cortex learns extinction by inhibiting it.
- **Across disorders.** Overactive to threat and to fearful faces in anxiety and post-traumatic stress with weak prefrontal restraint. Overactive to negative faces in depression. Reduced volume and abnormal face processing in schizophrenia and in autism.
- **The lesion picture.** Bilateral loss gives Kluver-Bucy syndrome, with placidity, hyperorality and loss of fear, and abolishes the startle potentiation that fear normally produces.

WHAT EARNS THE MARKS

- **The three nuclear groups, with their traffic direction.**
- **Two roads in and named routes out.** Each output tied to the sign it produces earns more than a list of nuclei.
- **The direction of change per disorder,** up or down, not simply involvement.

SEE ALSO Slot I - 20 limbic system in psychiatric disorders SPRINT

Day 1, Functional Neuroanatomy and Neurophysiology

I - 18

Describe the neuroanatomy and cellular organization of cerebral cortex. Describe its functional circuitry with a suitable diagram. [3+3+4]

3 + 3 + 4 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Gross organisation	Lobes, allocortex against neocortex, Brodmann mapping
3 Cellular organisation	Six layers, two cell classes, the column as the unit
4 Functional circuitry	The diagram: what enters at four, what leaves at five and six

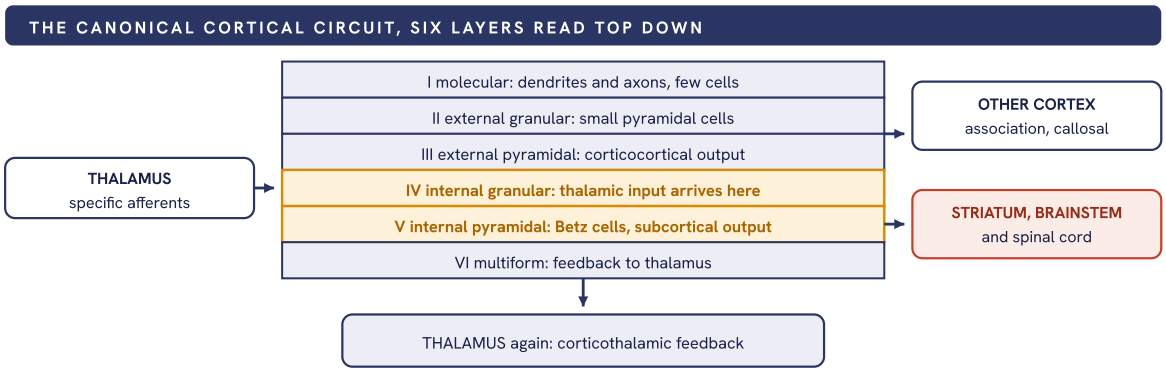


Fig 018.1 The six laminae with the traffic each carries: input at layer four, corticocortical output from layers two and three, subcortical output from layer five, thalamic feedback from layer six.

- **Gross organisation.** Neocortex is six layered and covers most of the surface; allocortex, meaning the hippocampal and olfactory cortex, has three layers. Brodmann divided the surface by cytoarchitecture, and those numbers are still the working map.
- **Two cell classes.** Pyramidal cells are excitatory, glutamatergic and projecting; interneurons are inhibitory, GABAergic and local. Parvalbumin interneurons pace gamma rhythm, and their loss is a leading account of cognitive symptoms in schizophrenia.

UNIT	WHAT IT IS	WHY IT MATTERS
Minicolumn	A vertical chain of about a hundred cells sharing one receptive field	The smallest repeating processing unit
Cortical column	Minicolumns grouped across all six layers	Same circuit everywhere, tuned by its input; a Brodmann number names a set of them

WHAT EARNS THE MARKS

- **Six layers named in order,** with the traffic of each, not simply the list.
- **The diagram, since the question asks for one.** Input at four, output at five, feedback at six is the whole circuit.
- **Granular against agranular cortex** explained by layer thickness rather than asserted.

SEE ALSO Slot I - 19 thalamic nuclei Slot I - 16 prefrontal cortex **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 19

Describe functions of Thalamus. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **What it is**	The gateway to cortex, and the one sense that bypasses it
4 **Nuclei and their traffic**	Grouped by the lamina, each with its cortical target
2 **Beyond relay**	Gating, arousal, and the generation of sleep rhythms
2 **Clinical correlates**	Amnesia, thalamic pain, and the schizophrenia findings

NUCLEUS	MAIN INPUT	PROJECTS TO	WHAT ITS LOSS SHOWS
Anterior	Mammillary bodies, by the mammillothalamic tract	Cingulate gyrus	Amnesia, on the Papez ring
Mediodorsal	Amygdala, olfactory and limbic cortex	Prefrontal cortex	Amnesia, executive loss
Ventral posterior	Medial lemniscus, spinothalamic, trigeminal	Postcentral gyrus	Hemisensory loss, thalamic pain
Geniculate bodies	Optic tract, inferior colliculus	Visual and auditory cortex	Field defect, deafness
Reticular and intralaminar	Cortex and brainstem collaterals	Thalamus itself and diffuse cortex	Impaired arousal and attention

The reticular nucleus is the exception in the table: it is GABAergic, it projects to the thalamus rather than to cortex, and it is the gate the others pass through.

- **More than a relay.** Every sense but olfaction is routed here, and the relay is not passive: the reticular nucleus gates what reaches cortex, which is the anatomy behind selective attention.
- **Rhythm generation.** Thalamocortical loops with the reticular nucleus produce sleep spindles and the slow oscillations of deep sleep, so thalamic damage disturbs sleep architecture as well as sensation.
- **Psychiatric correlates.** Reduced mediodorsal volume and disrupted thalamocortical connectivity are among the more replicated structural findings in schizophrenia, and the pulvinar is implicated in the attention deficits.

THE LINE WORTH WRITING FIRST

The thalamus is the obligatory gateway to the cerebral cortex for all sensory modalities except olfaction, and every cortical area sends a return projection to the thalamic nucleus that supplies it. Relay is reciprocal, never one way.

WHAT EARNS THE MARKS

- **The olfaction exception, stated as an exception.** It is the cheapest mark on the page.
- **Nuclei grouped, each with its cortical target.** A bare list of names without destinations is recall.
- **Gating and rhythm named alongside relay,** which is what turns a list into a function answer.
- **Two clinical anchors,** diencephalic amnesia and thalamic pain, each tied to its nucleus.

SEE ALSO Slot I - 15 Papez circuit Slot I - 18 cortical circuitry **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 20

a) Write about functional neuroanatomy of limbic system. b) Discuss role of limbic system in psychiatric disorders. [5+5]

5 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What the term covers	Broca to MacLean, and why the boundary is disputed
3	The components	Three tiers, cortical, subcortical, diencephalic
3	Disorder by disorder	Which node, which direction of change
2	What follows for treatment	Targets that are actually used in practice

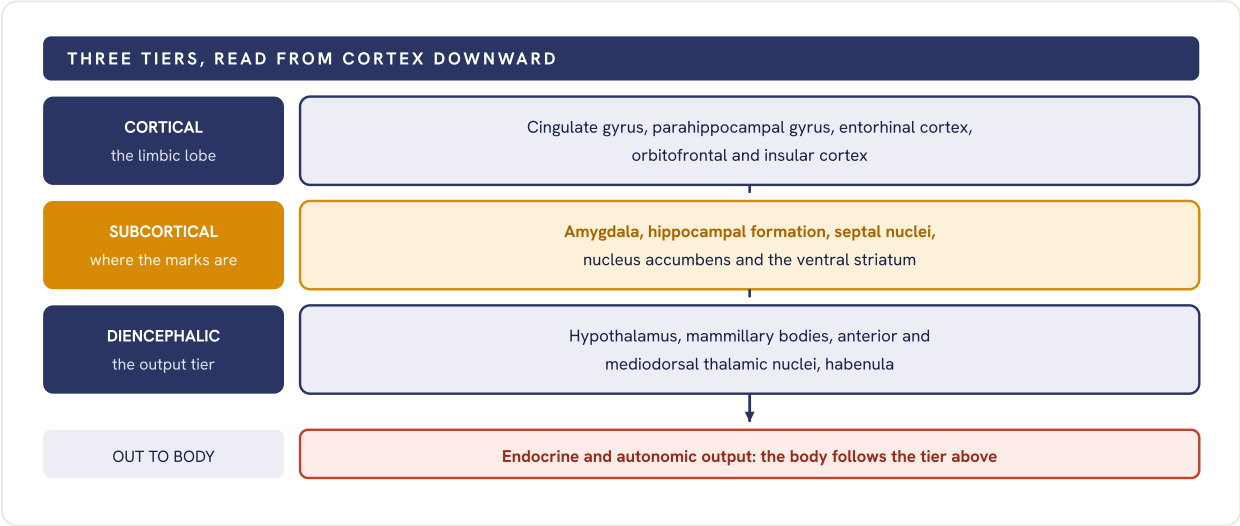


Fig 020.1 The limbic system laid out as three tiers rather than a ring, so that the cortical, subcortical and diencephalic members can be named separately and the output route stays visible.

- **What the term covers.** Broca named the limbic lobe as the ring of cortex around the corpus callosum; Papez gave it a circuit and MacLean gave it a name. The boundary has never been settled, so name your list before you use it.
- **Across disorders, and where they are targeted.** Amygdala overactivity with weak prefrontal restraint in anxiety and post-traumatic stress; smaller hippocampal volume in recurrent depression; subgenual cingulate overactivity in depression, where deep brain stimulation remains investigational; ventral striatal underactivity to reward in anhedonia and negative symptoms. Cingulotomy and capsulotomy are last resort options in severe refractory obsessive compulsive disorder.
- **Do not stop at the emotional brain.** It also carries episodic memory, olfaction, reward and endocrine control, and the triune brain framing MacLean built around it is no longer supported.

WHAT EARNS THE MARKS

- **A defined membership list before any function.** Saying the boundary is disputed reads as command, not hedging.
- **Three tiers rather than one heap,** with the output route to hypothalamus named.
- **Direction of change per disorder.** Amygdala up, hippocampus down, ventral striatum down to reward.

SEE ALSO Slot I - 15 Papez circuit Slot I - 17 amygdala **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 21

Describe the functional neuroanatomy of basal ganglia. Describe different parts of hippocampus. Discuss the psychiatric implications of basal ganglia and hippocampus. [3+2+5]

3 + 2 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Basal ganglia	The nuclei, then the direct and indirect pathways
2 Hippocampal parts	Dentate, the four CA fields, subiculum
5 Implications	Structure by structure, with the direction of the finding

THE ORGANISING IDEA

Both structures work as loops rather than as centres. Cortex projects to the striatum, the striatum acts on the pallidum and thalamus, and the thalamus returns to the same cortical area. The hippocampus runs a comparable loop through entorhinal cortex. Naming the loop is worth more than naming the parts.

- **Basal ganglia, the parts.** Caudate and putamen make the striatum, globus pallidus has an external and an internal segment, with subthalamic nucleus and substantia nigra completing the set.
- **The two pathways.** The direct pathway carries D1 receptors and releases movement; the indirect pathway carries D2 receptors and suppresses it. Antipsychotic blockade of striatal D2 receptors tips the balance toward suppression, which is parkinsonism.
- **Hippocampal parts.** Dentate gyrus, the cornu ammonis fields CA1 to CA4, and the subiculum, together with entorhinal cortex forming the hippocampal formation. CA1 is the field most vulnerable to hypoxia.

STRUCTURE	THE FINDING	WHERE IT IS SEEN	WHAT IT MEANS CLINICALLY
Caudate	Overactivity in the orbitofrontal loop	Obsessive compulsive disorder	Settles as the illness responds
Caudate	Head atrophy, boxcar ventricles	Huntington disease	Chorea with psychiatric onset
Striatum	D2 blockade above the therapeutic range	Antipsychotic treatment	Parkinsonism, akathisia, dystonia
Hippocampus	Reduced volume, most in CA1 and CA3	Recurrent depression, post-traumatic stress	Partly reversible with recovery

WHAT EARNS THE MARKS

- **Direct against indirect pathway, with D1 and D2 attached.** It is the line that explains extrapyramidal side effects.
- **All hippocampal parts named,** dentate, four CA fields and subiculum, with CA1 flagged as the vulnerable one.
- **Findings given a direction,** up or down, and a disorder. Involvement alone earns little.

SEE ALSO Slot I - 23 hippocampal subfields Slot I - 24 caudate nucleus **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 22

Discuss the functions of frontal lobe with a suitable diagram. Write 3 syndromes associated with injury to frontal lobe. Describe signs and symptoms of parietal lobe atrophy. [3+4+3]

3 + 4 + 3 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Frontal functions	The diagram: motor at the back, executive in front
4 Three syndromes	Each named, sited, and given its picture
3 Parietal atrophy	Dominant against non-dominant, then the bilateral case

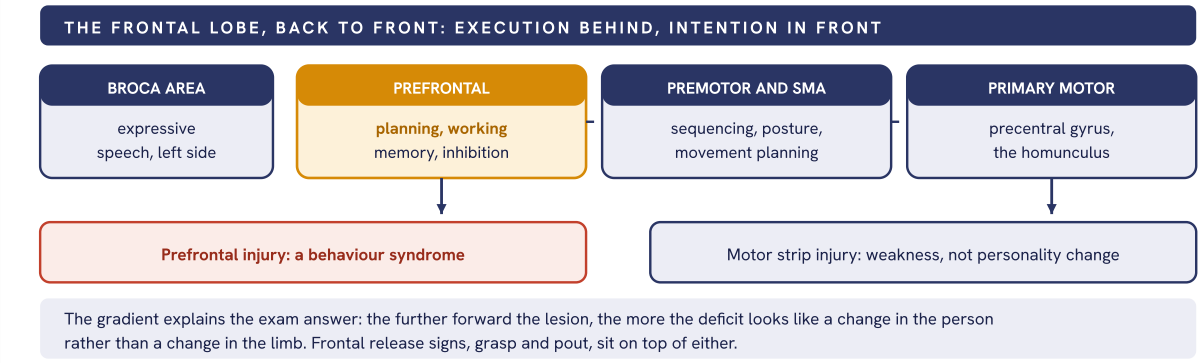


Fig 022.1 The frontal lobe as a back to front gradient, execution at the central sulcus and intention at the pole, with the two kinds of injury picture that follow from it.

SYNDROME	WHERE THE LESION SITS	THE PICTURE
Dysexecutive	Dorsolateral prefrontal	Poor planning and sequencing, perseveration, concrete thinking, reduced verbal fluency
Disinhibited	Orbitofrontal	Tactless, impulsive, jocular, socially coarse, utilisation behaviour, preserved formal testing
Apathetic	Medial frontal and anterior cingulate	Abulia, loss of initiative, sparse speech, akinetic mutism when the lesion is large and bilateral

- **Parietal atrophy, dominant side.** Gerstmann syndrome, meaning agraphia, acalculia, finger agnosia and right to left disorientation, with ideomotor apraxia and receptive language difficulty.
- **Parietal atrophy, non-dominant side.** Contralateral neglect and anosognosia, dressing and constructional apraxia, and loss of topographical sense. Bilateral posterior atrophy gives Balint syndrome: optic ataxia, oculomotor apraxia and simultanagnosia.

WHAT EARNS THE MARKS

- **The diagram, since the question asks for one,** drawn as a back to front gradient rather than a list of areas.
- **Exactly three syndromes, each with its site.** The question counts them, so the answer should too.
- **Parietal signs split by side** before the bilateral case. An unsided list loses the localising mark.

SEE ALSO Slot I - 16 prefrontal cortex Slot I - 25 occipital cortex **SPRINT**

Day 34, Stroke, TBI, delirium and focal brain syndromes

I - 23

What is hippocampal formation? Discuss its role in learning. Describe hippocampal subfield volumes and its use in psychiatry. [2+3+5]

2 + 3 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Dentate, CA1 to CA4, subiculum, entorhinal: a formation, not a nucleus
3 Role in learning	The trisynaptic circuit, long term potentiation, consolidation
5 Subfield volumetry	How it is measured, what it shows, and what it cannot do

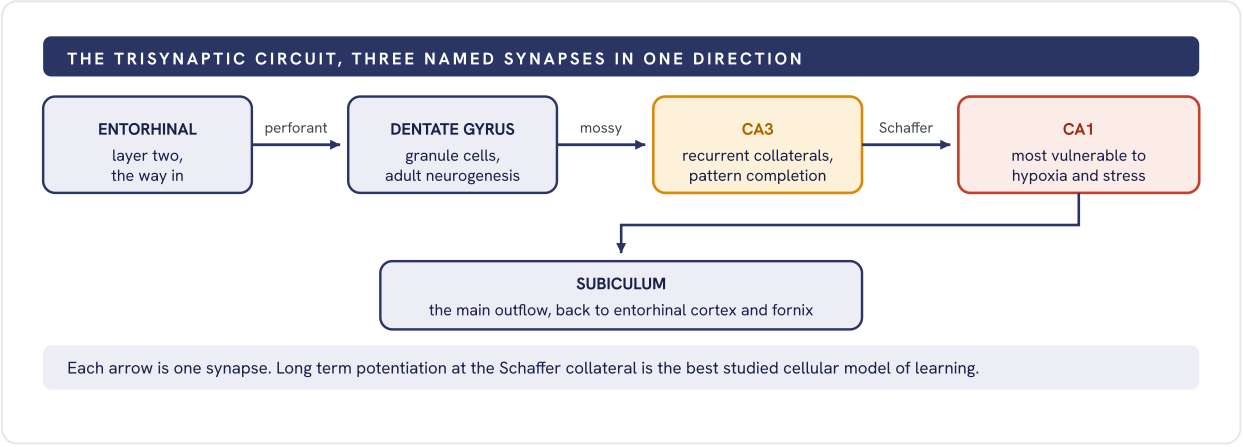


Fig 023.1 The trisynaptic circuit as a one way chain, with the field that completes patterns in amber and the field that dies first in red.

- **Role in learning.** It binds a new episode into a single trace, then hands it to neocortex over months, which is systems consolidation. Damage gives dense anterograde amnesia with graded retrograde loss and intact procedural learning, the pattern first described in the patient H M.

SUBFIELD VOLUMETRY: WHAT IT SHOWS, AND WHAT IT CANNOT

High resolution imaging with automated segmentation gives per field volumes rather than one hippocampal number. CA1 and subiculum loss tracks early Alzheimer disease; CA3 and dentate loss tracks chronic stress and recurrent depression. But values overlap heavily between the ill and the well, and depend on the software and field strength. It does not diagnose an individual, and saying so is a mark.

WHAT EARNS THE MARKS

- **The formation listed as a set,** with entorhinal cortex included.
- **Three synapses named,** perforant path, mossy fibres, Schaffer collaterals, with long term potentiation attached.
- **Subfields tied to specific conditions,** with the limits of the measure stated. An answer that claims diagnostic use has overreached.

SEE ALSO Slot I - 21 basal ganglia and hippocampus Slot I - 13 synaptic plasticity **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 24

Describe Caudate nucleus and its functions. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Anatomy	Shape, parts, relations, and what it is made of
2 Connections	Where its input comes from and where its output goes
4 Functions by loop	Motor, executive, limbic and oculomotor, one line each
2 Clinical correlates	The four conditions that turn on this nucleus

LOOP	CORTEX IT SERVES	WHAT THE LOOP DOES	WHEN IT FAILS
Motor	Motor and premotor cortex	Selects and releases a wanted movement	Chorea, bradykinesia
Executive	Dorsolateral prefrontal cortex	Set shifting, habit learning, sequencing	Perseveration, poor planning
Limbic	Orbitofrontal and anterior cingulate	Weighs reward, stops an unwanted act	Obsessions, compulsions, apathy
Oculomotor	Frontal eye fields	Directs voluntary saccades	Impaired saccade control

One nucleus, four parallel loops. Naming the loop that failed is what turns a movement sign or a psychiatric sign into the same explanation.

- **Anatomy.** A C-shaped grey mass with head, body and tail, curving along the lateral ventricle. The head bulges into the frontal horn; the tail ends near the amygdala. It joins the putamen across the internal capsule through grey bridges, and together they are the striatum.
- **Cells and transmitters.** Mostly GABAergic medium spiny neurons, receiving glutamate from cortex and dopamine from the substantia nigra pars compacta, with local cholinergic interneurons. D1 cells begin the direct pathway, D2 cells the indirect.
- **The four clinical anchors.** Head atrophy with boxcar ventricles in Huntington disease, caudate and orbitofrontal overactivity in obsessive compulsive disorder, caudate involvement in Sydenham chorea, and reduced volume in Tourette syndrome.

PITFALL

Do not treat the caudate as a purely motor structure. Its largest loops serve prefrontal cortex, which is why a caudate answer in a psychiatry paper must reach obsessive compulsive disorder, apathy and habit learning, and not stop at chorea.

WHAT EARNS THE MARKS

- **The C shape with head, body and tail,** and its relation to the lateral ventricle.
- **Medium spiny neurons with their three inputs.** Cortical glutamate, nigral dopamine, local acetylcholine.
- **Four loops, each with its cortex,** which is the structure of the four-mark half.
- **Both a movement disorder and a psychiatric disorder** among the clinical correlates.

SEE ALSO Slot I - 21 basal ganglia Slot I - 16 prefrontal cortex SPRINT

Day 1, Functional Neuroanatomy and Neurophysiology

I - 25

Functions of the occipital cortex. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Anatomy**	Striate and extrastriate cortex, and the retinotopic map
3	**The two streams**	Dorsal where, ventral what, and what each computes
3	**Lesion syndromes**	Field defects, then the agnosias, sited
2	**Psychiatric relevance**	Visual hallucination, and what its shape tells you

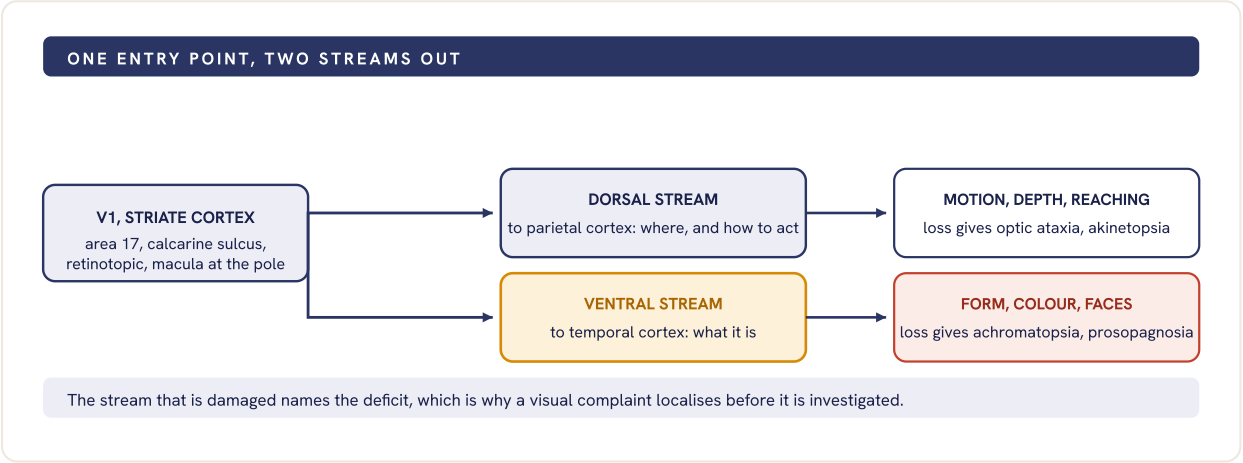


Fig 025.1 Visual cortex as one retinotopic entry point feeding two streams, each with the recognisable deficit that follows when it is cut.

- **Anatomy.** Striate cortex, area 17, lines the calcarine sulcus and is named for the stria of Gennari, an unusually thick layer four. Extrastriate areas 18 and 19 surround it and carry the higher processing.
- **Psychiatric relevance.** Occipital and temporal lesions give formed visual hallucinations with insight preserved. Charles Bonnet syndrome, formed hallucinations in an eye-diseased older person with intact cognition, is the presentation not to mistake for psychosis.

LESION	THE SIGN	WHAT IT TELLS YOU
Unilateral occipital	Homonymous hemianopia with macular sparing	Posterior cerebral territory, dual blood supply at the pole
Bilateral occipital	Cortical blindness, sometimes denied	Anton syndrome, blindness with anosognosia
Left occipital with splenium	Alexia without agraphia	A disconnection, not a language cortex lesion
Ventral occipitotemporal	Prosopagnosia, achromatopsia	The what stream, not the eye

WHAT EARNS THE MARKS

- **Retinotopy stated, with macular sparing explained.** It is the classic clinical consequence of the anatomy.
- **Two streams named and separated,** each with the deficit its loss makes.
- **Charles Bonnet syndrome named,** since this is a psychiatry paper and the differential is the point.

SEE ALSO Slot I - 14 corpus callosum Slot I - 22 parietal lobe signs **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

AREA 03 OF 12 · P1-A07

Personality theory and psychodynamics

10	10.5%	I-26	I-35
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 HIGH 1 SINGLE 8

REVISION ORDER

The two defence-mechanism slots together, they are one answer at two widths, and the Anna Freud slot sits directly on top of them. The three neo-Freudian slots next, where Jung is most of all three. The topographical model and the concept of self after that. Transactional analysis is short and independent. The family-theories slot is the one here that needs reading of its own: it is a critical evaluation, not a description.

WHAT IS IN THIS AREA

Defence mechanisms in two slots, one asking for them and one asking for their classification and their classical use in treatment. The neo-Freudians in three, twice with Carl Jung named inside the question and once with Jung alone. Anna Freud, the topographical model with primary and secondary process thinking, the concept of self, transactional analysis, and the slot that asks whether the family theories of schizophrenia are still relevant.

I - 26

Ego defence mechanisms. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KUHS, NTRUHS, RGUHS - 6 APPEARANCES, 6 OF 78 SESSIONS

SKELETON

2	**Definition**	What the ego defends against, and with whose authority
2	**The organising frame**	Vaillant's four levels, named as levels
4	**The mechanisms**	Each at its own level, one line, one example
2	**The confusable pairs**	The four separations that mark a good answer

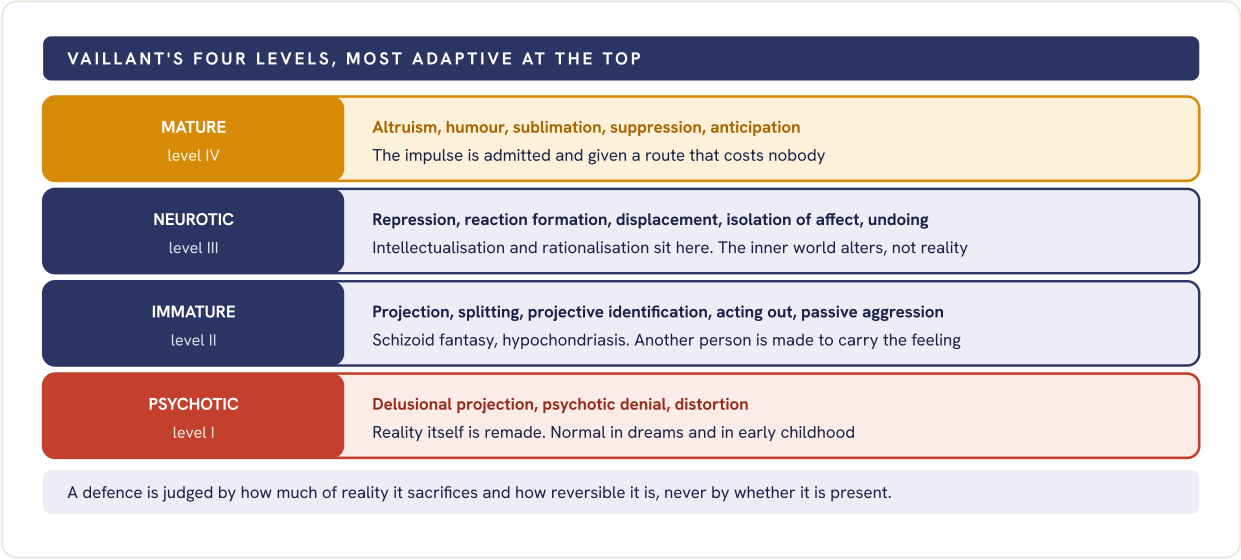


Fig 026.1 The four levels as rungs, the mature rung in amber because it is the one most often left out, the psychotic rung in red because only there does reality testing itself fail.

- **The definition, with its authority.** An unconscious operation of the ego that keeps an unacceptable impulse, affect or idea out of awareness and so holds anxiety down. Anna Freud gave the first systematic account in The Ego and the Mechanisms of Defence, 1936; her father had named repression in 1894 as the prototype from which the rest follow.
- **How they are recognised.** A defence is inferred from what is absent: the affect that fails to arrive with the content, or the theory offered in place of a feeling.
- **The four separations.** Projection puts one's own impulse into another; projective identification also makes the other feel and act it. Denial refuses a perception; repression removes an idea that was once conscious. Isolation of affect keeps the idea and strips the feeling; intellectualisation puts a theory where the feeling was. Suppression is conscious, repression is not.

WHAT EARNS THE MARKS

- **The definition attributed.** Anna Freud 1936, repression as Freud's prototype.
- **Four levels, named as levels,** with the mechanisms inside them rather than in one flat list. A list with no level is the standard mid-band answer.
- **The confusable pairs separated.** Projection against projective identification is the one examiners probe.

SEE ALSO Slot I - 29 classifying the defences and using them in treatment Slot I - 34 Anna Freud SPRINT

Day 5, social, learning and personality psychology

I - 27

Relevance of family theories of Schizophrenia today. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

1	The claim they made	The family as cause, in its own era
5	The theories	Each with author, year and verdict
2	What survived	Expressed emotion, and why it lived
2	Relevance today	From blame to burden

THEORY	WHAT IT CLAIMED	WHOSE, AND WHEN	WHERE IT STANDS TODAY
Schizophrenogenic mother	A cold, rejecting mother produces the illness	Fromm-Reichmann, 1948	Abandoned; it harmed a generation of families
Double bind	Contradictory injunctions, no escape, comment forbidden	Bateson, Jackson, Haley, Weakland, 1956	Not causal; survives as a description of confusing talk
Marital schism and skew	Open chronic conflict, or one partner's dominance accepted	Lidz, 1957	Not causal; discord is a stressor like any other
Pseudomutuality and rubber fence	A surface of harmony that forbids real difference	Wynne, Ryckoff, Day and Hirsch, 1958	Not causal; the boundary idea survives clinically
Communication deviance	Fragmented, unclear attention in family speech	Singer and Wynne, from 1963	Predicts thought disorder in high-risk samples
Expressed emotion	Criticism, hostility and over-involvement predict relapse	Brown, Birley and Wing, 1972; Vaughn and Leff, 1976	**Stands; replicated, and the basis of family intervention**

The fourth column answers the word today. Five rows are history; the amber row is the one still in the guidelines.

- **Why expressed emotion survived when the rest did not.** It was measured rather than asserted, on the Camberwell Family Interview, and it made a prospective claim about relapse rather than a retrospective claim about cause. It predicts relapse; it does not predict onset. Vaughn and Leff set high contact at more than thirty-five hours a week face to face.
- **The error the stem is testing.** Treating expressed emotion as a modern version of the schizophrenogenic mother. One is a blaming aetiological claim with no evidence; the other is a measured predictor of course that says nothing about who caused the illness.
- **The Indian evidence, and its limit.** Expressed emotion runs lower in Indian families than in the British samples it was derived from, and the World Health Organization comparative studies reported a better course in centres in developing countries. Both are contested on method, and neither licenses the claim that the joint family is protective in itself.

WHAT EARNS THE MARKS

- **Each theory with its author and decade.** The stem is historical, so unattributed theories earn little.
- **A verdict against every row,** not a description of every row.
- **Expressed emotion separated from blame.** Predicting relapse is not claiming cause, and the shift from blame to burden is what the answer ends on.

SEE ALSO Slot I - 30 the Neo-Freudians and their use in psychotherapy SPRINT

Day 5, social, learning and personality psychology

I - 28

What is the concept of self? Discuss its perspective in psychiatric disorders. [4+6]

4 + 6 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**What the self is**	The knower and the known
2	**Formal features**	Jaspers' four, named as four
4	**Failure by disorder**	Each against the condition that breaks it
2	**The psychodynamic self**	What Kohut and Winnicott add

THE DEFINITION

The self is the person as known to himself: at once the subject that experiences and the object experienced. William James, 1890, separated the I, the knower, from the Me, the known, and divided the Me into material, social and spiritual selves.

ASPECT OF THE SELF	HOW IT FAILS	WHERE THE FAILURE IS SEEN	WHOSE ACCOUNT
Awareness of activity	Thought and act are experienced as authored from outside	**Thought insertion and passivity in schizophrenia**	Jaspers
Awareness of unity	More than one self is experienced at one time	Dissociative identity presentations	Jaspers
Awareness of continuity	The thread back to the earlier self is cut	Dissociative amnesia and fugue	Jaspers
Awareness of boundaries	Self and world stop being demarcated	Ego boundary loss in schizophrenia; drug and ecstatic states	Jaspers
The bodily and narrative self	The self feels unreal, or will not hold one shape across roles	Depersonalisation; borderline identity diffusion	Schilder, Winnicott

- **The four accounts worth naming.** James, 1890, the I and the Me. Cooley, 1902, the looking-glass self, assembled from what we believe others see. Mead, 1934, the self arising in social interaction through the generalised other. Jaspers, 1913, four formal characteristics of self-awareness.
- **The psychodynamic self.** Kohut made the self, not the drive, the unit: built by selfobject responses of mirroring, idealisation and twinship, and fragmenting when they fail. Winnicott separated a true self, rooted in the body, from a false self built to comply.
- **Three words that are not synonyms.** Self-esteem is the value placed on the self, self-concept is its content, self-awareness is the formal fact of having one. Only the third breaks in schizophrenia.

WHAT EARNS THE MARKS

- **The I and the Me, attributed to James.** Two of the four concept marks live in that one sentence.
- **Jaspers' four characteristics named as four,** because the disorder half is hung on them.
- **A named disorder against each characteristic,** not a general claim that the self is disturbed in mental illness.

SEE ALSO Slot I - 31 Carl Jung and the Self as archetype Slot I - 33 the topographical theory of mind **SPRINT** Day 5, social, learning and personality psychology

I - 29

How do you classify the defense mechanisms? Describe their classical use in treatment of psychiatric disorders. [3+7]

3 + 7 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**The classifications**	Three schemes, and what each one is for
4	**The treatment decision**	Support, interpret or convert, decided by level
2	**The sequence**	Resistance before content, defence before impulse
1	**The limit**	Where interpreting a defence does harm

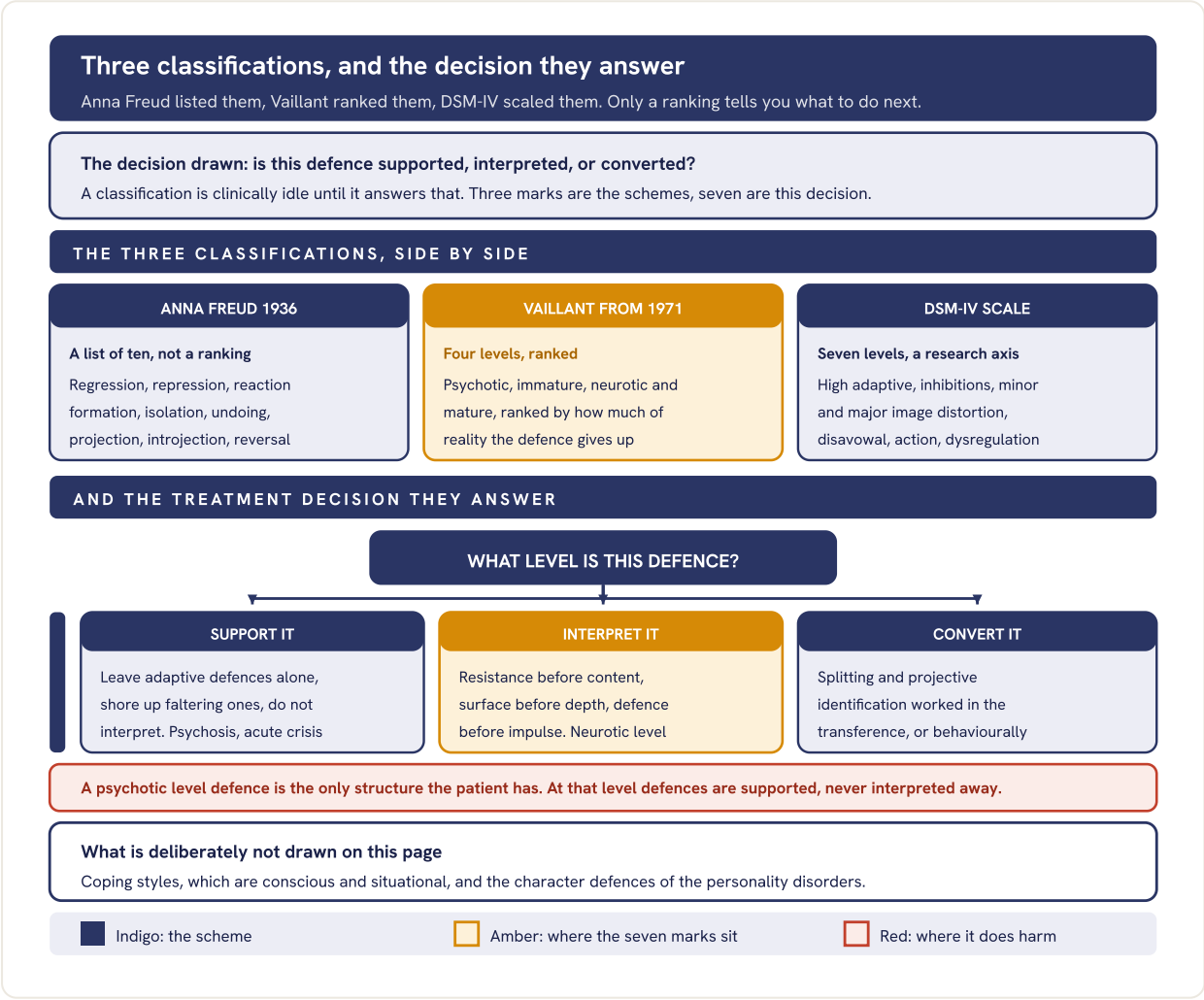


Fig 029.1 The three schemes across the top, the level question as the single node, and the three treatment lanes below it, with the interpretive lane in amber.

- **The sequence, which is the classical use.** Interpret resistance before content, surface before depth, and the defence before the impulse it hides. The aim is never to remove a defence but to replace it with a more adaptive one doing the same job.
- **What each scheme is for.** Anna Freud's list is what a clinical description draws on. Vaillant's levels are what a formulation and a prognosis draw on, because only a ranking supports a decision.

WHAT EARNS THE MARKS

- **Three schemes with their authors,** not one undifferentiated list of mechanisms.
- **The level used to make a decision.** The stem says classical use, so stopping at the classification throws away seven marks.
- **The interpretive sequence, in order.** Resistance, then content; defence, then impulse.
- **The red strip.** Supporting rather than interpreting a psychotic level defence is what shows clinical use.

SEE ALSO Slot I - 26 the ego defence mechanisms described Slot I - 34 Anna Freud **SPRINT**

Day 5, social, learning and personality psychology

I - 30

Describe various theories of Neo-Freudians. Discuss their use in psychotherapy. [5+5]

5 + 5 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1	The axis	What makes a theorist Neo-Freudian rather than Freudian
4	The theories	Each with its break from Freud and its signature concept
4	Use in psychotherapy	The mechanism each one gave the consulting room
1	The honest limit	Where the label itself is loose

THEORIST	THE BREAK WITH FREUD	SIGNATURE CONCEPTS	WHAT IT GAVE PSYCHOTHERAPY
Alfred Adler	Social striving replaces the sexual drive	Inferiority and compensation, style of life, social interest	**Encouragement and goal-focused brief work; an ancestor of cognitive therapy**
Carl Jung	Libido is general psychic energy	Collective unconscious, archetypes, individuation, introversion and extraversion	Dream and symbol work; the tasks of the second half of life
Karen Horney	Basic anxiety, not libido, is the root of neurosis	Moving toward, against and away from people; the tyranny of the should	Work on the idealised self; the founding critique of Freud on women
Harry Stack Sullivan	Personality exists only between people	Self-system; good me, bad me, not me; parataxic distortion	**The therapist as participant observer; the parent of interpersonal psychotherapy**
Erich Fromm	Society and the burden of freedom form character	Escape from freedom, productive orientation	Existential and humanistic therapies
Erik Erikson	Development is psychosocial and runs the whole life span	Eight stages, epigenetic principle, identity, moratorium	Identity work in adolescence; life review in later life

The fourth column is the second half of the stem. Amber marks the two whose method survives under its own name in current practice.

- **The axis, stated before the list.** Every one of them kept the unconscious and the developmental frame and gave up the primacy of the sexual drive, putting culture, relationship or social striving in its place. Otto Rank and Wilhelm Reich are sometimes counted with them.
- **The five psychotherapy marks, which most answers lose.** Name a mechanism per theorist rather than a school: encouragement and goal setting from Adler, symbol work from Jung, the idealised self from Horney, the participant observer from Sullivan, identity and moratorium from Erikson.

WHAT EARNS THE MARKS

- **The axis in one line before any name.** A list of theorists with no stated criterion reads as recall.
- **Each theorist tied to what he or she abandoned,** not merely to a concept.
- **A therapeutic mechanism per row.** The stem splits five and five, so the last column carries half the paper.

SEE ALSO Slot I - 32 Neo-Freudians against Post-Freudians Slot I - 31 Carl Jung in full SPRINT

Day 5, social, learning and personality psychology

I - 31

Carl Jung. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The man and the break	Dates, and the one revision that made him Jungian
4	Structure of the psyche	Collective unconscious, archetypes, complexes
2	Typology and method	Attitudes, functions, word association, amplification
2	Individuation	The goal, and why it belongs to the second half of life

THE MAN, AND THE ONE REVISION

Carl Gustav Jung, 1875 to 1961, Swiss psychiatrist, Freud's designated successor until the break of 1913. He founded analytical psychology on a single revision: libido is general psychic energy rather than sexual energy, and the unconscious is collective as well as personal.

DOMAIN	THE CONCEPT	WHAT JUNG MEANT BY IT	WHERE IT SHOWS UP
Structure	Collective unconscious	An inherited layer common to all people, beneath the personal unconscious	The recurrence of the same myths and symbols
Structure	Archetypes	Inherited forms that organise experience: persona, shadow, anima and animus, the Self	Persona and shadow are the two most often asked for
Structure	Complexes	Feeling-toned clusters of ideas in the personal unconscious	Shown on his own Word Association Test
Typology	Attitudes and functions	Introversion and extraversion, crossed with thinking, feeling, sensation, intuition	The ancestor of later personality typologies
Development	Individuation	Lifelong integration of the unconscious into a whole centred on the Self	The task of the second half of life
Method	Amplification	A symbol elaborated through myth rather than reduced to biography	Dream work, and active imagination

- **The two confusions that cost marks.** The Self is not the ego: the ego is the centre of consciousness, the Self is the centre and the totality of the whole psyche and is the goal of individuation. The shadow is not the evil in a person but everything unlived and unacknowledged, undeveloped good included.
- **Where he still touches practice.** Symbol and dream work in analytical psychotherapy, midlife presentations read as a developmental task rather than a decline, and the word association method, which was an early objective demonstration that unconscious material can be measured.

WHAT EARNS THE MARKS

- **The revision named, not just the split.** Libido as general psychic energy is what makes him Jungian.
- **Four archetypes named,** and the Self distinguished from the ego.
- **Typology given as two attitudes crossed with four functions,** which is the form Jung used.
- **Individuation defined as a goal,** not as a synonym for growing up.

SEE ALSO Slot I - 32 Neo-Freudians against Post-Freudians Slot I - 28 the concept of self SPRINT

Day 5, social, learning and personality psychology

I - 32

Who are Neo-Freudians? How they are different from Post-Freudians? Write about contributions of Carl Jung. [2+3+5]

2 + 3 + 5 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Who they are	The names, and the criterion that groups them
3 The difference	Broke away against built upon, domain by domain
4 Jung's contributions	Five, each named in his own terms
1 The caveat	Where the two labels overlap

DOMAIN	NEO-FREUDIANS	POST-FREUDIANS	WHAT SETTLES IT
Who	Adler, Jung, Horney, Sullivan, Fromm	Anna Freud, Hartmann, Klein, Winnicott, Fairbairn, Bion, Kohut	**Whether drive theory was abandoned or extended**
Relation to Freud	Broke away, usually in person as well as in theory	Stayed inside psychoanalysis and built on it	**Broke away against built upon**
What drives behaviour	Social striving, culture, inter-personal security	The drive kept, with the ego or the internal object added	The place still given to libido
Central unit	The person in a social field	The ego and its functions, or the internal object	Field against internal structure
Period	Roughly 1910 to 1940	Roughly 1930 onward	Suggestive, not decisive
Technique	Shorter, more active, more directive	The analytic frame kept, inter-pretation deepened	How far the method moved

The amber cells are the discriminators. The rest of the grid supports them, and saying which is which is itself a mark.

- **Jung's contributions, which carry five of the ten marks.** The collective unconscious and its archetypes, persona, shadow, anima and animus, and the Self. Complexes, demonstrated on the Word Association Test. A typology of two attitudes crossed with four functions. Individuation as the task of the second half of life. And amplification: the symbol elaborated rather than reduced.
- **The caveat that earns the last mark.** The label is loose. Jung and Adler are counted as Neo-Freudian because they broke away; Erikson is often counted with them although he never left. State the criterion you are sorting on before you sort the names.

WHAT EARNS THE MARKS

- **Both lists of names given,** because the stem asks who before it asks how.
- **The discriminator identified as the discriminator.** Abandoned against extended, not six differences of equal weight.
- **Five Jung contributions, separately named.** The last third of the stem is half the paper.

SEE ALSO Slot I - 31 Carl Jung in full Slot I - 30 the Neo-Freudian theories in psychotherapy SPRINT

Day 5, social, learning and personality psychology

I - 33

What is primary and secondary process of thinking as proposed by Freud? Write a brief note on topographical theory of mind. [4+6]

4 + 6 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Primary process**	The mode of the unconscious
2	**Secondary process**	The mode of the ego
4	**The topography**	Three systems, and what bars each door
2	**Its successor**	What the structural model added in 1923

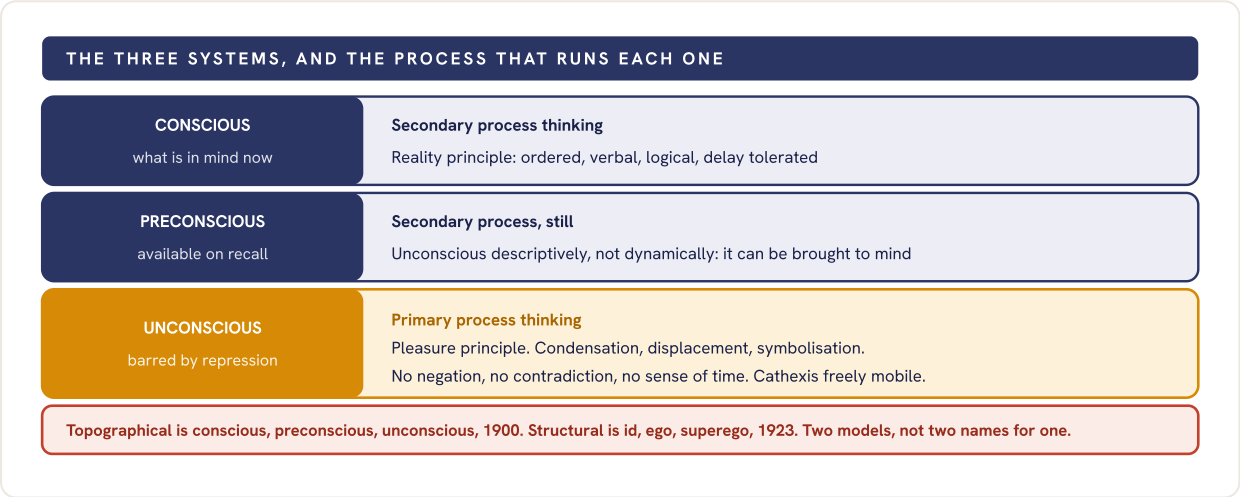


Fig 033.1 The three systems as strata with the correct mode of thinking hung on each. Amber marks the unconscious stratum, where the primary process marks are won; red marks the model most often confused with this one.

- **The two processes, as Freud defined them.** Primary process is the mode of the unconscious and of the infant: immediate discharge under the pleasure principle, cathexis freely mobile, working by condensation, displacement and symbolisation. Secondary process is the mode of the ego: cathexis bound, the reality principle, logic, language, and delay tolerated.
- **Where each is seen.** Primary process shows in dreams, in free association, in slips and jokes, and in the formal thought disorder of psychosis. Secondary process is ordinary waking thought, and its loss is what makes psychotic speech read as dreamlike.
- **Why it needed a successor.** The topographical model was supplemented in 1923, not abandoned. Freud found that much of the ego and all of the superego are themselves unconscious, which a model built on the single axis of awareness cannot hold.

WHAT EARNS THE MARKS

- **Both processes defined by their principle,** pleasure against reality, not by their qualities alone.
- **Condensation, displacement and symbolisation named.** They are what make primary process specific.
- **Preconscious separated from unconscious properly.** Descriptive against dynamic is the distinction examiners check.

SEE ALSO Slot I - 29 classifying the defences and using them in treatment Slot I - 28 the concept of self **SPRINT** Day 5, social, learning and personality psychology

I - 34

Anna Freud. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The person	Dates, position, and the book
4	The contributions	Each stated in its own terms
2	The dispute with Klein	What each held, and what it split
2	What survives	Defence analysis, developmental profile

THE PERSON, AND THE BOOK

Anna Freud, 1895 to 1982, youngest child of Sigmund Freud, a lay analyst who founded child psychoanalysis in the British tradition and, with Hartmann, ego psychology. The book every answer must name is The Ego and the Mechanisms of Defence, 1936.

CONTRIBUTION	WHAT SHE ACTUALLY DID	THE CONCEPT TO NAME	WHERE IT STILL SHOWS
Defence, made systematic	Gathered scattered ideas into one account of how the ego wards off anxiety	**Ten mechanisms, and defence analysis as a technique**	Every later classification descends from it
Ego psychology	Moved analytic attention from the id to the ego and its adaptation	The ego as an agent with its own development	Formulation in terms of ego strength
Child analysis	Analysed children through play, education and a real working alliance	The child's ego needs support before it can bear interpretation	Child psychotherapy technique
Developmental lines	Traced development as continuous strands rather than stages alone	From dependency to emotional self-reliance, and others	Developmental profile in child assessment
Direct observation	Ran wartime residential nurseries for separated children in London	Observation admitted as psychoanalytic evidence	The empirical strand Bowlby then took up

- **Her definition of defence, which is the quotable line.** An unconscious operation by which the ego wards off an unacceptable instinctual demand, affect or idea, holding anxiety within bounds. She listed ten: regression, repression, reaction formation, isolation, undoing, projection, introjection, turning against the self, reversal and sublimation.
- **Anna Freud against Melanie Klein, one line each.** Anna Freud held the child's ego too immature for classical interpretation, so the work opens with an educational and supportive alliance. Klein held play to be the child's free association, and interpreted deeply and early. The disagreement split British psychoanalysis into three groups.

WHAT EARNS THE MARKS

- **The 1936 book named with its year.** On a named-person stem the title is a mark on its own.
- **Contributions kept separate,** not merged into one paragraph about child analysis, and ego psychology credited to her and Hartmann together.
- **The Klein dispute given as two positions,** with what it split, rather than as a personal quarrel.

SEE ALSO Slot I - 26 the ego defence mechanisms described

Slot I - 29 classifying the defences and using them in treatment **SPRINT** Day 5, social, learning and personality psychology

I - 35

Transactional analysis. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The frame	Berne, and the four levels of analysis
3	Ego states	Three, with divisions and voice
3	Transactions	Complementary, crossed, ulterior
2	Positions and games	Life positions, strokes, the script

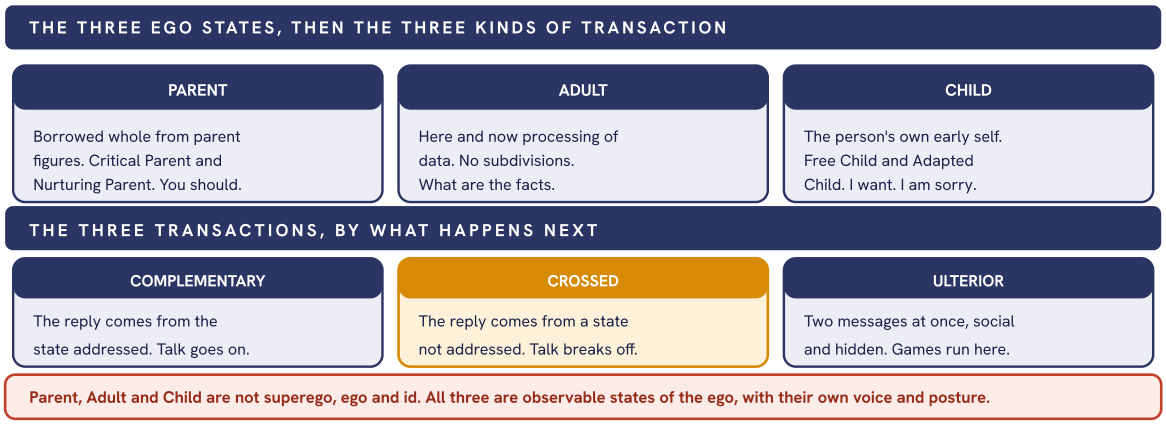


Fig 035.1 Ego states above, transactions below, sorted by what happens to the conversation. Amber marks the crossed transaction, the one this question asks to be illustrated.

LIFE POSITION	STANCE	WHERE IT IS SEEN
I am OK, you are OK	Get on with	Healthy and mutual; the therapy goal
I am OK, you are not OK	Get rid of	Blaming; paranoid and antisocial
I am not OK, you are OK	Get away from	Commonest; depressive, dependent
I am not OK, you are not OK	Get nowhere	Futility; chronic hopelessness

- **The frame, and its four levels.** Eric Berne, from 1958, built a contractual therapy in plain language. Structural analysis names the ego states, transactional analysis reads the exchanges, game analysis names repeated ulterior sequences with a predictable payoff, set out in Games People Play, 1964, and script analysis reads the life plan laid down in childhood.
- **Strokes and time.** A stroke is a unit of recognition, and people take negative strokes over none, which is what keeps a game running. Berne ranked six ways of structuring time: withdrawal, ritual, pastime, activity, game, intimacy.

WHAT EARNS THE MARKS

- **The four levels of analysis named as four.** Structural, transactional, game, script.
- **The three transactions with an example each,** crossed illustrated rather than merely defined.
- **Ego states kept apart from the structural model.** It is the discrimination this question is built on.

SEE ALSO Slot I - 30 the Neo-Freudian theories in psychotherapy Slot I - 33 the topographical theory of mind SPRINT Day 5, social, learning and personality psychology

AREA 04 OF 12 · P1-A05

Learning, memory and general psychology

9	9.9%	I-36	I-44
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 4 · HIGH 2 · RECURRENT 1 · SINGLE 2

REVISION ORDER

The learning block first: four slots and largely one answer, with the two conditioning slots almost interchangeable. The two emotion slots next, likewise, and the James-Lange slot carries the evaluation the other does not ask for. Intelligence after that, theories first and then the Indian test. Emotional intelligence and social learning theory are short and stand on their own.

WHAT IS IN THIS AREA

Learning in four slots: the definition with the models and their clinical importance, classical against operant conditioning with an example of each, Pavlovian conditioning inside behaviour therapy, and social learning theory. Emotion in two, the basic emotions with the theories, and the theories again with James-Lange critically evaluated. Intelligence in three, the theories with their application, emotional intelligence against IQ, and the Indian standardised test. This is the anchor-heaviest area in the book: four of its nine slots have come back across boards and years.

I - 36

Theories of intelligence and their application in Psychiatry. [10]

10 LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KNRUHS, RGUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	Definition	What the construct is, and the general factor debate
4	The theories	Each theorist, his claim, the structure proposed
2	How it is measured	Ratio and deviation quotient, and what a test yields
2	Use in psychiatry	Where the figure changes a clinical decision

THE DEFINITION

Wechsler defined intelligence as the global capacity to act purposefully, to think rationally and to deal effectively with the environment. Binet called it judgement applied to circumstances. Both are functional definitions: intelligence is inferred from performance, never observed directly.

THEORIST	THE CLAIM	STRUCTURE PROPOSED	WHAT IT IS USED FOR
Spearman, 1904	One general ability runs through every task	Two factors, general and specific	The rationale for a single quotient
Thurstone, 1938	There is no single ability, only several	Seven primary mental abilities	Profile testing, subtest scatter
Cattell and Horn	Ability splits by how it ages	Fluid and crystallised intelligence	Separating decline from a low baseline
Guilford, 1967	Ability is a three way classification	Operations by contents by products	Divergent thinking and creativity
Gardner, 1983	Intelligences are plural and independent	Seven, later eight, intelligences	Strengths based educational planning
Sternberg, 1985	Intelligence is what succeeds in context	Analytic, creative and practical	Vocational and rehabilitation planning

- **Measurement.** Stern's ratio quotient, mental age over chronological age times a hundred, fails in adults because mental age plateaus. Wechsler replaced it with the deviation quotient, mean a hundred, standard deviation fifteen. Vernon's hierarchical model is the shape modern batteries take.
- **Application, and its limit.** Intellectual disability needs a low quotient **and** impaired adaptive functioning; the older bands read mild fifty to sixty nine, moderate thirty five to forty nine, severe twenty to thirty four, profound below twenty. Fluid ability falls in dementia while crystallised ability holds, which is what makes that pair useful.

WHAT EARNS THE MARKS

- **A definition with a name attached,** written out before the theories start.
- **Every row of the grid carrying a theorist and a date.** Theories listed without authors read as recall.
- **The amber row, fluid against crystallised,** because it is the one that does clinical work at the bedside.
- **Adaptive functioning named alongside the quotient.** A number alone does not make the diagnosis.

SEE ALSO Slot I - 43 the Indian standardised intelligence test Slot I - 38 emotional intelligence and its relation to quotient

SPRINT Day 4, general and developmental psychology

I - 37

Social learning theory. [10]

10 LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, RGUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 **The claim**	Learning by watching, without doing and without reward
3 **The four processes**	Drawn in order, with the point where learning is complete
3 **The constructs**	Reciprocal determinism, self efficacy, vicarious reinforcement
2 **Clinical use**	The treatments it produced, and the harms it explains

THE CLAIM

Behaviour is acquired by observing a model, held in symbolic form, and performed later when the incentive is right. Direct reinforcement of the learner is not required, and that is what separates this from operant theory.

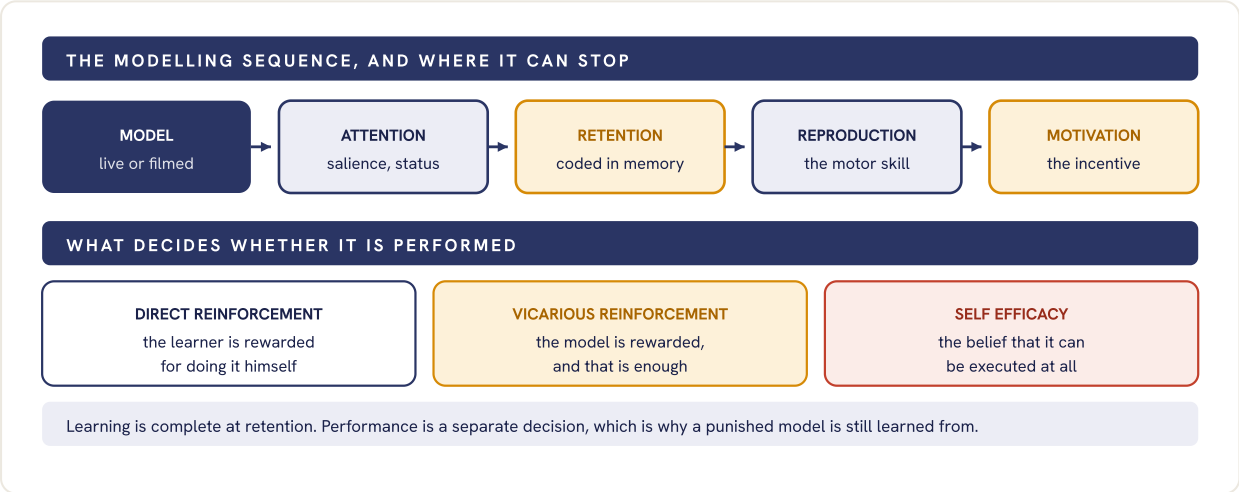


Fig 037.1 The four mediational processes in order, with the two amber stages that carry the theory's whole argument, and the three influences that decide whether what was learned is ever shown.

- **The evidence and the constructs.** Children who watched an adult attack an inflatable doll reproduced the aggression; those who saw the model punished performed less, yet showed on incentive that they had learned as much. Reciprocal determinism holds that person, behaviour and environment each shape the other two.
- **Clinical use, both directions.** Participant modelling in phobia work, behavioural rehearsal in social skills training, video modelling with children. The same mechanism explains learned illness behaviour in a family and imitative self harm after media coverage.

WHAT EARNS THE MARKS

- **Learning separated from performance,** which is the sentence the whole theory turns on.
- **All four mediational processes named in order,** exactly as the figure draws them.
- **Self efficacy named and defined,** built from mastery, vicarious experience, persuasion and bodily state.

SEE ALSO Slot I - 40 the models of learning classified Slot I - 41 classical and operant conditioning SPRINT Day 5, social, learning and personality psychology

I - 38

What is emotional intelligence? Describe components of emotional intelligence. Mention the relationship between emotional intelligence and I.Q. [2+4+4]

2 + 4 + 4 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Definition	What it is, and who named it in which year
4	The components	Three rival models, each with its own component list
3	Against the quotient	What each predicts, and what the overlap really is
1	Clinical bearing	The disorders that sit at the low end

THE DEFINITION

Salovey and Mayer, who named the construct in 1990, defined it as the ability to monitor one's own and other people's feelings, to discriminate among them, and to use that information to guide thinking and action. Goleman's 1995 book widened it well beyond that.

MODEL	COMPONENTS	HOW IT IS MEASURED	STANDING
Ability, Salovey and Mayer	Perceiving emotion, using emotion to aid thought, understanding emotion, managing emotion	Performance test with scored right answers	**The only model that behaves like an intelligence**
Mixed, Goleman	Self awareness, self regulation, motivation, empathy, social skill	Self report and rating by others	Popular, evidence weaker than the claims
Social and emotional, Bar-On	Intrapersonal, interpersonal, adaptability, stress management, general mood	Self report inventory	Broad, close to wellbeing
Trait, Petrides	Emotional self perceptions sited within personality	Self report questionnaire	Overlaps heavily with personality

- **The relationship with the quotient.** Positively but only modestly related, and not substitutes. The quotient remains much the stronger predictor of academic attainment and of cognitively demanding work. Ability emotional intelligence adds a small increment for interpersonal outcomes once quotient and personality are accounted for; trait and mixed measures add least, being largely personality under another name.
- **Clinical bearing, and the trap.** The low end is recognisable in clinic: alexithymia, the empathy profile of autism, the intact cognitive with impaired affective empathy of antisocial personality, the emotion dysregulation of borderline personality. The trap is not naming which model you mean, because a performance test and a self report questionnaire do not measure the same thing.

WHAT EARNS THE MARKS

- **The 1990 definition with both authors,** kept separate from Goleman's later version.
- **Components given model by model,** because the models disagree about what they are.
- **The amber row.** Only the ability model is measured the way an intelligence is measured.

SEE ALSO Slot I - 36 theories of intelligence Slot I - 39 basic emotions and theories of emotion **SPRINT** Day 4, general and developmental psychology

I - 39

What are the basic emotions? Discuss briefly the theories of emotion. [2+8]

2 + 8 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Basic emotions	The criteria first, then the named sets
5	The theories	Each one as a sequence, with its support and its flaw
2	The appraisal turn	What cognition added, and what it displaced
1	Why it matters	One line linking the theory to a treatment

THEORY	THE SEQUENCE PROPOSED	ITS STRONGEST SUPPORT	WHERE IT FAILS
James and Lange, 1884	Bodily change first, emotion is its reading	Facial feedback; blunting after cord injury	Visceral change too slow, too alike
Cannon and Bard, 1927	Thalamus fires to cortex and body at once	Emotion survives loss of feedback	The thalamus is too simple a relay
Schachter and Singer, 1962	Arousal, then a label taken from context	Adrenaline felt as anger or elation by context	Replication has been uneven
Lazarus, 1966	Appraisal first, and it can be automatic	One event, different appraisals, different emotions	Appraisal is hard to measure apart
Papez and MacLean	A closed circuit carries the experience	The connections named are real	Superseded as an account of emotion

- **What makes an emotion basic.** Universal recognition across cultures, a distinct facial expression, quick onset and brief course, a distinct physiological signature, automatic appraisal, a counterpart in other primates.
- **The three sets worth naming.** Ekman gives six, from universally recognised facial expressions: happiness, sadness, fear, anger, disgust, surprise. Plutchik gives eight as four opposed pairs on a wheel, joy against sadness, trust against disgust, fear against anger, surprise against anticipation. Panksepp names seven systems from the circuits themselves: seeking, rage, fear, lust, care, panic and grief, play.
- **The clinical pay-off.** Schachter and Singer is the panic model in embryo. Benign arousal receives a catastrophic label, and it is the label, not the arousal, that interoceptive exposure changes.

PITFALL

These theories are not a league table with a winner. Each was framed against the one before it, and the position now held is that bodily change, appraisal and context all contribute. Writing one as simply correct throws away the discussion marks.

WHAT EARNS THE MARKS

- **The criteria for basic, before any list.** A list with no criteria is recall.
- **Each theory written as a sequence,** exactly as the second column sets it out.
- **Named sets with their authors,** six, eight and seven, not a single unattributed list.

SEE ALSO Slot I - 42 the theories of emotion evaluated at depth Slot I - 38 emotional intelligence SPRINT Day 4, general and developmental psychology

I - 40

Define learning. Write in brief about the basic tenets of different models of learning and their clinical importance in psychiatry. [2+8]

2 + 8 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Definition**	What counts as learning, and what is excluded
3	**The models drawn**	One axis, three branches, the theorists attached
3	**The basic tenets**	The mechanism of each model, one line each
2	**Clinical importance**	A named therapy standing under every branch

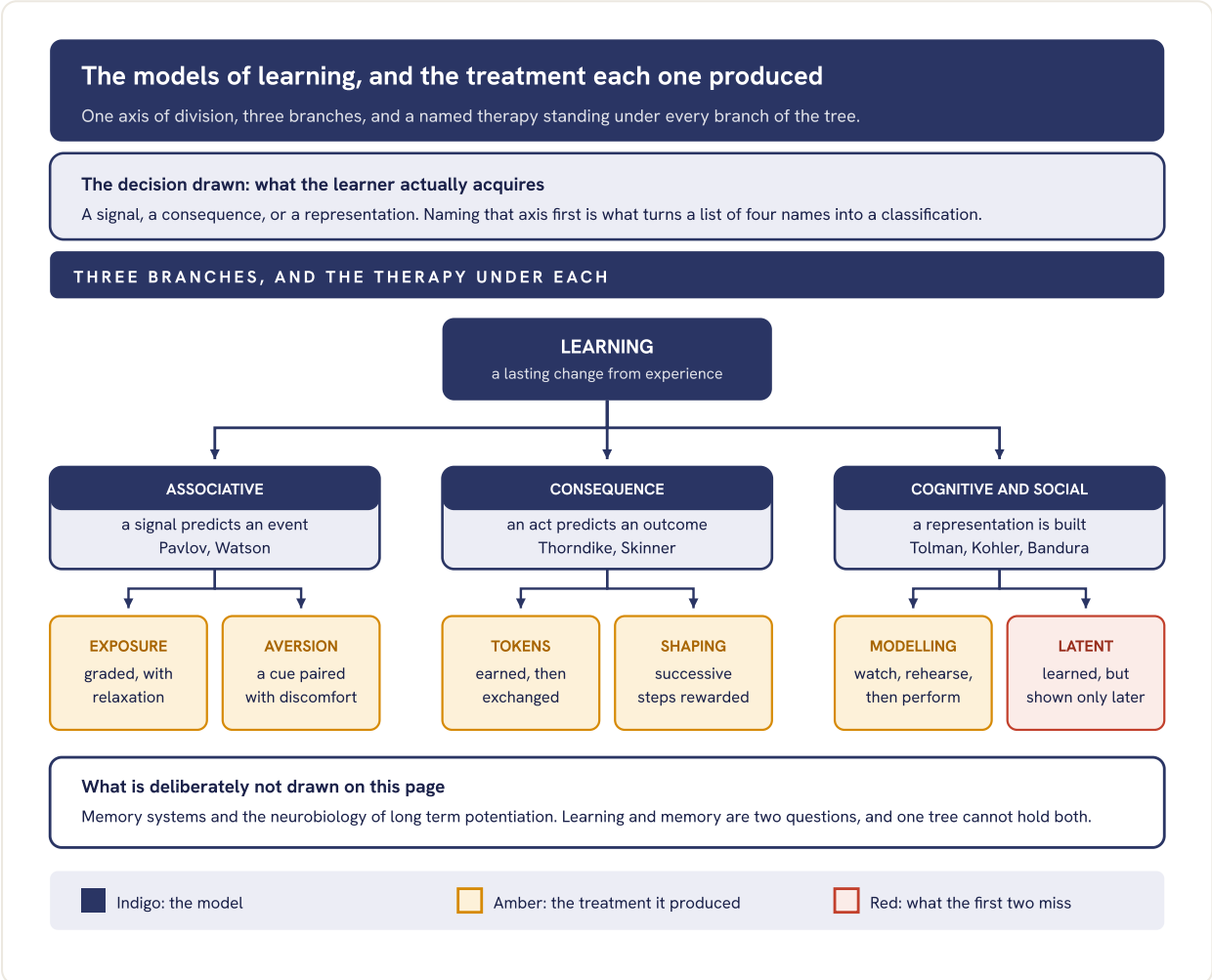


Fig 040.1 The models of learning divided on what the learner acquires, with the therapy each branch produced set in amber beneath it, and latent learning marked in red because it is the finding the associative and consequence models cannot account for.

- **The tenets, one line each.** Classical conditioning pairs a neutral stimulus with one that already elicits a reflex until it elicits that reflex alone. Operant conditioning makes a behaviour more or less likely by what follows it. Observational learning is acquired by watching a model and stored before it is performed. Cognitive learning builds an internal representation, shown by latent learning and by insight.

- **The definition, and what it excludes.** Learning is a relatively permanent change in behaviour, or in the capacity for it, following experience. Change from fatigue, maturation, injury or a drug is not learning.

PITFALL

Naming the models without naming the treatment each produced answers half the question. Clinical importance carries the second eight marks, and every branch has a named therapy under it.

WHAT EARNS THE MARKS

- **The definition with its exclusions,** before the classification begins.
- **The axis stated in one line.** A tree that does not name its axis is a list.
- **Every branch with its theorists,** Pavlov, Skinner, Tolman, Bandura.
- **A named therapy under each branch.** The amber leaves are the clinical marks.

SEE ALSO Slot I - 41 classical and operant conditioning compared Slot I - 44 Pavlovian conditioning in behaviour therapy

SPRINT Day 5, social, learning and personality psychology

I - 41

Classical conditioning and Operant conditioning. Discuss their application in clinical setting with one example for each. [10]

10 | ASKED AT 15 MARKS | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 Classical	The paradigm, and the four terms in their order
2 Operant	The four consequences, named without error
4 Applications	A named treatment and one example for each
2 Shared phenomena	Extinction, generalisation, discrimination, schedules

FEATURE	CLASSICAL	OPERANT	WHAT SETTLES IT
What is learned	A signal predicts an event	An act produces an outcome	**Is the behaviour elicited or emitted**
The response	Reflexive, autonomic, involuntary	Skeletal, voluntary, acting on the world	Could the person withhold it
Where the event sits	The signal comes before the response	The consequence comes after it	**Which side of the behaviour the event falls**
The law invoked	Contiguity of two stimuli, Pavlov and Watson	Effect of the consequence, Thorndike and Skinner	Pairing against payoff
Clinical example	Graded desensitisation for a phobia of heights	A token economy on a rehabilitation ward	Is the cue changed, or the consequence

- **The operant terms, used correctly.** Reinforcement raises the rate of a behaviour and punishment lowers it; positive means a stimulus is added and negative means one is removed. That gives four cells, not two. Negative reinforcement is therefore not a punishment, and it is precisely what maintains avoidance in every anxiety disorder.
- **What both paradigms share.** Acquisition, extinction, spontaneous recovery, stimulus generalisation and discrimination all appear on either side. Among the operant schedules a variable ratio produces the behaviour hardest to extinguish, which is why the machine rather than the salary is the model for gambling.

PITFALL

The commonest error is calling negative reinforcement a punishment. The second is offering exposure as the operant example: graded exposure works by extinguishing a conditioned response and belongs on the classical side of this table. Aversion therapy sits there too.

WHAT EARNS THE MARKS

- **Both paradigms defined before the comparison begins,** each with its theorists named.
- **The two amber cells,** elicited against emitted, and which side of the behaviour the event falls.
- **One named treatment and one concrete example for each,** which is what the stem asks for twice.

SEE ALSO Slot I - 40 the models of learning classified Slot I - 44 Pavlovian conditioning in behaviour therapy SPRINT Day 5, social, learning and personality psychology

I - 42

Discuss various theories of emotions and critically evaluate James-Lange theory and its implications. [10]

10 LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, RGUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	The field in brief	Four positions, one line each, in date order
2	The claim itself	James and Lange stated so it can be tested
4	The evaluation	Test by test, with a verdict on each
2	Implications	What survives, and what it is used for now

THE CLAIM, STATED SO IT CAN BE TESTED

James held that bodily change is not the expression of emotion but its cause: we are sad because we weep. Lange said the same of vasomotor change. Stated strongly, peripheral feedback is necessary and sufficient for feeling; stated weakly, it only modulates intensity.

THE TEST APPLIED	WHAT WAS FOUND	HOW THE THEORY STANDS	VERDICT
Cutting visceral feedback	Emotional behaviour persists in animals	Feedback is not necessary for it	**Strong form refuted**
Comparing autonomic patterns	Fear and anger look largely alike	Some patterning since demonstrated	Partly answered
Timing the visceral response	Viscera are slow, sparsely innervated	Facial and muscular feedback is faster	Partly answered
Injecting adrenaline	Arousal without any felt emotion	A cognitive label must be added	**Reframed by Schachter**
Studying spinal cord injury	Higher lesions, less intense feeling	Feedback modulates intensity	Weak form supported

- **The field, in one line each.** Cannon and Bard put the thalamus at the centre, sending to cortex and body at once. Schachter and Singer made emotion arousal plus a label. Lazarus put appraisal first. Papez and MacLean supplied the circuit, not the psychology. LeDoux separated a fast subcortical route from a slower cortical one.
- **Implications, the second half of the stem.** Facial feedback and its therapeutic offshoots; relaxation working on the body to change the feeling; beta blockade for performance anxiety, where removing the peripheral signal lowers the fear; interoceptive exposure in panic; and Damasio's somatic marker account of how bodily states bias decision.

WHAT EARNS THE MARKS

- **The strong and weak forms separated in the definition box.** Critical evaluation is impossible until the claim is stated precisely.
- **A verdict in every row,** not a list of criticisms with no conclusion attached.
- **The implications answered separately.** They carry their own marks and most answers omit them entirely.

SEE ALSO Slot I - 39 basic emotions and the theories in brief Slot I - 38 emotional intelligence SPRINT

Day 4, general and developmental psychology

I - 43

What do you understand by the term intelligence? Describe the one test commonly used for measuring intelligence which has been standardized for use in India. [10]

10 | ASKED AT 15 MARKS | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	What intelligence is	A definition, and the quotient it is expressed as
2	The Indian tests	Named, with the one you will describe marked
3	The chosen test	Structure, administration, scoring
2	Interpretation	What the figure settles, and what it cannot

TEST	AUTHOR AND YEAR	AGE RANGE	WHERE IT IS USED
Malin's Intelligence Scale for Indian Children	Malin, 1969	Six to fifteen years	**The standard child scale, described below**
Binet-Kamat Test of Intelligence	Kamat, 1934	Childhood into early adulthood	Indian adaptation of the Binet tradition
Bhatia Battery of Performance Tests	Bhatia, 1955	Eleven years and above	Wholly performance based, so language and literacy do not limit it
Vineland Social Maturity Scale, Indian adaptation	Malin	Infancy to adulthood	Social maturity, not intelligence; used alongside, never instead

- **Structure of the chosen test.** An Indian adaptation of the Wechsler children's scale, with Indian norms and content. Eleven subtests in two halves. Six verbal: information, general comprehension, arithmetic, analogies and similarities, vocabulary, digit span. Five performance: picture completion, block design, object assembly, coding, mazes. It yields verbal, performance and full scale quotients.
- **Administration and scoring.** Individually administered by a trained examiner in about an hour, alternating the two halves to hold attention. Raw scores convert to scaled scores, and those to a deviation quotient of mean a hundred, standard deviation fifteen. Subtest scatter is read alongside the total.

INTERPRETATION, AND THE HONEST LIMITS

Used for intellectual disability certification, for specific learning disorder where a verbal and performance discrepancy matters, and for documenting decline against a baseline. The limits belong in the answer: norms drift, schooling inflates the verbal half, sensory and motor impairment depress the performance half, and a quotient never diagnoses disability alone.

WHAT EARNS THE MARKS

- **A definition before the test.** The first part of the stem carries its own marks.
- **One test chosen and named,** with author and year, rather than a survey of five.
- **The subtests in their two halves,** which is the body of the description.

SEE ALSO Slot I - 36 theories of intelligence Slot I - 49 rating scales and their psychometric properties SPRINT Day 4, general and developmental psychology

I - 44

Discuss Pavlovian conditioning. Discuss its role in behaviour therapy. [5+5]

5 + 5 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The paradigm	Four terms, in the order in which they occur
3	The phenomena	Extinction onward, each with its named effect
3	In behaviour therapy	Each treatment traced back to a phenomenon
2	Why relapse happens	Extinction is inhibition, never erasure

THE PARADIGM

An unconditioned stimulus already produces an unconditioned response. A neutral stimulus presented repeatedly just before it comes to produce a similar response alone; it is then a conditioned stimulus, its effect a conditioned response. Rescorla's correction is worth a line: what is learned is prediction, not mere pairing.

- **The phenomena to name.** Acquisition, extinction when the signal comes without the event, spontaneous recovery after a rest. Generalisation and discrimination. Higher order conditioning. Experimental neurosis, from demanding a discrimination that cannot be made. Blocking, where a predictive cue prevents a new one being learned. Preparedness, which is why snakes condition readily and sockets do not. Taste aversion, learned in one trial across hours.
- **The therapy lineage.** Watson and Rayner conditioned fear in an eleven month old infant; Jones then removed a child's fear by pairing the animal with food and with unafraid children, the first behaviour therapy on record. Wolpe formalised it as systematic desensitisation: a graded hierarchy, a response incompatible with anxiety, exposure to each step until it settles. Response prevention in obsessive compulsive disorder, flooding, aversion therapy, cue exposure for craving, the bell and pad for enuresis and interoceptive exposure in panic all descend from it.
- **Conditioning as pathogenesis, not only treatment.** Conditioned craving and conditioned withdrawal explain relapse on returning to the place of use. Anticipatory nausea before chemotherapy is a conditioned response to the clinic. Agoraphobic avoidance begins as generalisation from a single panic.

PITFALL

Extinction does not erase the original learning. It builds a new inhibitory association tied to its context, which is why fear returns with time, in a new setting, or after one unexpected exposure.

WHAT EARNS THE MARKS

- **The four terms in the right order,** with prediction rather than pairing named as what is learned.
- **Named phenomena, not a paraphrase.** Blocking, preparedness and taste aversion are the ones most answers miss.
- **The relapse paragraph.** Context dependent extinction is the discriminating point on this stem.

SEE ALSO Slot I - 41 classical and operant conditioning compared Slot I - 40 the models of learning classified SPRINT Day 5, social, learning and personality psychology

AREA 05 OF 12 · P1-A09

Psychometry and assessment

9	9.1%	I-45	I-53
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 4 SINGLE 5

REVISION ORDER

The three projective slots together: they are one body of material asked at three widths, and the catalogue slot half writes the other two. The two rating-scale slots next, they share their psychometrics. The mini mental state examination and the structured assessment slot after that, both short. The personality-tools slot and the socioeconomic slot are independent of everything else in this area.

WHAT IS IN THIS AREA

Projective testing in three slots: the Thematic Apperception Test in full, the classification with any one instrument described in detail, and the catalogue. The mini mental state examination. The classification of rating scales with the psychometric properties and the standardisation of a newly built intelligence test. The scales of depression, with one described as used in practice. Structured clinical assessment, and a slot that wants five theories of personality evaluated with four assessment tools. One slot here asks for the determination of socioeconomic strata in India. That is a social classification question the boards set inside an assessment area, and it is answered on its page as it was set.

I - 45

What is apperception? Describe administration and interpretation of Thematic Apperception Test. [2+4+4]

2 + 4 + 4 | PSYCHOMETRY AND ASSESSMENT

DNB, KNRUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Apperception	Perception plus the meaning the perceiver adds
4 Administration	Cards, instruction, sessions, the inquiry
3 Interpretation	Hero, need, press, thema, across stories
1 Standing	A battery supplement, never a diagnosis

THE DEFINITION

Apperception is perception interpreted: a new percept absorbed into, and given meaning by, the perceiver's past experience, needs and expectations. Make the stimulus ambiguous enough and the meaning can come only from the perceiver, which is the idea Murray built the test on.

STAGE	WHAT THE TESTER DOES	WHAT IS RECORDED
Selection	Picks a subset from the card series using the age and sex codes each card carries, one card being blank	Which cards, and in what order
Instruction	Asks for a story with what led up to the scene, what is happening, what the figures think and feel, and how it ends	**The story, verbatim, in the subject's own words**
Sessions	Around twenty cards over two sittings classically; about ten in ordinary clinical use	Latency to first word, time per card
Inquiry	Returns afterwards to ask about vague endings, omitted figures and refusals	Blocks, evasions and corrections

The cards are copyrighted. Describe the procedure and the codes, never the pictures and never a story.

- **Murray's frame.** Identify the hero, the hero's needs, the press acting on the hero from outside, and the outcome. Needs and press together give the thema.
- **Read across, not down.** A single story means little. Recurrent theme, repeated conflict, constant emotional tone and stereotyped resolution across the set are the data.
- **The limit.** Formal systems such as Bellak's exist, but routine use stays qualitative, so interpreter dependence is the stated weakness. It yields no diagnosis and supplements a battery when self-report is guarded.

WHAT EARNS THE MARKS

- **Apperception defined as meaning added to a percept,** not as a synonym for perception.
- **The instruction written out in its four parts.** Past, present, thoughts and feelings, ending.
- **Hero, need, press and thema named as the interpretive frame,** with Murray attached.
- **The honest limit.** Supplement, not a diagnostic test, and interpreter dependent.

SEE ALSO Slot I - 48 projective tests classified Slot I - 53 projective tests, short note **SPRINT**

Day 6, psychometry, assessment and descriptive psychopathology

I - 46

Describe the methods of determination of socioeconomic strata in India. [10]

10 PSYCHOMETRY AND ASSESSMENT KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Why it is measured	A covariate in every Indian clinical study
3	The household-head route	Kuppuswamy: education, occupation, income
3	The per capita route	Prasad, and why it needs re-indexing
2	Rural and asset routes	Pareek, standard of living, wealth index

SCALE	UNIT IT SCORES	DOMAINS SCORED	WHERE IT FITS
Modified Kuppuswamy	Head of the family	Education, occupation, monthly family income	**Urban and semi-urban, the default in Indian psychiatry papers**
B G Prasad	Per capita monthly income	Income alone, in five graded classes	**Rural and urban, single-variable, quick**
Udai Pareek	The rural household	Caste, occupation, education, land, housing, possessions, social participation	Village and agrarian samples
Standard of living index	Household assets	Housing, sanitation, fuel, durables, land	Large surveys where income recall fails
Wealth index	Household assets	Same asset set, weighted by principal component analysis	National survey work and quintile comparison

Amber marks the two an examiner expects you to be able to apply, not merely name.

- **Kuppuswamy, the structure.** Three components scored separately: education one to seven, occupation one to ten, family income one to twelve. The total runs from three to twenty-nine and falls into five classes, upper, upper middle, lower middle, upper lower and lower.
- **The number you must not memorise.** The income slabs of both Kuppuswamy and Prasad are re-indexed every year to the consumer price index for industrial workers. Quote the year of the revision you used, never a remembered rupee figure.
- **Choosing one.** Head of family for urban clinic samples, per capita for community samples, asset based where income is under-reported or seasonal.
- **Where they fail.** Joint families dilute per capita income, agricultural income is seasonal and under-declared, and none of these scales measures caste disadvantage or debt.

WHAT EARNS THE MARKS

- **Named scales, not the phrase socioeconomic status.** Four names on the page is four ticks.
- **Kuppuswamy's three components with their score ranges,** and the five classes they produce.
- **The re-indexing rule stated explicitly.** It is the single point that separates a current answer from a copied one.
- **One line on choosing between them** for a stated study setting.

SEE ALSO Slot I - 50 structured clinical assessment Slot I - 83 the National Mental Health Survey

I - 47

Mini mental state examination. [10]

10 PSYCHOMETRY AND ASSESSMENT KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What it is	Author, year, and the one job it does
4 Structure	Seven domains, thirty points, the allocation
2 Interpretation	The traditional cut, and what shifts it
2 Limits in India	Literacy load, and the adaptation to use instead

THE DEFINITION

The Mini Mental State Examination (Folstein, Folstein and McHugh, 1975) is a brief clinician-administered screen of global cognitive function, scored out of thirty and completed in about five to ten minutes. It screens. It does not diagnose, it does not localise, and it is not a neuropsychological battery.

DOMAIN	POINTS	WHAT IT SAMPLES
Orientation to time	5	Temporal orientation, the earliest to go
Orientation to place	5	Spatial orientation across nested levels
Registration	3	Immediate encoding of new material
Attention and calculation	5	Sustained attention and working memory
Recall	3	Short-term retention after interference
Language	8	Naming, repetition, comprehension, reading, writing
Construction	1	Visuoconstructional copying

Thirty points across seven domains. The amber rows are where delirium and early dementia declare themselves first.

- **The cut.** Below twenty-four traditionally suggests cognitive impairment, with twenty-four to thirty read as unimpaired, eighteen to twenty-three as mild and seventeen or below as severe. Age and education move the threshold, so the cut is a prompt to test further, never a diagnosis.
- **What it misses.** It is insensitive to mild impairment and largely blind to executive and visuospatial failure: a patient can score full marks and still be grossly dysexecutive. The Montreal Cognitive Assessment, also out of thirty, is preferred when early change is the question.
- **The Indian caveat.** It is language and literacy loaded, so Western cut-offs misclassify non-literate patients. Use a validated vernacular adaptation such as the Hindi Mental State Examination, and screen in a language the patient reads.

WHAT EARNS THE MARKS

- **The point allocation reproduced,** domain by domain, summing to thirty.
- **The cut given with its qualifier.** A bare "less than twenty-four" without age and education loses the second mark.
- **Screen, not diagnosis,** written as a sentence rather than assumed.
- **The vernacular adaptation named.** It is the Indian mark on this question.

SEE ALSO Slot I - 50 structured clinical assessment Slot I - 95 disorders of consciousness SPRINT

Day 6, psychometry, assessment and descriptive psychopathology

I - 48

What do you understand by projective tests? Name different projective tests and describe any one in detail. [2+2+6]

2 + 2 + 6 | PSYCHOMETRY AND ASSESSMENT

DNB, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **The projective hypothesis**	Ambiguous stimulus, hidden key, broad band
2 **The classification**	Five response modes, named tests under each
4 **One in detail**	Rorschach: material, phases, scoring codes
2 **Standing**	What it is good for, and the honest weakness

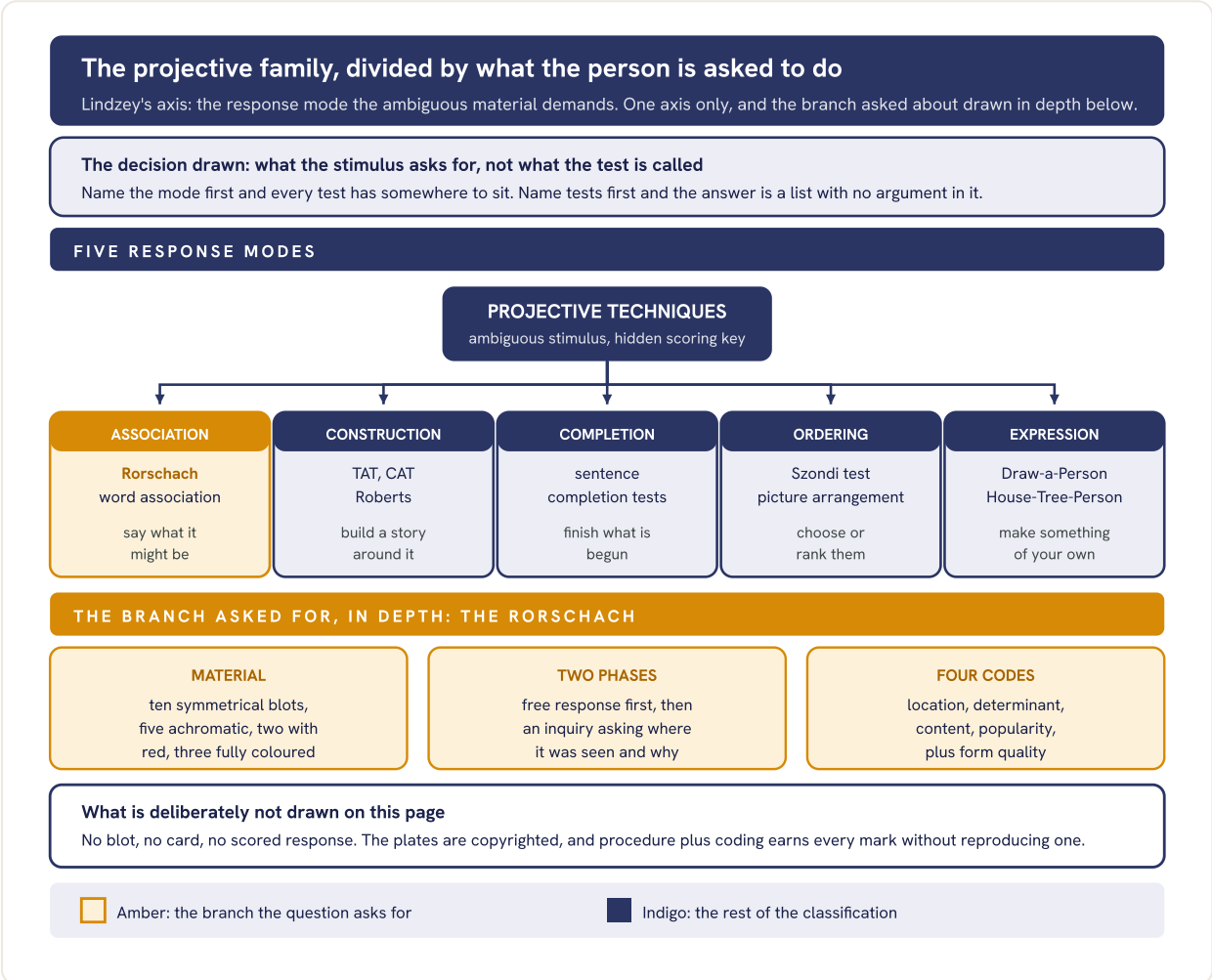


Fig 048.1 The projective techniques divided on one axis, the response mode, with the association branch opened out into the material, the phases and the coding of the Rorschach.

- **The projective hypothesis.** When the stimulus is ambiguous and the scoring key hidden, the structure the person imposes must come from within, so needs, conflicts and defences are read off it. Sampling is broad band and it is harder to fake than self-report.
- **Rorschach in one line.** Hermann Rorschach, Psychodiagnostik, 1921; scored today by the Exner Comprehensive System or its successor, the Rorschach Performance Assessment System.

- **The honest weakness.** Inter-rater and criterion validity are contested, norms are culture bound, administration is long. It supplements a battery when thought process is the referral question, never standing alone.

WHAT EARNS THE MARKS

- **The axis named before the list.** Response mode, then the five branches, then the tests.
- **Every branch of the plate reproduced.** Five modes with a named test under each is the two-mark listing part complete.
- **Depth only in the branch chosen,** material, phases and coding, and nowhere else.
- **The contested psychometrics named,** not defended and not hidden.

SEE ALSO Slot I - 45 the Thematic Apperception Test Slot I - 53 projective tests, short note **SPRINT**

Day 6, psychometry, assessment and descriptive psychopathology

I - 49

Classify psychiatry rating scales. What are the psychometric properties of a psychiatric scale? Describe different procedures of standardization of a newly developed intelligence test. [2+2+6]

2 + 2 + 6 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Classify the scales	By rater, by purpose, by breadth, by scaling
2	The properties	Reliability, validity, and sensitivity to change
5	Standardisation	Items, pilot, fixed procedure, norms, IQ scale
1	Release	What has to be true before it is used

STEP	WHAT IS DONE	EVIDENCE IT YIELDS	PROPERTY SECURED
Blueprint and items	Define the construct, write a surplus of items, pilot them	Difficulty and discrimination	Content validity
Fix the procedure	Freeze instructions, order, timing, stop rules, scoring key	One administration everywhere	Standardisation
Norming	A large sample stratified by age, sex, education, region	Age-graded norm tables	Comparability
Transformation	Raw score to a deviation quotient, fixed mean and spread	The deviation IQ	Interpretability
Reliability, validity	Re-test, split-half, alternate forms, inter-rater; then correlate against an established test	A coefficient per method	Both, in that order

STATUS: WHAT HAS TO BE TRUE BEFORE IT IS USED

A test becomes usable when the procedure is frozen, the norms come from the population it will be used on, and a manual exists that a second examiner can follow to the same score. An imported test used without local norms is unstandardised, whatever its reputation abroad.

- **Classifying the scales.** By rater, self, clinician or informant. By purpose, screening, diagnostic, severity or outcome. By breadth, global against symptom specific. By scaling, categorical, ordinal, Likert or visual analogue.
- **The properties.** Reliability is consistency: re-test, internal consistency, alternate forms, inter-rater agreement. Validity is accuracy: face, content, criterion and construct evidence. A severity scale needs one more, sensitivity to change.
- **The order that matters.** Reliability is necessary and not sufficient, since a test can be consistently wrong. Validity evidence comes after reliability, never instead of it.

WHAT EARNS THE MARKS

- **A classification with a stated axis,** not an unsorted list of scale names.
- **Reliability and validity each broken into their named subtypes.**
- **Norming written as a step separate from procedure fixing.** They are the two halves of standardisation and most answers merge them.

SEE ALSO Slot I - 52 scales in research and depression instruments Slot I - 80 reliability and validity in study design

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

I - 50

What is structured clinical assessment? Discuss its role and limitations in psychiatry. [2+5+3]

2 + 5 + 3 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Fixed questions, fixed probes, operational scoring
3 What it fixes	Information variance and criterion variance
2 Where it is used	The named instruments, by setting
3 Limitations	Time, rigidity, culture, and what it cannot do

THE DEFINITION

Structured clinical assessment replaces the free interview with fixed questions in a fixed order, with defined probes and operational rules for scoring each response against diagnostic criteria. A fully structured instrument fixes the wording and can be given by a trained lay interviewer; a semi-structured one fixes coverage and criteria but leaves judgement in the rating, so it needs a clinician.

- **The two variances it removes.** Information variance, two clinicians eliciting different facts because they asked different questions. Criterion variance, two clinicians applying different thresholds to the same facts. Structuring the interview removes the first; operational criteria remove the second. Together they are the main source of unreliable diagnosis.
- **The instruments, by setting.** Semi-structured and clinician-rated for research diagnosis and for schedules built on a glossary. Fully structured and lay-administrable for large epidemiological surveys, which is how national and cross-national prevalence figures are generated at all. Brief structured instruments for screening in busy clinical services, and child versions for developmental work.
- **What else it buys.** A reproducible case definition for a trial, a teachable framework for a trainee, and a defensible record where the diagnosis may be examined later.
- **The limits.** Long, needing training and re-calibration. It rates presence rather than severity or function, is rigid with comorbid and subthreshold presentations, and inherits every error in the classification it encodes. Translated instruments need re-validation, not only translation.

PITFALL

Treating the schedule as the assessment. It produces a diagnosis, not a formulation. Nothing in it asks why this person became ill now, and the rapport lost to a questionnaire is the same rapport the treatment will need.

WHAT EARNS THE MARKS

- **Structured and semi-structured told apart,** by who may administer and what is fixed.
- **Information variance and criterion variance named as terms.** They are the reason the whole method exists.
- **Instruments placed by setting,** research, survey, clinic, rather than listed loose.
- **Diagnosis separated from formulation** in the limitations, which is where the last marks sit.

SEE ALSO Slot I - 47 the mini mental state examination Slot I - 49 rating scales and their properties SPRINT

Day 6, psychometry, assessment and descriptive psychopathology

I - 51

a) What are the 5 major theories of personality? b) Critically evaluate those major theories. c) Briefly write about any four such personality assessment tools related to those theories. [2+4+4]

2 + 4 + 4 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Name the five	One line each, with the theorist attached
4 Critical evaluation	One strength, one failure, per theory
3 Four tools	Each tied to the theory that generated it
1 The verdict	Which framework the evidence now favours

THEORY	CORE CLAIM	THE CRITIQUE THAT STICKS	TOOL IT GENERATED
Psychoanalytic	Personality is structure and unconscious conflict, laid down early	Retrospective, hard to falsify, weak predictive evidence	**Rorschach, Thematic Apperception Test**
Trait and dispositional	Personality is a small set of stable, measurable dimensions	Describes without explaining; underrates the situation	**16PF, NEO-PI-R, Eysenck questionnaire**
Behavioural and social learning	Personality is learned behaviour, cued and reinforced by situations	Under-explains cross-situational consistency and inner life	**Behavioural analysis, locus of control scale**
Humanistic	Personality is the self, organised around growth and self-actualisation	Optimistic, poorly operationalised, thin on psychopathology	Q-sort, self-concept measures
Cognitive and constructivist	Personality is the system of personal constructs used to anticipate events	Narrow range, limited clinical purchase	**Repertory grid**

Amber marks the four tools that answer part c. Naming a tool without the theory behind it answers half the question.

- **The evaluation, in one argument.** The five differ on where personality is located: inside, in traits, in situations, in the self, or in constructs. The trait framework has won on measurement because it alone produced replicable dimensions with normative data; the psychoanalytic framework has kept its clinical usefulness in formulation while losing the measurement argument.
- **The trap in part c.** A projective and an inventory are not interchangeable tools. Projective tools follow from the psychoanalytic claim that the important material is not available to self-report; inventories follow from the trait claim that it is.
- **Indian use.** Trait inventories need local norms before a raw profile means anything, and translated inventories need re-validation rather than translation alone.

WHAT EARNS THE MARKS

- **Five named theories with a theorist each,** before any evaluation begins.
- **One critique per theory,** written as a limitation rather than as dislike.
- **Four tools, each bolted to its parent theory.** That link is what part c is testing.
- **A stated verdict** on which framework the measurement evidence supports.

SEE ALSO Slot I - 48 projective tests classified Slot I - 53 projective tests, short note SPRINT

Day 5, social, learning and personality psychology

I - 52

a) What are the types of scales used in psychiatry from research point of view? b) Name the rating scales and questionnaires for depression and describe one rating scale in detail that you are using in your clinical practice. [4+6]

4 + 6 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4 Types of scales	Measurement level first, then rater, then purpose
2 Name them	Clinician-rated and self-rated depression instruments
3 One in detail	Structure, range, bands, response and remission
1 Its bias	Where the chosen scale reads high, and why

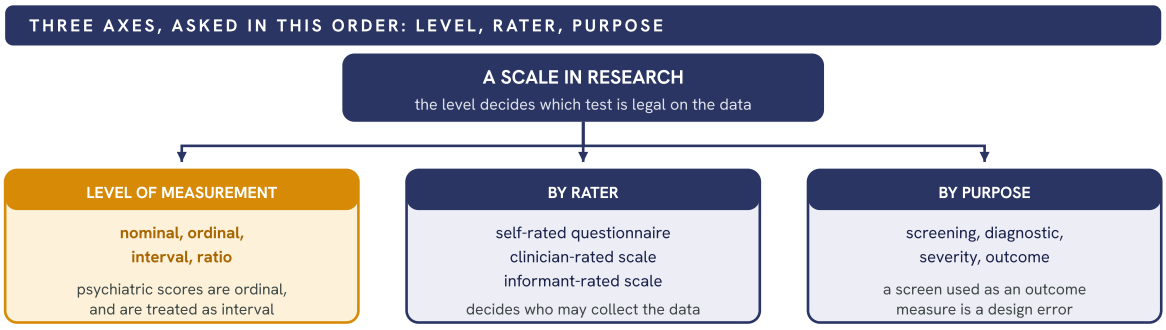


Fig 052.1 The three axes a research scale is classified on, the measurement level in amber because it decides which statistical test the data will allow.

ONE SCALE IN DETAIL: THE HAMILTON DEPRESSION RATING SCALE

Hamilton, 1960. Clinician-rated at interview over the past week, usually in its seventeen-item form: nine items score zero to four and eight zero to two, so the total runs zero to fifty-two. Bands: zero to seven none, eight to sixteen mild, seventeen to twenty-three moderate, twenty-four and above severe. In trials, response is a fall of half and remission a total of seven or below.

- **Name them.** Clinician-rated: Hamilton, Montgomery Asberg. Self-rated: Beck Depression Inventory, the nine-item Patient Health Questionnaire, Zung. Population specific: Geriatric Depression Scale, Edinburgh postnatal scale.
- **Where it reads high.** Hamilton is weighted to sleep, appetite and somatic items, so it inflates in physical illness and in the elderly. Montgomery Asberg was built less somatic and is preferred there.
- **The one rule.** A rating scale measures severity, never diagnosis. A high score is a reason to interview.

WHAT EARNS THE MARKS

- **Measurement level answered first,** with the consequence for test choice stated.
- **Both rater classes named for depression,** with instruments under each.
- **Its known bias stated.** The somatic weighting reads as practice rather than recall.

SEE ALSO Slot I - 49 classifying rating scales and standardisation Slot I - 80 reliability and validity in study design

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

I - 53

Projective tests. [10]

10 PSYCHOMETRY AND ASSESSMENT RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	The hypothesis	Ambiguity forces the answer to come from within
5	The catalogue	Test, material, task, what is read from it
2	Clinical use	The referral questions they actually answer
1	Standing	One honest sentence on the evidence

TEST	MATERIAL	THE TASK	WHAT IS READ FROM IT
Rorschach	Ten symmetrical inkblots	Say what it might be, then show where	Perceptual organisation, thought process, reality testing
Thematic Apperception Test	Ambiguous interpersonal scenes	Tell a story with a past and an ending	Needs, external press, object relations
Children's Apperception Test	Animal or human scenes, ages three to ten	Tell a story	Childhood conflict around feeding, rivalry, control
Sentence completion	Incomplete written stems	Finish each stem with the first response	Attitudes to defined areas of life
Draw-a-Person	Blank paper and a pencil	Draw a person, then one of the other sex	Body image and self concept

Amber marks the two an examiner will follow up on. The materials are copyrighted and are described in words only.

- **The projective hypothesis.** The stimulus is unstructured and the scoring key is hidden, so the person has nothing external to lean on and the structure they impose is taken to reveal needs, conflicts and defences. Sampling is broad band, the whole personality rather than one trait.
- **What they are actually used for.** Where self-report is guarded or defended, where object-relational and motivational material is the referral question, and as one element of a battery in personality assessment. Never as a stand-alone diagnostic instrument.
- **Graded transparency.** Sentence completion is the most structured and most transparent, so the easiest to fake; the inkblot is the least. Word association, the oldest of the group, reads complexes from blocking and long latency.

PITFALL

Calling a projective test objective because it has a scoring system. The two families measure personality by opposite logic: ambiguous stimulus with inferential scoring against fixed-choice items with an empirical key. The reliability weakness follows from that logic and better scoring has not removed it.

WHAT EARNS THE MARKS

- **The hypothesis stated before any test is named.** A bare list is a mid-band answer.
- **Material and task given separately for each test.** That pairing is what the grid is for.
- **The referral question each one answers,** rather than a claim that they diagnose.
- **One honest line on the contested psychometrics.**

SEE ALSO Slot I - 48 projective tests classified and the Rorschach in detail Slot I - 45 the Thematic Apperception Test

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

AREA 06 OF 12 · P1-A04

Genetics in psychiatry

9	9.0%	I-54	I-62
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 HIGH 1 SINGLE 7

REVISION ORDER

The two mapping slots together, they are the same answer asked twice, and the association-study slot sits directly on top of them. Twin and adoption designs next, as a pair. The schizophrenia slot after that: it is the anchor here and it reuses the family-risk ladder. Epigenetics, the gene-environment slot and the genetic-correlation slot are independent and each is short.

WHAT IS IN THIS AREA

The methods in five slots: twin studies with the bipolar findings, adoption designs with schizophrenia, the gene-mapping strategies with their strengths and limits, gene mapping set against linkage and association, and the genome-wide association studies. Then the genetics of schizophrenia, epigenetics, gene and environment in neuropsychiatric disease, and the slot that runs genetic correlation and pleiotropy across substance use and psychiatric disorders.

I - 54

Genetics of Schizophrenia. [10]

10 GENETICS IN PSYCHIATRY KNRUHS, RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 The architecture	Highly heritable and highly polygenic, said in one line
3 Family, twin, adoption	The risk gradient, and what each design controls for
3 Molecular findings	Common variants, then the rare ones with big effects
2 The models	Two-hit and neurodevelopmental, and where they meet

THE LINE WORTH WRITING FIRST

Schizophrenia is among the most heritable of psychiatric disorders and among the most polygenic. Twin studies put heritability at about eighty per cent. Thousands of common variants of very small effect carry most of that, and a small number of rare copy number variants carry large effects in a few families.

RELATIONSHIP TO AN AFFECTED PERSON	GENES SHARED	APPROXIMATE LIFETIME RISK
No relation, general population	Baseline	About 1 in 100
Sibling	One half	About 9 in 100
Child of one affected parent	One half	About 13 in 100
Dizygotic co-twin	One half	About 17 in 100
Monozygotic co-twin	Essentially all	**About 48 in 100**

The pooled family study figures. The last two rows are the argument: both twin types share a womb and a household, only the genes differ, and the risk trebles. Note also that a monozygotic co-twin is far from certain to be affected, which is where environment enters.

- **What molecular work added.** The 2014 consortium study named 108 genome-wide significant loci, and later analyses several hundred. The strongest signal sits in the major histocompatibility complex on chromosome 6 and is driven by complement C4, which tags synaptic pruning. Rare copy number variants, the 22q11.2 deletion above all, carry far larger individual risk.
- **The two models to close with.** Two-hit: genetic vulnerability is the first hit, an environmental insult the second. Neurodevelopmental: early insults stay latent until adolescent synaptic pruning unmasks them. The C4 finding is where the two models meet.

WHAT EARNS THE MARKS

- **The monozygotic against dizygotic comparison stated as an argument,** not offered as two loose numbers.
- **Polygenic architecture named before any single gene.** A candidate-gene answer reads as dated.
- **Complement C4 tied to pruning.** It is the one molecular finding with a mechanism attached.

SEE ALSO Slot I - 58 twin studies in bipolar affective disorder Slot I - 60 adoption study designs SPRINT Day 3, Psychiatric Genetics

I - 55

Explain the genetic and environmental factors in neuropsychiatric diseases. [5+5]

5 + 5 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**The genetic side**	Complex and polygenic is the rule; name the exceptions
2	**Heritability, read correctly**	What the number is and is not about
3	**The environmental side**	Ordered by timing, prenatal to adult
2	**How the two meet**	Interaction, correlation, epigenetic mediation

CONDITION	GENETIC SIDE	ENVIRONMENTAL SIDE	WHAT THE PAIR SHOWS
Huntington disease	One dominant gene, a CAG repeat	Almost none	**Genes can be sufficient**
Schizophrenia	High heritability, polygenic	Obstetric events, cannabis, urban birth, migration	Neither side alone is sufficient
Depression	Moderate heritability	Childhood adversity, loss events, chronic illness	Environment carries more weight
Alcohol use disorder	Moderate heritability	Availability, price, peer group	**Policy can move the phenotype**

The amber cells are the two ends of the spectrum. Everything examined in psychiatry sits between a gene that is sufficient and an exposure that can be legislated.

- **Environment, ordered by timing.** Prenatal: maternal infection, famine, hypoxia, valproate. Perinatal: obstetric complications. Childhood: abuse, neglect, separation, urban upbringing, migration. Later: cannabis, stimulants, alcohol, head injury, social defeat.
- **Three ways genes and environment meet.** Interaction, where an exposure harms only the vulnerable. Correlation, where genes shape which exposures a person meets, passively, evocatively or by active choice. Mediation, where the exposure leaves an epigenetic mark on expression.

PITFALL

High heritability is not fixity. Heritability describes how much of the variance in one population at one time is genetic; it says nothing about how modifiable an individual is. Phenylketonuria is fully genetic and fully preventable by diet, and that one example settles the point.

WHAT EARNS THE MARKS

- **Complex inheritance named as the rule,** with Mendelian conditions given as the exception rather than the model.
- **Exposures ordered by timing,** which shows a developmental frame rather than a memorised list.
- **All three modes of interplay,** not interaction alone.
- **The heritability caveat.** It is the sentence that separates a top answer here.

SEE ALSO Slot I - 54 genetics of schizophrenia Slot I - 59 epigenetics in psychiatry **SPRINT** Day 3, Psychiatric Genetics

I - 56

What are Genome Wide Association Studies? Describe their contribution to understanding etiology of psychiatric disorders. [10]

10 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Hypothesis-free, common variants, cases against controls
3 How it is done	The four steps, and the threshold that guards them
3 What it has contributed	Architecture, shared risk, and one drug-target lead
2 What it cannot do	The honest limits, stated as limits

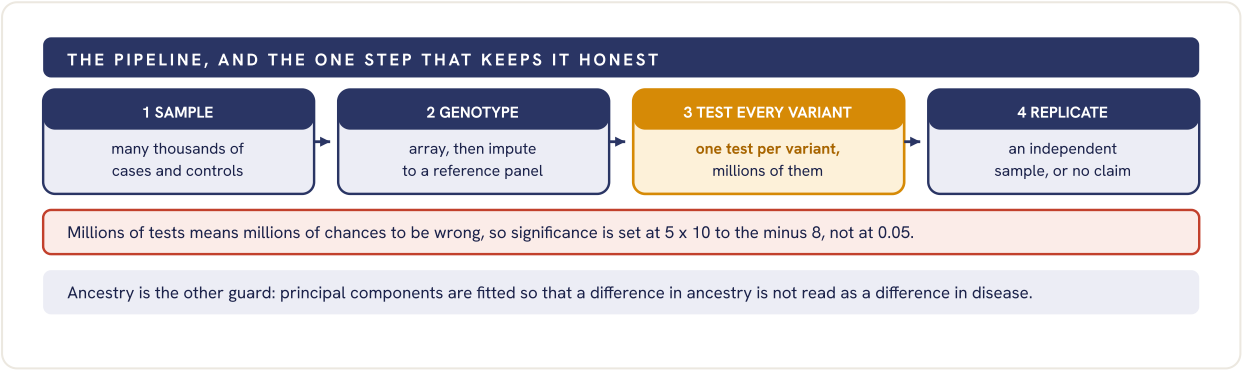


Fig 056.1 The four steps, with the two safeguards that make the output interpretable printed underneath rather than buried inside the method.

- **What it has contributed.** It settled the architecture: psychiatric disorders are highly polygenic, built from many common variants of tiny effect, not from a few broken genes. It showed substantial shared genetic risk across schizophrenia, bipolar disorder and depression. It produced polygenic risk scores, useful for research. In schizophrenia it delivered the complement C4 lead and pointed at calcium channel genes.
- **What it cannot do.** Each hit explains almost nothing on its own, and together they still leave heritability unaccounted for. The signal is a region, not a gene, because of linkage disequilibrium. Rare variants and copy number variants are invisible to it. Most samples are of European ancestry, so risk scores transfer poorly. Nothing here is a clinical test.

WHAT EARNS THE MARKS

- **Hypothesis-free stated explicitly,** and contrasted with the candidate-gene era it replaced.
- **The genome-wide threshold given with its reason.** Multiple testing, not convention.
- **Contribution framed as architecture, not as a gene list.** Polygenicity and cross-disorder overlap are the findings.
- **Limits named as limits,** including the ancestry bias, which is the one most candidates miss.

SEE ALSO Slot I - 61 gene mapping against linkage and association Slot I - 62 genetic correlation and pleiotropy

SPRINT Day 3, Psychiatric Genetics

I - 57

What are the different gene mapping strategies? Write down the basic idea, strength and limitations of the strategies. [3+2+2+3]

3 + 2 + 2 + 3 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The strategies	Name the axis, then the four families of method
2 Basic idea of each	One sentence per strategy, no more
2 Strengths	What each one is uniquely good at finding
3 Limitations	The failure mode of each, and the one they share

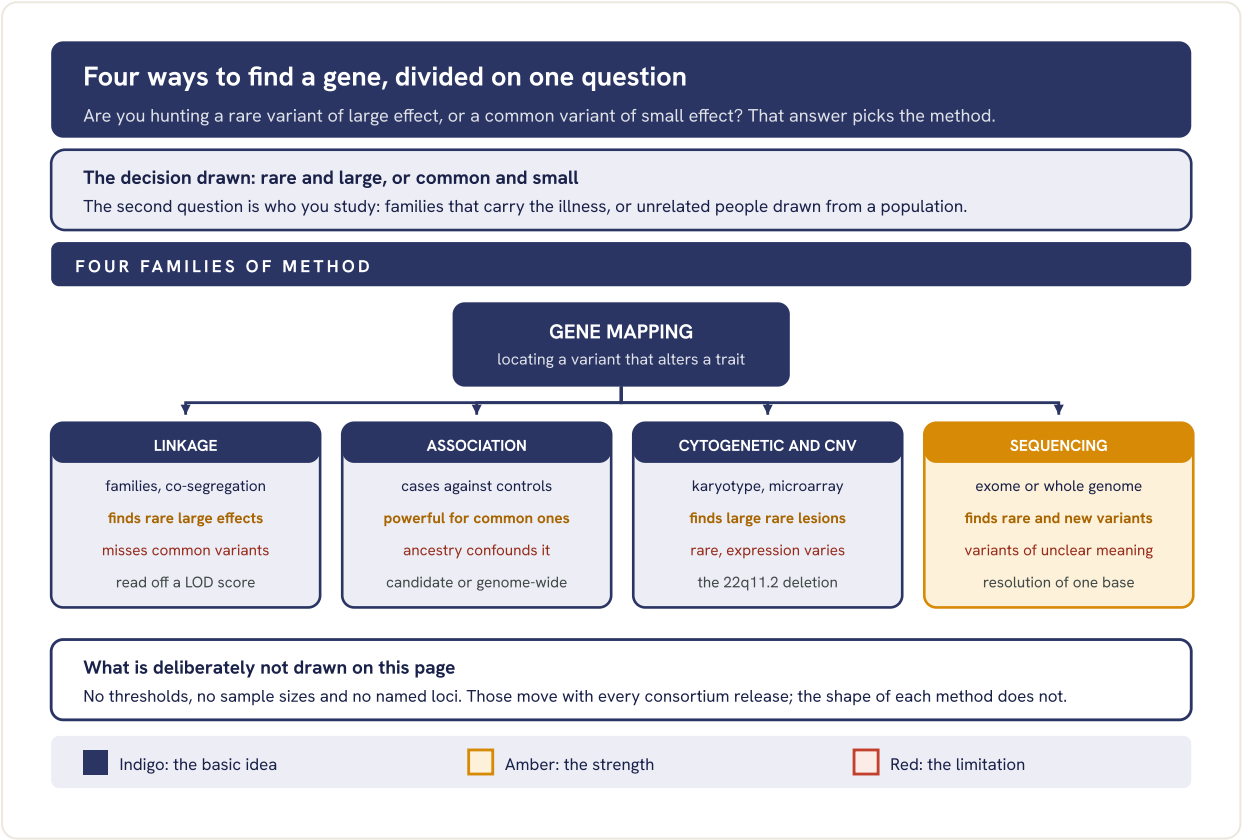


Fig 057.1 One axis, four branches, and inside each branch the same four lines in the same order, so the strategies can be read down the page as well as across it.

- **The basic ideas in one line each.** Linkage asks whether a marker travels with the illness through a family. Association asks whether an allele is commoner in cases than controls. Cytogenetics and microarray look for missing or duplicated stretches of chromosome. Sequencing reads the bases and compares them.
- **How they hand over to each other.** Linkage narrows a large region; association and sequencing resolve it to a variant. Practice today runs association genome-wide first, and keeps linkage for pedigrees with a striking inheritance pattern.

PITFALL

A genome-wide hit is not the gene. Linkage disequilibrium means the associated marker usually stands in for a nearby causal variant, and fine mapping and functional work are separate jobs that often fail. Saying so is the limitation mark that most answers leave on the table.

WHAT EARNS THE MARKS

- **The axis stated before the list.** Rare and large against common and small is what organises the four.
- **Idea, strength and limitation for every strategy,** in that order, because the stem asks for all three.
- **Linkage and association placed under gene mapping,** not offered as its alternatives.
- **The shared limitation.** A located region is not a mechanism.

SEE ALSO Slot I - 61 gene mapping against linkage and association Slot I - 56 genome wide association studies

SPRINT Day 3, Psychiatric Genetics

I - 58

Twin studies in Bipolar affective disorder. [10]

10 GENETICS IN PSYCHIATRY KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The twin method	Why two kinds of twin answer a genetic question
3	Design and assumptions	Zygosity, concordance, and the modelling that follows
3	What it shows in bipolar	The pattern, the heritability, and the spectrum finding
2	Limitations	The assumption that carries the whole method

THE METHOD IN TWO SENTENCES

Monozygotic twins share essentially all their segregating genes, dizygotic twins about half, and both kinds share a womb and a household. So any excess of monozygotic over dizygotic concordance is attributed to genes, provided the two kinds of twin really do share their environment equally.

ELEMENT	WHAT IT MEANS	WHAT IT DELIVERS	WHERE IT IS CHALLENGED
Zygosity	Established by genotyping, not by looks	The two comparison groups	Misclassification dilutes the estimate
Probandwise concordance	Counts each affected twin as a proband	The figure comparable with population risk	**Pairwise counting gives a lower number for the same data**
Equal environments	Both twin types share family environment equally	Makes the excess interpretable as genetic	**Identical twins are treated and treat each other more alike**
Variance modelling	Splits variance into genes, shared and unique environment	The heritability estimate	Assumes no gene-environment interaction

Single concordance percentages are not printed here on purpose: they swing with how wide the diagnostic boundary is drawn and with how the pairs were counted. Quote the pattern and the assumption, and you cannot be caught out on the number.

- **What twin studies show in bipolar disorder.** Monozygotic concordance is several times dizygotic concordance, and heritability estimates sit around eighty per cent, among the highest in psychiatry. Concordance is nowhere near complete, so environment and chance still matter.
- **The finding examiners want beyond the numbers.** Co-twins who are not bipolar are still at raised risk of unipolar depression and of other affective illness, which is evidence that what is inherited is a broad affective liability rather than a diagnosis.

WHAT EARNS THE MARKS

- **The logic of the method before any figure.** Two twin types, one difference, one inference.
- **The equal environments assumption named,** and named as the method's weak point.
- **Incomplete concordance used as an argument,** not treated as a failure of the genetic case.
- **The broadened phenotype in co-twins,** which is what makes twin work informative about nosology.

SEE ALSO Slot I - 54 genetics of schizophrenia Slot I - 60 adoption study designs SPRINT

Day 3, Psychiatric Genetics

I - 59

Define epigenetics. Discuss its contribution and implications in Psychiatry. [2+4+4]

2 + 4 + 4 | GENETICS IN PSYCHIATRY | DNB, MUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	Change in expression without change in sequence
4 Mechanisms	Three marks, each with its direction of effect
2 Contribution	What it explains that genetics alone could not
2 Implications and limits	Targets and biomarkers, and why to be careful

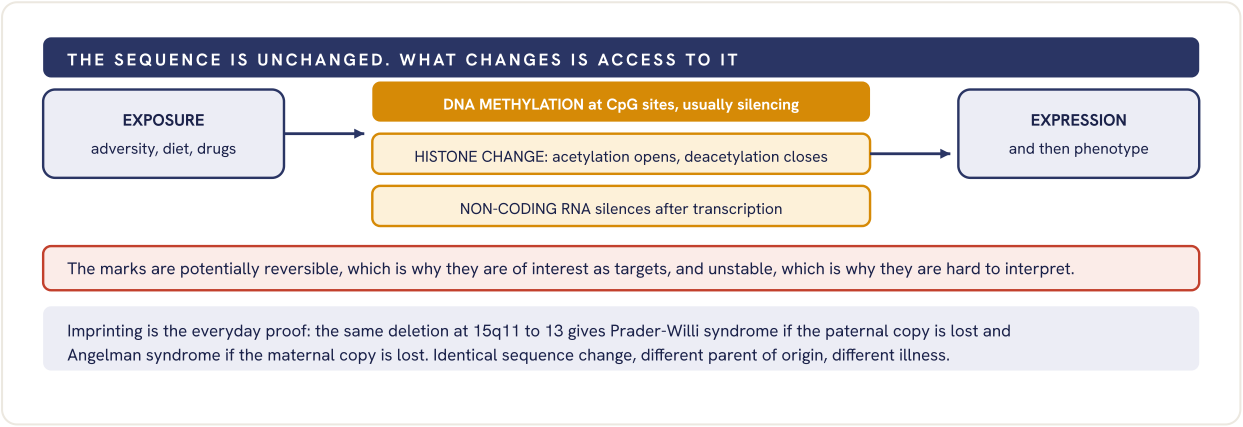


Fig 059.1 The three marks drawn as one layer between exposure and expression, with imprinting placed beneath as the demonstration that parental origin alone can change a phenotype.

- **Contribution to psychiatry.** It supplies a mechanism for gene-environment interplay, so early adversity can leave a lasting mark on stress-axis gene expression. It helps explain why monozygotic twins are discordant. Valproate is a histone deacetylase inhibitor, and folate and vitamin B12 feed the methyl supply, so the mechanism already touches prescribing.
- **The limits to state plainly.** Most human work measures blood, and blood is not brain. Direction of causation is rarely established: illness and its treatment change methylation too. Findings are strongly tissue and cell specific, and few replicate. No epigenetic test is in clinical use in psychiatry.

WHAT EARNS THE MARKS

- **The definition tied to sequence.** Expression changes, sequence does not, and heritable through cell division.
- **All three mechanisms, with a direction each.** Methylation alone is the commonest half answer.
- **One worked example.** Imprinting at 15q11 to 13 is the cleanest one available.
- **Reversibility named as the reason for interest,** and instability named as the reason for caution.

SEE ALSO Slot I - 55 genetic and environmental factors Slot I - 54 genetics of schizophrenia **SPRINT** Day 3, Psychiatric Genetics

I - 60

What do you understand by adoption studies? Describe different designs of adoption studies with special reference to Schizophrenia. [3+7]

3 + 7 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 What they are	The one thing adoption separates that twins cannot
4 The four designs	Who is sampled first decides which design it is
3 Findings and limits	What schizophrenia work established, and what to concede

DESIGN	WHO IS SAMPLED FIRST	WHAT IT SEPARATES	WHAT SCHIZOPHRENIA WORK SHOWED
Adoptee study	Adopted-away children of affected biological parents	Genes from rearing, prospectively	**Raised rates despite rearing away from the affected parent**
Adoptee's family method	Adopted people who are already ill	Biological relatives from adoptive relatives	**Excess illness in biological relatives only**
Cross-fostering	Children of well parents reared by affected adoptive parents	The effect of rearing on its own	Being reared by an affected parent did not raise risk
Adoptive family study	High-risk and low-risk adoptees, with the adoptive family assessed	Interaction between the two	High genetic risk mattered mainly in disturbed adoptive families

The amber rows are the two designs that carry the genetic claim, from opposite ends. The fourth put interaction back into a literature that had been read as proof of genes alone.

- **What adoption adds that twins cannot.** A twin pair shares its rearing, so a twin study cannot fully separate genes from family environment. Adoption breaks the two apart: the child carries the genes of one family and grows up in another.
- **The spectrum finding.** Biological relatives of adopted people with schizophrenia showed an excess not only of schizophrenia but of schizotypal and paranoid presentations. That is the evidence base for the idea of a schizophrenia spectrum, and it came from adoption work.
- **Limitations to concede.** Placement is not random and adoptive homes are screened, which narrows the environmental range. Late placement leaves early rearing shared. The biological mother still supplies the prenatal environment. Numbers are small, and adoption itself selects an unusual population.

WHAT EARNS THE MARKS

- **The comparison with twin studies made explicit,** since it is the reason the design exists.
- **All four designs named and told apart by who is sampled first.**
- **The spectrum finding credited to adoption work,** which is the "special reference to schizophrenia" the stem is asking for.
- **The prenatal environment named among the limits.** It is the one candidates most often miss.

SEE ALSO Slot I - 54 genetics of schizophrenia Slot I - 58 twin studies in bipolar affective disorder SPRINT Day 3, Psychiatric Genetics

I - 61

What is gene mapping? How is it different from linkage analysis and association studies? [3+7]

3 + 7 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Gene mapping defined	The umbrella, with the other two placed under it
4 The grid	Domain by domain, with the discriminators marked
3 Overlap and what follows	Where the two methods meet, and how they are sequenced

DOMAIN	LINKAGE ANALYSIS	ASSOCIATION STUDY	WHAT SETTLES IT
Who is studied	Families with several affected members	Unrelated cases and controls, or trios	**Related people or strangers**
What is tracked	Co-segregation of a marker with illness within a family	Allele frequency difference between groups	**Within a pedigree or across a population**
Distance it works over	Long, tens of millions of bases	Very short, the span of linkage disequilibrium	**Resolution: a region against a variant**
Statistic	The LOD score	A p value against a genome-wide threshold	Different traditions, same logic
Best suited to	Rare variants of large effect	Common variants of small effect	**Effect size and allele frequency**
Main failure mode	Underpowered for common small effects	Confounded by population stratification	Each fails where the other works

Amber cells are the discriminators. Each reduces to the same pair of questions: whose DNA is compared, and how big is the effect being hunted.

- **The overlap.** Both are gene mapping, and family-based association tests sit in between: the transmission disequilibrium test compares the alleles a parent passes on with the alleles the same parent does not, so it is an association test that cannot be confounded by ancestry.
- **What follows from the difference.** Linkage narrows the search to a region; association resolves the region to a variant; sequencing reads it. They are stages, not rivals, and saying so answers the stem's hidden question.

PITFALL

The stem invites you to treat all three as parallel alternatives. They are not. Gene mapping is the goal; linkage and association are two ways of reaching it. Opening with that sentence earns the first three marks before any comparison begins.

WHAT EARNS THE MARKS

- **Gene mapping defined as the umbrella,** in the first line, not implied by the layout.
- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **The discriminators identified as discriminators,** rather than a flat list of six differences.
- **The family-based association test offered as the overlap,** which is the depth mark on this question.

SEE ALSO Slot I - 57 gene mapping strategies Slot I - 56 genome wide association studies SPRINT Day 3, Psychiatric Genetics

I - 62

Genetic correlation, pleiotropy, and associations between substance use and psychiatric disorders. [10]

10 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The two terms	Defined apart, because they are not the same claim
2	How they are measured	Twin designs first, then summary-statistic methods
3	Substance use findings	What overlaps with what, and the measurement caveat
2	What it does not prove	Correlation, direction, and how direction is tested

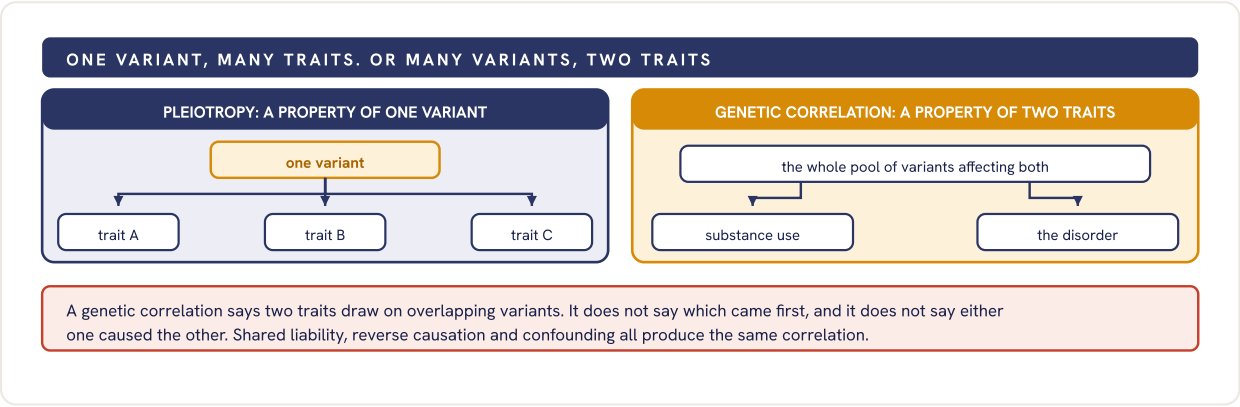


Fig 062.1 The two constructs drawn side by side because they are routinely merged: pleiotropy is a statement about one variant, genetic correlation a statement about two traits.

- **How they are measured, and what is found.** Twin designs give cross-twin cross-trait correlations; modern estimates come from genome-wide summary statistics by linkage disequilibrium score regression. Substantial genetic correlation runs across schizophrenia, bipolar disorder, depression and ADHD, and substance use traits correlate with all of them, most strongly with ADHD and with depression.
- **The measurement caveat, and the causal test.** How much a person drinks and whether the drinking has become a disorder are genetically different traits that correlate differently with psychiatric illness, so say which you mean. For direction the tool is Mendelian randomisation, run both ways between cannabis and schizophrenia.

PITFALL

Do not use the two terms as synonyms. Separate horizontal pleiotropy, one variant affecting two traits independently, from vertical, acting on the second only through the first. Mendelian randomisation is valid only under the horizontal assumption.

WHAT EARNS THE MARKS

- **Two separate definitions,** one about a variant and one about a pair of traits.
- **Horizontal separated from vertical pleiotropy,** which is the discriminator this question is built around.
- **Consumption distinguished from disorder** when reporting any substance use finding.
- **Causation addressed explicitly,** with Mendelian randomisation named as the method that tests it.

SEE ALSO Slot I - 56 genome wide association studies Slot I - 54 genetics of schizophrenia SPRINT Day 3, Psychiatric Genetics

AREA 07 OF 12 · P1-A08

Social, cross-cultural and evolutionary psychology

8	7.8%	I-63	I-70
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 7

REVISION ORDER

Build one culture answer properly and four of the eight slots open. Take the definitional slot first and the culture-bound syndromes last of the four, because the syndromes are a list and the other three are an argument. The social theories of suicide next, it is the one that needs its own reading. Religion, marriage and imprinting are independent and all three are short.

WHAT IS IN THIS AREA

Culture in four slots: its role in psychiatry, the definition with illness behaviour and symptomatology, the culture-bound syndromes with five Indian ones and the cultural theories of mental illness, and the slot that runs culture through expression, assessment and treatment and adds immigration. Then the social theories of suicide, religion and psychiatry, marriage as an institution, and imprinting, which is the one ethology slot in this book.

I - 63

What is culture bound syndrome? Describe any five Indian culture bound syndrome. Discuss different theories of mental illness from cultural perspective. [1+5+4]

1 + 5 + 4 | SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1	Definition	The term, and what DSM-5 put in its place
5	Five Indian	Each with its region and its presentation
3	The theories	Etic against emic, and the category fallacy
1	The caution	Where the concept is criticised

THE DEFINITION

A recurrent, locality-specific pattern of aberrant behaviour and distressing experience that is recognised as an illness within its own culture and often has no direct counterpart in the international classifications.

SYNDROME	WHERE	THE PRESENTATION	BEST UNDERSTOOD AS
Dhat	North and west India, South Asia widely	Fatigue, weakness and anxiety attributed to semen passed in urine	**An idiom of distress; usually depression or anxiety**
Koro	Epidemics in Assam and West Bengal	Acute terror that the genitals are retracting, with fear of death	Acute anxiety, spread by contagion
Possession states	Across India, more often in women	Trance, altered identity, a spirit speaking through the person	Dissociative trance and possession disorder
Ascetic syndrome	Described in India by Neki	Adolescent, usually male: withdrawal, severe religiosity, self-imposed austerity	Often the prodrome of schizophrenia
Gilhari syndrome	Rajasthan	A swelling believed to move under the skin and to kill on reaching the neck	A culturally shaped conversion presentation

- **The theories, which are the last four marks.** The universalist or etic position holds disorders to be the same everywhere, with culture only colouring them. The relativist or emic position holds categories to be meaningful only inside the culture that made them. Kleinman named the category fallacy, the error of carrying a category built in one culture into another where it has no coherence, and set the patient's explanatory model against the professional one.
- **What DSM-5 did to the term.** It retired culture-bound syndrome and put three things in its place: cultural syndromes, cultural idioms of distress, and cultural explanations of distress. Saying so is worth a mark.

WHAT EARNS THE MARKS

- **Exactly five, each with a region and a presentation.** The stem says five, so a sixth earns nothing and a fourth loses a mark.
- **Etic and emic named as positions,** not described as attitudes.
- **The category fallacy, attributed.** It is the single highest-value term in the last section.

SEE ALSO Slot I - 70 culture, illness behaviour and symptomatology

Slot I - 67 the role of culture across the clinical pathway **SPRINT** Day 5, social, learning and personality psychology

I - 64

Concept of imprinting in ethology. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The definition**	Rapid, timed, unrewarded, lasting
3 **The classic work**	Lorenz, the geese, and the window
3 **The two kinds**	Filial and sexual, and their separate windows
2 **Use in psychiatry**	The line from ethology to attachment

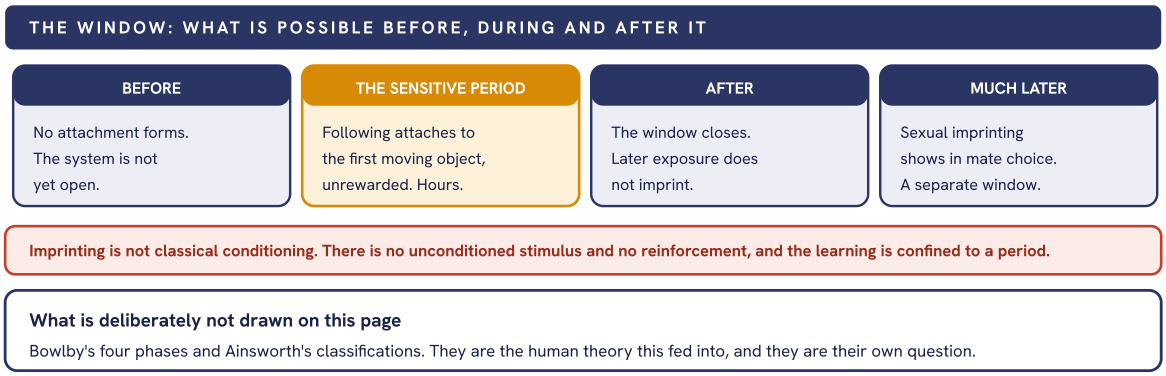


Fig 064.1 Imprinting drawn as a window rather than a process. Amber marks the sensitive period itself, which is the claim the concept stands or falls on; the fourth panel is the separate, much later window for sexual imprinting.

- **The definition, with its authority.** A rapid form of learning, confined to a sensitive period early in life, by which a young animal forms an attachment to a moving object, usually the parent, without reward and with lasting effect. Konrad Lorenz established it in greylag geese from 1935, the observation having been made by Spalding in the previous century. Lorenz shared the 1973 Nobel Prize with Tinbergen and von Frisch.
- **Why psychiatry keeps it.** It supplied the biological argument Bowlby used: attachment as a species-typical behavioural system with its own sensitive period, not a by-product of feeding. Harlow's monkeys made the same point from the other side, preferring the cloth mother to the one that fed them.

WHAT EARNS THE MARKS

- **All four defining features.** Rapid, period-bound, without reinforcement, and persistent.
- **Lorenz and the geese, with the year.** The classic experiment is the answer's evidence.
- **Filial and sexual imprinting separated,** with their windows named as separate.
- **The bridge to Bowlby.** On a psychiatry paper an ethology answer that never reaches attachment is incomplete.

SEE ALSO Slot I - 89 attachment theory SPRINT Day 5, social, learning and personality psychology

I - 65

Critically evaluate marriage as an institution. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The institution	What marriage is for, stated as functions
4	The case both ways	Domain by domain, for and against
2	The evidence	What the mental health data support
2	The verdict	A position, defended, not a fence

DOMAIN	THE CASE FOR	THE CASE AGAINST	WHAT THE EVIDENCE SHOWS
Mental health	Less psychiatric morbidity and lower suicide rates among the married	A bad marriage is worse than none	**Protection is larger for men; selection and causation both operate**
Social function	Legitimises children, transmits property, organises care of the old and ill	Concentrates unpaid care on women	The function is real, its distribution is contested
Support in illness	A confiding relationship buffers against depression	The spouse can be the source of the stress	A confidant protects; marriage does not guarantee one
Indian context	Near universal; family support is built in	Dowry, early marriage and domestic violence carry direct harm	Both hold at once and belong in one answer
Stability	Divorce is uncommon	Low divorce is not high satisfaction	Stability and quality are separate measures

The fourth column is what makes this a critical evaluation rather than a list. Amber marks the finding the verdict rests on.

- **The verdict, which is what the verb asks for.** Marriage is associated with better mental health; the association is stronger for men than for women; it is partly selection rather than protection; and the quality of the marriage predicts more than the fact of it. Commit to that and defend it, rather than balancing two lists and stopping.
- **The psychiatric handles.** Marital status is a standard variable in suicide and depression epidemiology, marital discord is a recognised focus of clinical attention, and couple therapy is an evidence-based treatment for depression where discord maintains it.

WHAT EARNS THE MARKS

- **A stated verdict.** Critically evaluate is marked on whether a position was taken and defended.
- **Both sides of at least four domains,** which is what stops the answer becoming an opinion.
- **Selection distinguished from protection.** It is the methodological point that lifts the answer.
- **The Indian material named,** not implied by the setting.

SEE ALSO Slot I - 66 social theories of suicide SPRINT Day 5, social, learning and personality psychology

I - 66

Social theories of suicide. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The claim	Suicide rates as social facts
4	Durkheim's four	Two axes, each failing in two directions
2	After Durkheim	Status integration, the critique, contagion
2	The limit	Rates against individuals

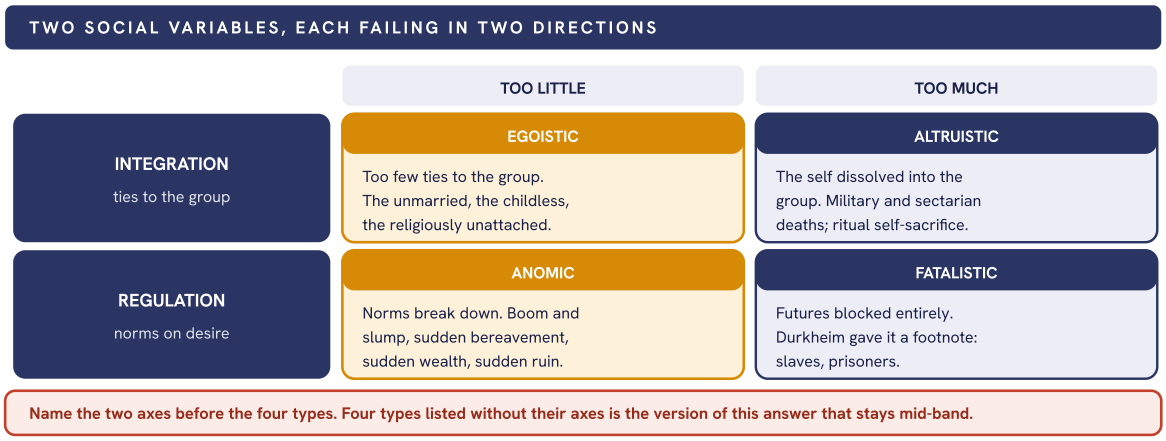


Fig 066.1 Durkheim's typology drawn as one grid: two social variables down the side, too little and too much across the top. Amber marks the two types that carry most of the modern evidence.

AFTER DURKHEIM	THE CLAIM	WHOSE
Status integration	Rates fall where a person's statuses are compatible and role conflict is low	Gibbs and Martin
The statistics critique	Official suicide rates are produced by coroners, so they are social constructions rather than raw facts	Douglas, 1967
Ecological studies	Suicide clusters in socially disorganised districts, whoever lives in them	Sainsbury, 1955
Media contagion	Reported suicides are followed by a rise, the Werther effect; responsible reporting reverses it, the Papageno effect	Phillips, 1974; Niederkrotenthaler, 2010

- **The limit, which is the last two marks.** Social theories explain rates, not individuals. They cannot say which patient will die, and an answer that offers Durkheim as a clinical risk assessment has mistaken the level of explanation.

WHAT EARNS THE MARKS

- **The two axes named before the four types,** and fatalistic included, since it is the type most often dropped.
- **At least two theories after 1897,** with their authors, since the stem says theories rather than Durkheim.
- **The level-of-explanation limit,** stated rather than implied.

SEE ALSO Slot I - 65 marriage as an institution SPRINT Day 5, social, learning and personality psychology

I - 67

Role of culture in Psychiatry: [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 The claim	Culture at every step, not only at diagnosis
4 The pathway	Each step, with its clinical consequence
2 The instrument	The cultural formulation, and what it asks
2 The two errors	Category fallacy and stereotyping

THE CLAIM TO OPEN WITH

Culture enters at every step of the clinical pathway, not only at the point of diagnosis. It shapes what counts as illness, how distress is spoken, who is consulted first, what treatment is accepted, and what outcome follows.

STEP	WHAT CULTURE DOES THERE	THE CLINICAL CONSEQUENCE
Aetiology	Alters exposure to risk and protection: family structure, migration, discrimination, drinking norms	Risk profiles differ between populations
Expression	Supplies the idioms of distress, and the content of delusion and hallucination	Somatic presentation is the rule in much of Indian practice
Help-seeking	Sets the pathway: faith healer, family elder, general practitioner, then psychiatrist	Long untreated intervals; the first contact is rarely psychiatric
Diagnosis	Decides whether a behaviour reads as normal, deviant or ill	**Risk of category fallacy; the Cultural Formulation Interview exists to check it**
Treatment	Governs adherence, and who makes the decision	Consent and disclosure are family matters more often than individual ones
Outcome	Shapes stigma, family tolerance, and re-entry to work and marriage	The WHO comparative studies reported a better course in developing-country centres

- **The instrument to name.** DSM-5 carries a Cultural Formulation Interview: the cultural definition of the problem, its perceived causes, supports and stressors, cultural factors in coping and past help-seeking, and factors in current help-seeking including the clinician relationship. Its predecessor is the DSM-IV Outline for Cultural Formulation.
- **Two errors, named.** The category fallacy is applying a category built in one culture where it has no coherence. Cultural stereotyping is reading a person as a specimen of a culture instead of asking what this person believes. The second is the commoner clinical error.

WHAT EARNS THE MARKS

- **A pathway, not a list of topics.** The structure is what turns six observations into an argument.
- **The formulation instrument named,** with at least three of the domains it asks about.
- **Both errors, kept apart.** One is a nosological error, the other is an interviewing error.

SEE ALSO Slot I - 69 cultural factors in expression, assessment and treatment Slot I - 63 culture-bound syndromes

SPRINT Day 5, social, learning and personality psychology

I - 68

Religion and Psychiatry. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The two frames	Religion as a variable, and as a context
4	The evidence	Association, coping, and the boundary tests
2	The boundary	When belief becomes a clinical concern
2	The Indian setting	Faith healing, and how to work with it

DOMAIN	WHAT IS ESTABLISHED	WHAT TO DO WITH IT
Religiosity and outcome	Religious involvement is associated with less depression, less substance use and lower suicide rates; the effects are modest and confounded	Ask about it as a resource, not only as a symptom
Religious coping	Pargament separated positive coping, collaborative and benevolent, from negative coping, punishing God and spiritual struggle	**Negative coping predicts worse outcome; the kind matters, not the amount**
Belief against delusion	A belief is not delusional because it is religious; the test is whether the person's own community shares it and holds it in that manner	The community is the reference, not the clinician
Obsession against devotion	Religious obsessions are intrusive, resisted and unwanted; devout practice is none of these	Ask about resistance and distress, not content
Possession and trance	Classified as dissociative states when involuntary, distressing and outside accepted ritual	Involuntary and unwanted is the threshold

- **The two frames to name at the start.** Religion as a variable, studied for its association with outcome, and religion as a context, which sets what a community counts as normal. An answer with only the first is epidemiology; an answer with only the second is anthropology.
- **The error runs in both directions.** Reading every religious experience as pathology, and refusing to read any of it as pathology. Both substitute a prior position for an assessment.

THE INDIAN SETTING

Temple and dargah healing is a common first contact and often runs in parallel with treatment. Collaboration is more useful than confrontation: attacking the belief system costs the alliance and the adherence, and the family usually holds both frames at once without difficulty.

WHAT EARNS THE MARKS

- **The two frames separated in the opening lines.** It is what organises everything after.
- **The community test for delusion,** stated as a test rather than as a caution.
- **Positive and negative religious coping distinguished.** It is the finding examiners look for.

SEE ALSO Slot I - 67 the role of culture across the clinical pathway SPRINT

Day 5, social, learning and personality psychology

I - 69

Discuss in detail cultural factors involved in expression, assessment and treatment of mental illness in an individual. Add a note on immigration and mental health. [10]

10 | ASKED AT 15 MARKS | SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Expression	Idioms of distress, and what culture supplies
3	Assessment	Whose normality, and the formulation instrument
3	Treatment	Explanatory models, family, and adherence
2	Immigration	The excess, and the mechanism proposed

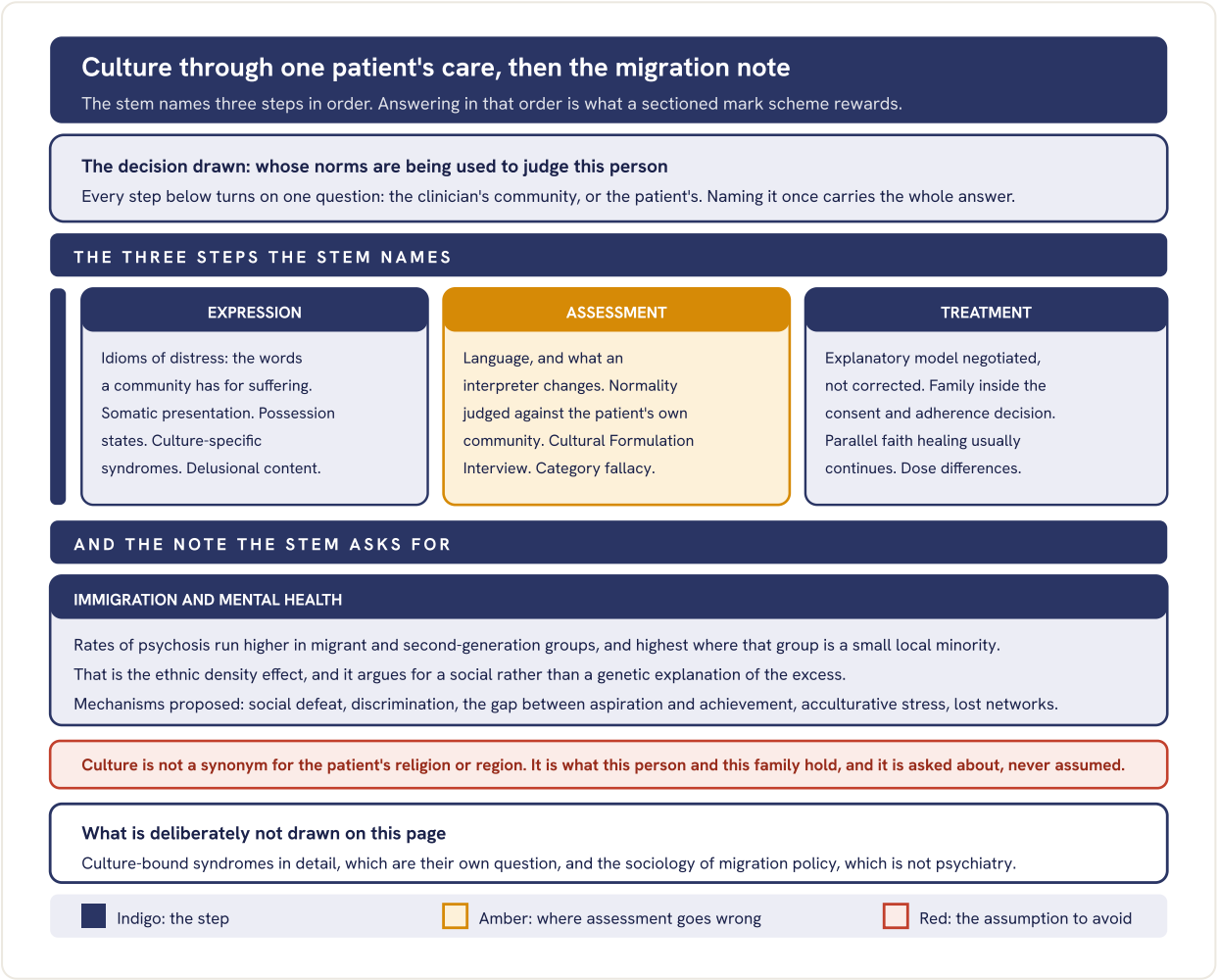


Fig 069.1 The three steps in the stem's own order along one spine, with the migration note as its own band below. Amber marks assessment, where the category fallacy is made.

- **Answer in the stem's order.** Expression, assessment, treatment, then the note. A fifteen-mark version of this question is marked by section, so a well-informed answer in the wrong order still loses marks it has earned.
- **The ethnic density effect, stated carefully.** Rates rise as the proportion of a migrant's own group in the local population falls. It is among the more robust findings in social psychiatry, and it is the sentence that turns a list of migration stressors into an argument.

WHAT EARNS THE MARKS

- **Three sections in the stem's order,** each headed, because the mark scheme is sectioned.
- **The formulation instrument named at the assessment step,** which is where its marks sit.
- **Migration answered as a mechanism,** not as a list of hardships.

SEE ALSO Slot I - 67 the role of culture across the clinical pathway

Slot I - 70 culture, illness behaviour and symptomatology **SPRINT** Day 5, social, learning and personality psychology

I - 70

Define culture. Describe the role of culture in shaping illness behaviour and symptomatology in mental disorders. [2+8]

2 + 8 | SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Define culture**	Learned, shared, transmitted, changing
4	**How culture acts**	The six effects, named as mechanisms
2	**Illness behaviour**	Mechanic and Parsons, and what each adds
2	**Symptomatology**	What is shaped, and what is not

THE DEFINITION

Culture is the shared, learned and transmitted system of meanings, values and practices by which a group interprets experience. It changes, and it is not inherited.

EFFECT	WHAT CULTURE DOES TO THE DISORDER	EXAMPLE
Pathogenic	Culture causes the disorder by supplying the belief	**Koro and dhat: the belief is the illness**
Pathoselective	Culture selects which reaction is chosen under stress	Amok; family suicide in some societies
Pathoplastic	Culture shapes the content of a universal disorder	Delusional content: technology or spirits
Pathoelaborating	Culture intensifies a universal behaviour into an extreme form	Trance elaborated through ritual
Pathofacilitative	Culture raises the frequency without changing the form	Alcohol use disorders where drinking is normative
Pathoreactive	Culture governs the reaction to the illness and what it is called	Stigma; a psychosis named as possession

The six are Tseng's. Amber marks the only row where culture is a cause, not a shaper.

- **Illness behaviour, defined and attributed.** Mechanic's term for the way symptoms are perceived, evaluated and acted upon, which varies between cultures independently of the disease. Parsons' sick role sets what a society exempts the sick person from, and what it asks in return: to seek help, and to want to get well.
- **The eight marks are the mechanisms, not the examples.** Name the effect, then attach the example. An answer built from colourful syndromes with no typology behind them stays mid-band however many it lists.

WHAT EARNS THE MARKS

- **A definition with all four properties.** Learned, shared, transmitted, changing. Two marks in one sentence.
- **The effects named as effects,** in the technical vocabulary, with one example each.
- **Illness behaviour attributed to Mechanic,** separated from the sick role, and one line on what culture does not change.

SEE ALSO Slot I - 63 culture-bound syndromes Slot I - 69 cultural factors in expression, assessment and treatment

SPRINT Day 5, social, learning and personality psychology

AREA 08 OF 12 · P1-A11

Biostatistics and epidemiology

8	7.6%	I-71	I-78
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 RECURRENT 1 SINGLE 6

REVISION ORDER

The two selector slots first and together: they open the other three, and the paired and unpaired t-tests belong inside them. Chi square next, it is the anchor here and it is short. Analysis of variance after that. The two odds-ratio slots are one pair and should be prepared as one. Regression stands alone and is the longest here.

WHAT IS IN THIS AREA

The tests of significance in five slots: the selector question twice, once opened out on the paired test and once on the unpaired, then chi square, the t-test and analysis of variance each alone. The odds ratio in two, once against relative risk and once with confounding, effect modification and mediation. Regression analysis takes the eighth.

I - 71

Chi Square test. [10]

10 BIOSTATISTICS AND EPIDEMIOLOGY

KNRUHS, KUHS, NTRUHS, RGUHS - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2 What it is	Observed against expected, for categorical data
4 The working	Expected counts, the statistic, the degrees of freedom
2 Assumptions	And the exact test that replaces it when they fail
2 Its three uses	Association, goodness of fit, homogeneity

THE DEFINITION AND THE FORMULA

A non-parametric test of whether two categorical variables are associated, built entirely on counts. The statistic is the sum over all cells of observed minus expected, squared, divided by expected. Each expected count is its row total times its column total, divided by the grand total, and the degrees of freedom are rows minus one times columns minus one.

CELL, ILLUSTRATIVE COUNTS	OBSERVED	EXPECTED	CONTRIBUTION
Adherent, relapsed	20	30	3.33
Adherent, no relapse	80	70	1.43
Not adherent, relapsed	40	30	3.33
Not adherent, no relapse	60	70	1.43
Total	200	200	Chi square 9.52, one degree of freedom

Made-up counts, arithmetic real. Row totals 100 and 100, column totals 60 and 140, so every expected count is 30 or 70. At one degree of freedom the statistic clears both the 3.84 cut at five per cent and the 6.63 cut at one per cent.

- **Assumptions.** Independent observations, raw counts and never percentages, and adequate expected counts: in a two by two no expected cell below five. When that fails, use Fisher's exact test. Paired categorical data need McNemar's test, not this one.
- **Its three uses.** Test of association between two categorical variables; goodness of fit against an expected distribution; and test of homogeneity of a proportion across several groups.
- **What it does not tell you.** A significant result says the variables are associated, not how strongly and not in which direction. Report an effect measure, an odds ratio or a relative risk, alongside it.

WHAT EARNS THE MARKS

- **The expected-count formula written out,** not just the chi square formula.
- **Degrees of freedom derived from the table shape,** with the number stated.
- **The expected-count-below-five rule with Fisher's exact test as the remedy.**
- **The three uses separated,** and McNemar named for paired data.

SEE ALSO Slot I - 74 tests of significance Slot I - 78 odds ratio and relative risk SPRINT

Day 7, research methods, biostatistics and epidemiology

I - 72

Enumerate various statistical tests used to measure the differences. Describe the paired t-test in detail with an example. Describe any two non-parametric tests used for group difference. [4+2+4]

4 + 2 + 4 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	The enumeration	Drawn as a selector, by data type and pairing
3	Paired t-test	What is tested, the statistic, an example
2	Two non-parametric	Mann-Whitney and Wilcoxon, and what each replaces
1	The choosing rule	Pairing first, then distribution

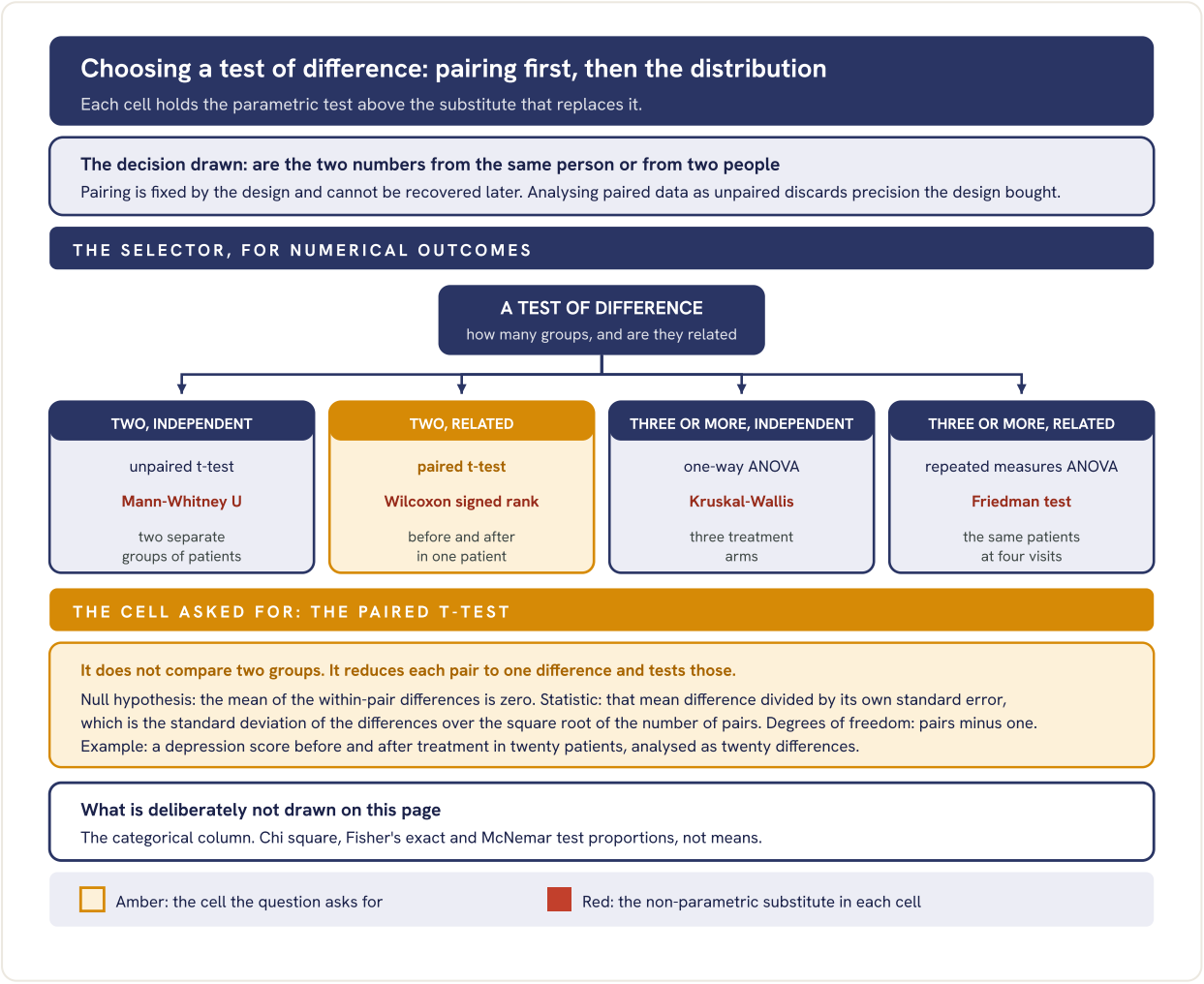


Fig 072.1 The difference tests as a two-way selector, groups against pairing, with the paired cell opened out below.

- **The two non-parametric tests.** Mann-Whitney U pools the observations, ranks them, and asks whether high ranks cluster in one group; it replaces the unpaired t-test. Wilcoxon signed rank ranks the absolute within-pair differences and compares positive against negative rank sums; it replaces the paired t-test.
- **The choosing rule.** Settle pairing from the design first, then distribution. Non-parametric tests need no normality, cost a little power when it does hold, and answer a question about ranks rather than means.

WHAT EARNS THE MARKS

- **The enumeration drawn as a selector,** so each test arrives with the situation it belongs to.
- **The paired test described as a one-sample test on differences,** which is what it actually is.
- **Degrees of freedom given as pairs minus one,** not as total observations minus two.
- **Each non-parametric test matched to the parametric one it replaces.**

SEE ALSO Slot I - 76 the unpaired t-test worked through Slot I - 77 analysis of variance **SPRINT** Day 7, research methods, biostatistics and epidemiology

I - 73

What is odd's ratio? Write briefly about confounding, effect modification and mediation. [4+2+2+2]

4 + 2 + 2 + 2 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	Odds ratio	Definition, formula, design, interpretation
2	Confounding	A distortion, and how it is removed
2	Effect modification	A real finding, reported not removed
2	Mediation	On the causal path, so never adjusted away

THE DEFINITION

The odds ratio is the odds of the outcome among the exposed divided by the odds among the unexposed, computed from a two by two table as a times d over b times c. One means no association, above one a positive association, below one a protective one. It is the measure a case-control study can produce, because such a study fixes the number of cases and so cannot yield a risk. Read it with its confidence interval: an interval containing one is not a significant result.

QUESTION	CONFOUNDING	EFFECT MODIFICATION	MEDIATION
Where it sits	Beside the exposure, linked to both exposure and outcome	Beside the exposure, changing how strongly it acts	**On the causal path, between exposure and outcome**
What it does	Distorts the estimate, up or down	Splits one true estimate into two real ones	Carries part of the true effect
How you find it	The estimate moves when you adjust for it	**The stratum estimates differ from each other**	The effect shrinks when you adjust, and that is expected
What you do	Remove it: restrict, match, randomise, or adjust	Report it: give the estimate stratum by stratum	**Do not adjust it away if the total effect is the question**

Amber marks the discriminators. One question separates all three: is the third variable on the causal path, beside it, or changing its strength.

- **The one-line test.** Adjusting for a confounder corrects the answer. Adjusting for a mediator destroys it. Adjusting for an effect modifier hides the finding, because the finding is that the effect differs.
- **Where it goes wrong.** Randomisation controls confounding by variables you never measured, which is its whole advantage; adjustment controls only what you thought to record. Effect modification is often reported as a subgroup result without a formal interaction test, which is how spurious subgroup claims enter the literature.

WHAT EARNS THE MARKS

- **The formula written as a times d over b times c,** with the design it comes from named.
- **The confidence interval mentioned** in the same breath as the point estimate.
- **All three third variables told apart by position,** on the path or beside it.
- **The remove, report, do-not-adjust triad,** which is the whole second half of the question.

SEE ALSO Slot I - 78 odds ratio against relative risk Slot I - 80 internal and external validity SPRINT
Day 7, research methods, biostatistics and epidemiology

I - 74

Discuss different types of test of significance in research. [10]

10 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The axis	Data type, number of groups, pairing, distribution
5	The selector	Parametric and non-parametric, side by side
2	Assumptions	What a parametric test demands before it is legal
1	Reading the result	What a p value is, and what it is not

WHAT IS COMPARED	PARAMETRIC TEST	NON-PARAMETRIC TEST	DATA NEEDED
Two independent groups	Unpaired t-test	Mann-Whitney U	Numerical
Two related measures	Paired t-test	Wilcoxon signed rank	Numerical
Three or more independent	One-way analysis of variance	Kruskal-Wallis	Numerical
Three or more related	Repeated measures analysis of variance	Friedman	Numerical
Two proportions, independent	Z test for proportions	Chi square, or Fisher's exact when cells are sparse	Categorical
Two proportions, paired	Not applicable	McNemar	Categorical
Association of two numerical variables	Pearson correlation	Spearman rank correlation	Numerical

Amber marks the categorical rows, because they are where a candidate who has memorised the t-test family runs out.

- **What a parametric test assumes.** Numerical data on an interval scale, approximately normally distributed, with comparable variances across groups, and independent observations. Fail any of these and the non-parametric column is not a compromise, it is the correct test.
- **Reading the result.** A p value is the probability of a result at least this extreme if the null hypothesis were true. It is not the probability that the null is true, and it says nothing about the size of the effect. Report the effect with its confidence interval alongside it.
- **The two errors.** A type one error rejects a true null, its rate set by the significance level. A type two error misses a real effect, its rate falling as power rises. Multiple testing inflates the first, which is why a correction or a pre-specified primary outcome is required.

WHAT EARNS THE MARKS

- **A selector, not a list.** Every test arrives attached to the comparison it serves.
- **Parametric and non-parametric paired row by row,** rather than in two separate lists.
- **The four parametric assumptions named.**
- **The p value defined correctly,** which is the single most commonly lost mark on this question.

SEE ALSO Slot I - 72 the selector drawn, and the paired test Slot I - 71 the chi square test SPRINT Day 7, research methods, biostatistics and epidemiology

I - 75

What is regression analysis? In which situations it is indicated? Discuss giving examples, how multiple regression analysis is conducted. [2+2+6]

2 + 2 + 6 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Modelling one outcome from one or more predictors
2 When it is indicated	Predict, adjust, quantify a dose-response
5 How it is conducted	Specify, check, fit, report, in that order
1 The caution	Association modelled is not causation shown

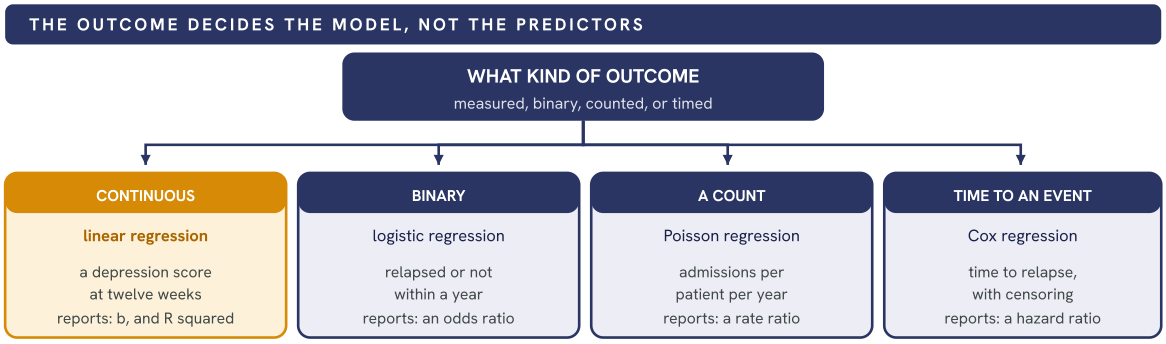


Fig 075.1 The regression family selected on the type of the outcome variable, each branch naming the effect measure it reports, with the linear case in amber as the one the question opens with.

HOW A MULTIPLE REGRESSION IS ACTUALLY CONDUCTED

Specify the outcome and choose predictors from clinical reasoning, never from a screening of p-values. Check the assumptions: a linear relation, independent residuals with constant spread and roughly normal distribution, and no severe collinearity between predictors. Keep enough data for the model, about ten observations for every predictor entered. Fit the model, entering variables together or in pre-planned blocks. Report each adjusted coefficient with its confidence interval, and the variance explained.

- **When it is indicated.** To predict an outcome from several variables, to adjust an exposure effect for measured confounders, and to quantify a dose-response relation across a range of exposure.
- **Two cautions.** Correlation quantifies the strength of a relation and makes no distinction between the two variables; regression names one as outcome and predicts it. And a stepwise search over many candidate predictors overfits, producing a model that does not replicate.

WHAT EARNS THE MARKS

- **The outcome type named as the thing that selects the model.**
- **Each model tied to the effect measure it reports,** coefficient, odds ratio, rate ratio, hazard ratio.
- **Assumptions checked before fitting,** written as a step of the method.

SEE ALSO Slot I - 73 confounding, effect modification and mediation Slot I - 83 Firth's penalized logistic regression

SPRINT Day 7, research methods, biostatistics and epidemiology

I - 76

Enumerate various statistical tests used to measure the differences. Describe the t-test in detail with an example. [4+6]

4 + 6 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 **Enumerate**	By pairing and by number of groups, in one line
2 **What the t-test is**	A signal to noise ratio, with its three forms
4 **The worked example**	Pooled spread, standard error, t, degrees of freedom
1 **Assumptions**	And the substitute when they fail

THE ENUMERATION, AND THE THREE FORMS OF THE T-TEST

Two independent groups take the unpaired t-test or Mann-Whitney U; two related measures take the paired t-test or Wilcoxon signed rank; three or more groups take analysis of variance or Kruskal-Wallis; proportions take chi square, Fisher's exact or McNemar. The t-test itself has three forms: one sample against a known value, two independent samples, and paired. Each is a difference divided by the standard error of that difference.

STEP	WORKING, ON ILLUSTRATIVE DATA	VALUE
The data	Ten patients per arm; means 18.0 and 22.0, standard deviations 4.0 and 5.0	n equals 10 and 10
Difference in means	22.0 minus 18.0	4.00
Pooled variance	Nine times 16, plus nine times 25, all over 18	20.50
Pooled standard deviation	The square root of 20.50	4.53
Standard error of the difference	4.53 times the square root of one fifth	2.02
The statistic and the verdict	4.00 divided by 2.02, on 18 degrees of freedom, against a two-sided cut of 2.10	**t equals 1.98, not significant**

Made-up data, arithmetic real. A four-point difference that looks clinically worth having fails to reach significance at this spread and this sample size, which is the lesson the example is here to teach.

- **Read the failure correctly.** Not significant does not mean no difference. It means this study could not distinguish the observed difference from chance, which is a statement about the study's power as much as about the treatment.
- **Assumptions.** Numerical outcome, roughly normal within each group, comparable variances, independent observations. Unequal variances take the Welch correction; non-normality with small samples takes Mann-Whitney U instead.

WHAT EARNS THE MARKS

- **The enumeration organised by pairing and group number,** not as an unsorted list.
- **The three forms of the t-test named,** which most answers reduce to two.
- **Arithmetic shown line by line** to the degrees of freedom and the critical value.
- **A verdict written in words,** including what a non-significant result does not prove.

SEE ALSO Slot I - 72 the selector drawn, and the paired t-test Slot I - 77 analysis of variance **SPRINT** Day 7, research methods, biostatistics and epidemiology

I - 77

Analysis of Variance (ANOVA). Its role in research in behavioral Medicine. [10]

10 | ASKED AT 7 MARKS | BIOSTATISTICS AND EPIDEMIOLOGY

TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What it is	Variance partitioned, then compared as a ratio
3	The working	The F ratio and its two degrees of freedom
2	Its forms	One-way, two-way, repeated measures, covariance
3	Role in the field	Where a behavioural trial needs it, and post hoc

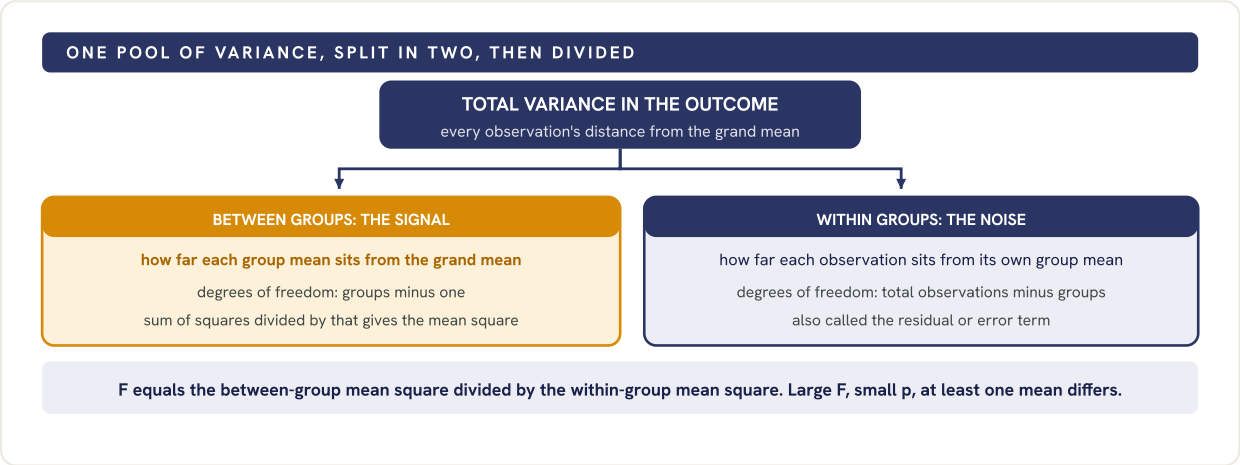


Fig 077.1 Analysis of variance drawn as what its name says: the total variation split into a between-group part and a within-group part, the ratio of the two being the test statistic.

- **Why not repeated t-tests.** Three comparisons at five per cent each carry about a fourteen per cent chance of a false positive. One F test holds it at five.
- **What a significant F does not say.** Not all means are equal, never which pair differs. Only then a post hoc test: Tukey, Bonferroni or Scheffe.
- **Its forms and its assumptions.** One-way for one factor; two-way for two, which makes an interaction testable; repeated measures over time; covariance to adjust for baseline. It assumes independence, normal residuals, comparable variances.

ITS ROLE IN BEHAVIOURAL RESEARCH

Behavioural work rarely compares two things once. It compares three or more arms, or the same participants at baseline, end of treatment and follow-up. Two-way and repeated measures forms are what let a trial test an interaction.

WHAT EARNS THE MARKS

- **The partition named,** between-group against within-group, before the F ratio.
- **Both degrees of freedom given,** groups minus one and observations minus groups.
- **The inflated error rate quantified,** not just asserted.
- **Interaction named as what two-way analysis buys.**

SEE ALSO Slot I - 74 tests of significance Slot I - 76 the t-test worked through SPRINT
Day 7, research methods, biostatistics and epidemiology

I - 78

What is odds ratio? How does it differ from relative risk? Describe the differences giving examples. [2+8]

2 + 8 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Both, defined	Odds against risk, and the table both come from
4 The grid	Design, denominator, meaning, behaviour
3 Worked both ways	A common outcome, then a rare one
1 The rule	When the two may be spoken of as one

DOMAIN	RELATIVE RISK	ODDS RATIO	WHAT SETTLES IT
What it divides	Risk in the exposed by risk in the unexposed	Odds in the exposed by odds in the unexposed	**Risk uses everyone as denominator, odds uses the non-affected**
Design it comes from	Cohort study or randomised trial	Case-control, and also cohort or trial	**A case-control fixes the cases, so it can yield no risk**
How it is read aloud	Directly: twice as likely	As odds, which is not the same as likely	Only relative risk may be spoken as times the chance
Common outcome	Stays interpretable	Sits further from one than the risk ratio does	**Above about ten per cent, the two separate**
Rare outcome	Close to the odds ratio	Approximates the relative risk	The rare disease assumption

WORKED TWICE, ON ILLUSTRATIVE COUNTS

Common outcome. Of 100 exposed, 30 develop the outcome; of 100 unexposed, 15 do. Risks are 0.30 and 0.15, so the relative risk is 2.00. Odds are 30 over 70 and 15 over 85, so the odds ratio is 2.43. The odds ratio has overstated the risk ratio. Rare outcome. Of 1000 exposed, 3 develop it; of 1000 unexposed, 1 does. The relative risk is 3.00 and the odds ratio is 3.01. Now they agree.

- **The rule the two examples prove.** The odds ratio approximates the relative risk only when the outcome is uncommon. When the outcome is common, the odds ratio exaggerates the effect in whichever direction it points, and reporting it as though it were a risk ratio overstates the finding.
- **Where this bites in practice.** Logistic regression returns odds ratios whatever the frequency of the outcome, so an adjusted odds ratio from a study of a common outcome is routinely read as a risk ratio and routinely overstated. Say odds, and give the confidence interval.

WHAT EARNS THE MARKS

- **Odds distinguished from risk in the first two lines,** by what sits in the denominator.
- **The design constraint stated,** why a case-control study cannot report a risk.
- **Numbers on the page.** The stem asks for examples, so the two worked sets are the examples.
- **The rare disease assumption named as an assumption,** with the condition under which it fails.

SEE ALSO Slot I - 73 the odds ratio and confounding Slot I - 75 regression and the measures it reports SPRINT Day 7, research methods, biostatistics and epidemiology

AREA 09 OF 12 · P1-A10

Research methodology

6	5.3%	I-79	I-84
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 5

REVISION ORDER

Reliability and validity first, three of the other five lean on it. Sampling and sample size next. The qualitative slot and the research-question slot after that, both of them arguments rather than lists. The survey slot and the registry slot are independent, and each carries a second half that has to be prepared separately from the first.

WHAT IS IN THIS AREA

Qualitative research set against quantitative. Reliability with its types, and internal against external validity. Sampling with the principles of sample-size calculation for two designs. The research question with the types of hypothesis and a critique of meta-analysis and network analysis. The National Mental Health Survey with Firth's penalised logistic regression. The clinical trials registry with the steps to be completed before a trial opens.

I - 79

What do you understand about qualitative research? How does it differ from quantitative research in terms of essential features, strength and limitations? [2+3+3+2]

2 + 3 + 3 + 2 | RESEARCH METHODOLOGY | DNB - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	**Both, defined**	Meaning and process against magnitude and frequency
4	**The grid**	Question, sampling, data, analysis, rigour
2	**Strengths and limits**	What each buys and what each cannot say
2	**Choosing**	The question decides, and mixed methods

DOMAIN	QUALITATIVE	QUANTITATIVE	WHAT SETTLES IT
The question	What is it like, how does it work, why does it happen	How many, how much, how strongly associated	**Does the question ask for meaning or for a number**
Sampling	Purposive, small, continued until saturation	Probability based, size fixed by a power calculation	**Saturation against a power calculation**
Data	Interviews, focus groups, observation, documents	Measurements, scores, counts	Words against numbers
Analysis	Coding into themes, framework analysis, grounded theory	Statistical tests and effect estimates	Interpretive against inferential
Rigour	Credibility, transferability, dependability, confirmability	Internal validity, external validity, reliability, precision	**Trustworthiness against validity**
Output	Theory and thick description, transferable not generalisable	A population estimate with a confidence interval	Whether a number can be quoted from it

Amber cells are the discriminators an examiner is checking for. The rest of the column supports them.

- **Strengths of the qualitative arm.** It reaches what a questionnaire cannot: illness explanation, treatment refusal, stigma, family decision-making. It generates the hypothesis that a later trial will test, and it explains why a trial that worked elsewhere failed here.
- **Its limits, stated honestly.** No prevalence, no effect size, no generalisation to a population. It is labour intensive, and the analyst is part of the instrument, which is why reflexivity, triangulation, member checking and an audit trail are demanded in its place.
- **Mixed methods.** Sequential where qualitative work builds the instrument a survey then uses, or explains a trial result; concurrent where both run inside one study and are integrated at interpretation.

WHAT EARNS THE MARKS

- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **Saturation named against power.** It is the sampling discriminator and most answers miss it.
- **The four trustworthiness criteria named,** rather than a claim that qualitative work has no rigour standard.
- **Mixed methods offered as the resolution,** not as a hedge.

SEE ALSO Slot I - 80 reliability and validity Slot I - 81 sampling and sample size SPRINT

Day 7, research methods, biostatistics and epidemiology

I - 80

What is reliability? Discuss the different types of reliability with examples. What is internal validity and external validity of a study? [2+4+4]

2 + 4 + 4 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Reliability defined	Consistency, and how it differs from accuracy
4	The four types	Each with its statistic and one example
2	Internal validity	Selection, information, confounding
2	External validity	Generalisability, and the trade-off between the two

THE DEFINITION

Reliability is the consistency of a measurement: the same instrument on the same unchanged subject gives the same answer again. Validity is accuracy, whether the instrument measures what it claims. A scale that always reads five kilograms heavy is perfectly reliable and wholly invalid, which is why reliability is necessary and never sufficient.

TYPE	WHAT IT ASKS	STATISTIC	EXAMPLE
Test-retest	Is the score stable over time in a stable subject	Correlation coefficient	The same scale re-given after two weeks
Inter-rater	Is the score independent of who rates it	Cohen's kappa for categories	Two clinicians rating one interview
Internal consistency	Do all items measure one construct	Cronbach's alpha, split-half	Odd items correlated with even items
Parallel forms	Are two versions interchangeable	Correlation between forms	Two equivalent versions given in turn

Split-half correlations are corrected upward by the Spearman-Brown formula; alpha is the average of all possible split halves.

- **Internal validity.** Whether the association observed inside this study is real for the people studied. It is threatened by selection bias, information and measurement bias, confounding, and chance. Randomisation, blinding, standardised measurement and adjustment are the defences.
- **External validity.** Whether the finding transfers to other patients, settings and times. It is judged from the sampling frame, the inclusion and exclusion criteria, the setting and the comparator used.
- **The trade-off.** They pull against each other. A tightly criteria-bound trial buys internal validity by excluding the comorbid, elderly and substance-using patients who fill an actual clinic, and pays for it in external validity. Internal validity is judged first: a biased study does not generalise, it only travels.

WHAT EARNS THE MARKS

- **Reliability separated from validity in the first two lines,** with the consistently-wrong example.
- **Four types, each with a statistic and an example.** The statistic is the mark most answers drop.
- **Internal validity given as its three named threats,** not as a definition alone.
- **The trade-off stated,** with internal validity judged first.

SEE ALSO Slot I - 49 psychometric properties of a scale Slot I - 79 qualitative and quantitative research SPRINT Day 7, research methods, biostatistics and epidemiology

I - 81

Describe different type of sampling in psychiatric research. What is the sample size? Describe principle and essential components of sample size calculation for any two types of research designs? [2+2+6]

2 + 2 + 6 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The sampling tree	Probability against non-probability, named methods
2 Sample size	The smallest n that can answer the question
4 Two designs	A prevalence survey and a two-arm trial
1 Inflation	Attrition, and the cluster design effect

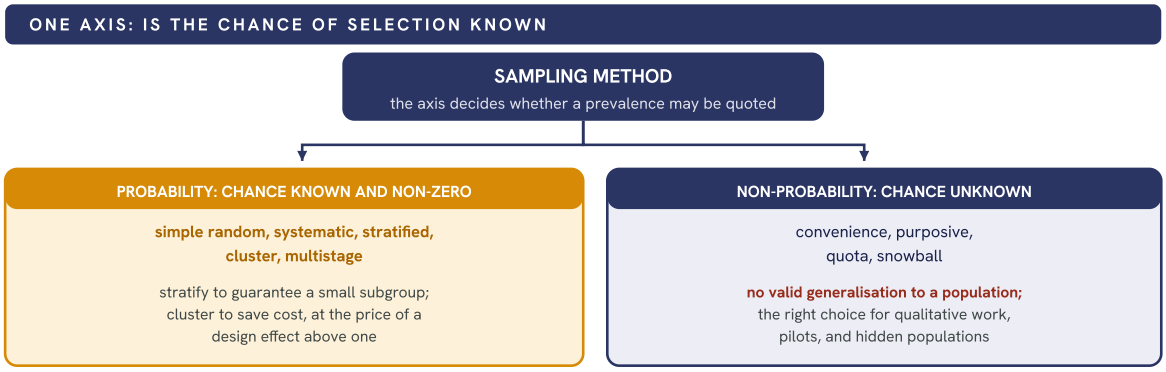


Fig 081.1 Sampling divided on the one axis that matters, whether the probability of selection is known, with the branch that permits a population estimate in amber.

TWO DESIGNS, THE COMPONENTS SPELLED OUT

A prevalence survey needs the expected proportion, the absolute precision wanted either side of it, and the confidence level, giving the familiar form n equals Z squared times p times one minus p, divided by d squared. A two-arm trial comparing means needs the smallest difference worth detecting, the standard deviation of the outcome, the significance level, usually five per cent two-sided, and the power, usually eighty or ninety per cent.

- **The principle.** Sample size is the smallest number that gives an acceptable chance of detecting an effect that is real and worth detecting. It is fixed before recruitment, from the primary outcome only, and never revised because the result was disappointing.
- **Then inflate it.** Add for expected attrition, and multiply by the design effect in any cluster or multistage design. An unadjusted cluster sample is over-precise and its confidence intervals are too narrow.

WHAT EARNS THE MARKS

- **The axis stated before the list,** known against unknown probability of selection.
- **Power and significance level both named,** with their conventional values.
- **The design effect,** which separates a competent answer from a memorised formula.

SEE ALSO Slot I - 79 qualitative and quantitative research Slot I - 82 research question and hypothesis **SPRINT** Day 7, research methods, biostatistics and epidemiology

I - 82

How to generate research question? Describe different types of hypothesis in research. Discuss advantages and disadvantages of meta analysis and network analysis. [2+2+6]

2 + 2 + 6 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Generating the question**	Where it comes from, then the two frameworks
2	**Types of hypothesis**	Null and alternative, directional, simple, complex
4	**Meta-analysis**	What pooling buys, and what it cannot repair
2	**Network meta-analysis**	Indirect comparison, and the assumption it rests on

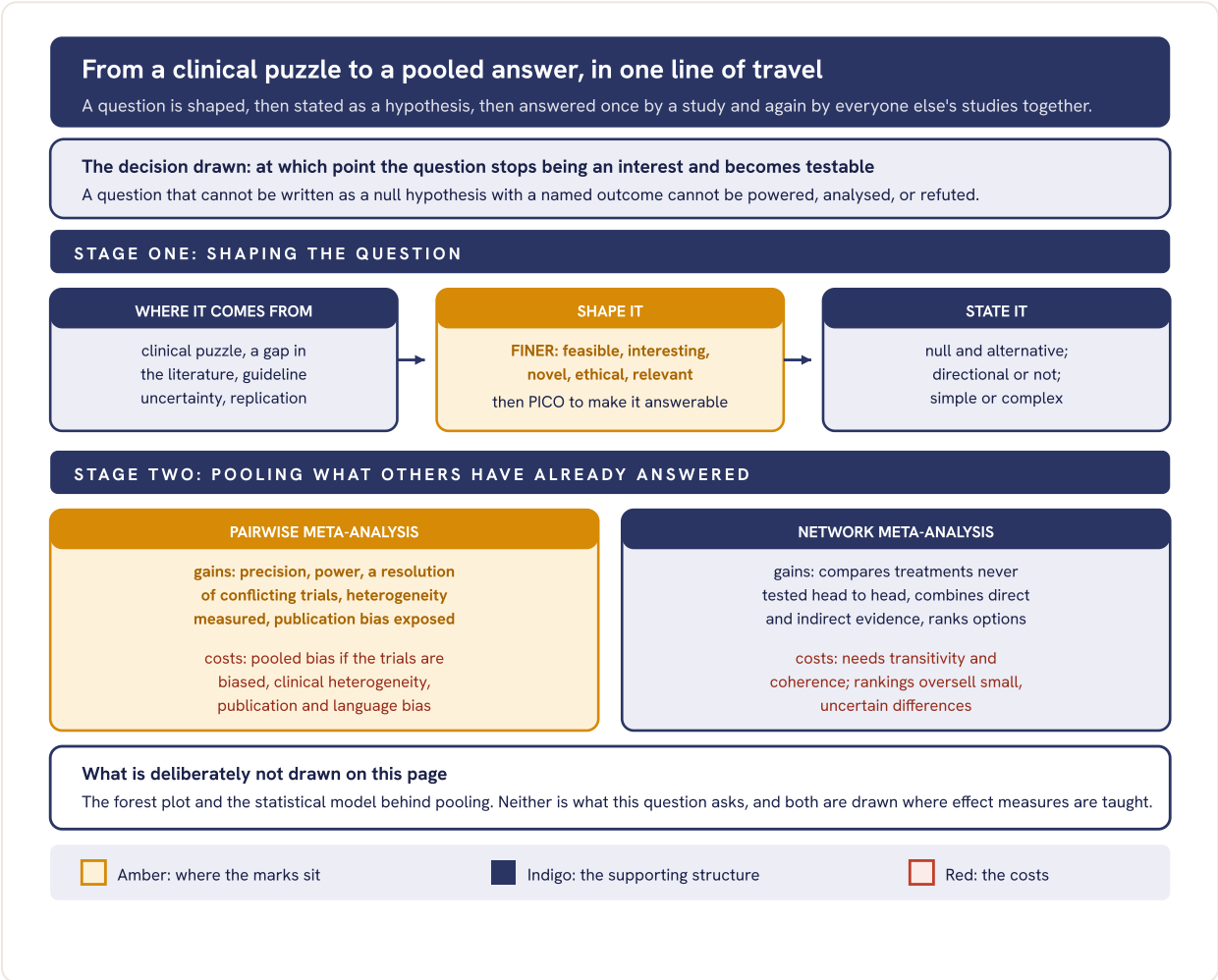


Fig 082.1 The question shaped and stated in the upper band, then answered a second time by synthesis in the lower band, with gains and costs kept apart inside each method.

- **The hypothesis types.** Null against alternative; directional, tested one-sided, against non-directional, tested two-sided; simple, one predictor and one outcome, against complex, several of either; and the research hypothesis, written in words, against the statistical hypothesis, written about parameters.
- **The other reading.** If the examiner means symptom network analysis rather than network meta-analysis, say so and answer briefly: symptoms modelled as nodes with partial correlations as edges, with central symptoms proposed as treatment targets, and stability of the estimated network the standing criticism.

WHAT EARNS THE MARKS

- **A named framework for shaping the question,** not a paragraph on curiosity.
- **Null and alternative written out,** with one-sided and two-sided attached to directionality.
- **Gains and costs kept in separate lists for each method,** as the plate keeps them.
- **Transitivity named** as the assumption a network analysis stands or falls on.

SEE ALSO Slot I - 81 sampling and sample size Slot I - 84 the clinical trials registry and pre-trial steps **SPRINT** Day 7, research methods, biostatistics and epidemiology

I - 83

Discuss various findings of National Mental Health Survey of India 2016. What is Firth's penalized logistic regression used in this survey? [6+4]

6 + 4 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What the survey was	Design and scope, before any number
4	The findings	Burden, the treatment gap, what it cannot say
3	Firth's method	The problem it solves in a rare-outcome model
1	Why it matters here	Rare disorders in small subgroups

WHAT IS ASKED	WHAT THE SURVEY REPORTED	WHY IT MATTERS
Design and scope	A cross-sectional multistage stratified random household survey across twelve states, run in 2015 to 2016	**Known selection probabilities, so a national estimate is legitimate**
Current burden	Weighted current prevalence of any mental disorder about 10.6 per cent	Roughly one adult in ten is ill now
Lifetime burden	Lifetime prevalence near 13.7 per cent	The denominator for any service projection
Treatment gap	**Above 80 per cent overall, and widest for substance use**	The policy headline, and the reason the survey is quoted at all
What it cannot say	These are prevalence figures, a stock of existing cases	Prevalence is roughly incidence times duration, so it cannot show whether new cases are rising

FIRTH'S PENALIZED LOGISTIC REGRESSION

Ordinary logistic regression fits by maximum likelihood, which is biased in small samples and breaks outright under separation, where a predictor perfectly or almost perfectly divides the outcome and the estimated odds ratio runs to infinity with an unusable standard error. Firth's method adds a penalty term to the likelihood, an invariant prior on the parameters, which removes most of the small-sample bias and always returns a finite estimate with a usable interval.

- **Why a prevalence survey reaches for it.** Individual disorders are uncommon and subgroup cells are small, so a model of a rare disorder against a rare exposure can easily contain a zero cell. The stem states the survey used it; what it buys, in general, is a finite and interpretable odds ratio where the ordinary fit would give none.
- **Read the survey as a design, not a table.** The examinable point is that this design, cross-sectional, multistage and probability sampled, is what makes those percentages quotable at all. A convenience sample of clinic attenders could not have produced a national figure whatever its size.

WHAT EARNS THE MARKS

- **The design named before the numbers,** cross-sectional and multistage.
- **Current and lifetime prevalence given as two separate figures,** with the treatment gap third.
- **Separation named** as the specific failure Firth's penalty repairs.
- **The prevalence-is-not-incidence line,** which is the honest limit of the whole dataset.

SEE ALSO Slot I - 81 sampling and sample size Slot I - 75 regression analysis SPRINT

Day 7, research methods, biostatistics and epidemiology

I - 84

What is clinical trial registry of India? Describe its role in research. What are the steps to be followed before starting a clinical trial in your hospital? [2+3+5]

2 + 3 + 5 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it is	Who runs it, since when, and its standing
3	Its role	Transparency, selective reporting, publication
4	The steps	Protocol, ethics, regulator, registration, consent
1	The line	Nothing is enrolled before all of it is done

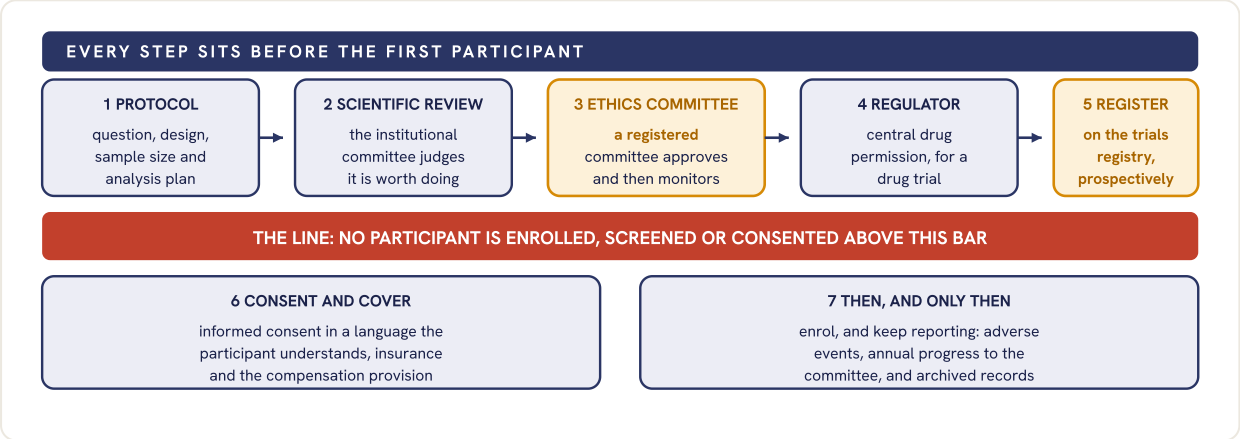


Fig 084.1 The pre-trial sequence with the enrolment bar in red, because the marks in the second half of this question are for putting each step on the correct side of it.

- **What the registry is.** The Clinical Trials Registry of India, launched in 2007 and run by the national medical statistics institute under the Indian Council of Medical Research. It is a primary registry of the World Health Organization platform, and prospective registration has been mandatory since June 2009.
- **Its role.** A public record of what was planned before any result existed, which makes selective outcome reporting and quiet abandonment visible, and which journals now require.
- **The Indian frame.** Human research is governed by the national ethical guidelines of 2017, a registered ethics committee being mandatory; a drug trial adds the new drugs and clinical trials rules of 2019.

PITFALL

Registering after recruitment has begun. Retrospective registration defeats the purpose of a registry, is refused by most journals, and cannot be repaired later.

WHAT EARNS THE MARKS

- **The registry placed institutionally,** who runs it and under whose platform.
- **Prospective registration named as the rule,** with the reason it exists.
- **Steps in order with the enrolment line drawn,** and both the 2017 guidelines and the 2019 rules cited for a drug trial.

SEE ALSO Slot I - 82 research question and hypothesis Slot I - 81 sampling and sample size **SPRINT** Day 7, research methods, biostatistics and epidemiology

AREA 10 OF 12 · P1-A06

Developmental psychology and attachment

6	5.1%	I-85	I-90
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 SINGLE 4

REVISION ORDER

The three Erikson slots together and in that order: the full life cycle first, then the childhood stages, then the biographical slot, which the first two have already written most of. Piaget next, and it is the highest-scoring question in this book. Attachment theory and theory of mind last, and both are short.

WHAT IS IN THIS AREA

Erikson in three slots: the eight stages of the life cycle, the childhood half of them, and the man with his contribution. Piaget's stages of cognitive development. Attachment theory. Theory of mind, with its place in normal development and in disorder.

I - 85

Describe the Piaget's theory and the various stages of cognitive development. [2+8]

2 + 8 | DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

DNB, KUHS, RGUHS, TN-MGRMU - 6 APPEARANCES, 6 OF 78 SESSIONS

SKELETON

2	The theory	Adaptation, and the four invariants that drive it
5	The four stages	Age, defining operation, and the error still made
2	Clinical use	Explanation, consent, delay, a child's account
1	Criticism	One honest limit, named rather than hinted at

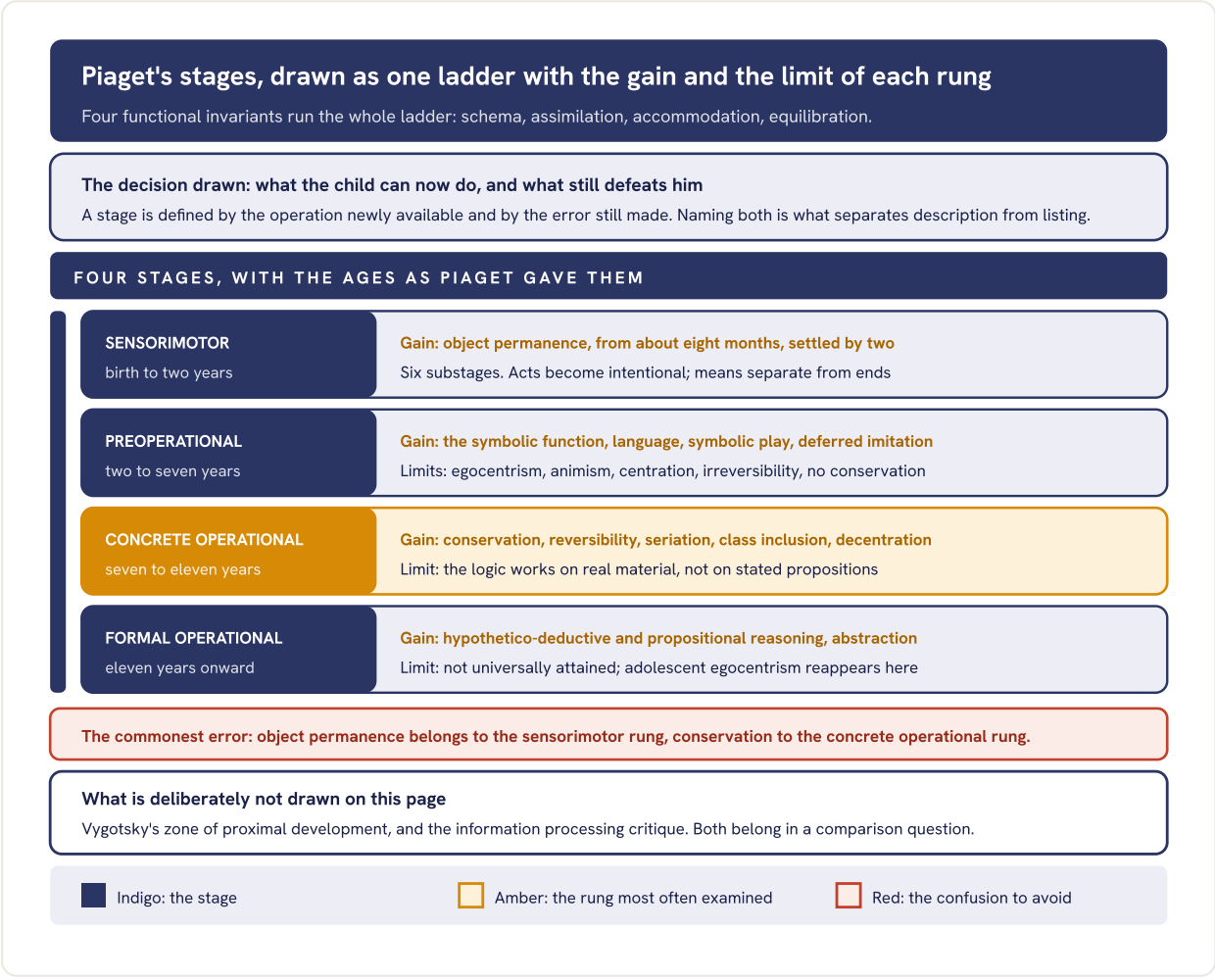


Fig 085.1 The four stages as rungs on a single spine, each carrying its age range, the operation it makes available in amber, and the error that still defeats the child at that rung.

- **The theory, not only the stages.** Intelligence is adaptation. The schema is the unit; assimilation fits experience into an existing schema, accommodation alters the schema to fit experience, equilibration drives the alternation. The sequence is invariant, the rate is not, and each stage is a different logic, not more of the last.
- **Clinical use, and the honest criticism.** The stages set how illness is explained to a child, the threshold for assent, and the weight given to a child's account. Piaget underestimated infants because his tasks demanded motor and verbal responses, underplayed teaching and culture, and formal operations are not universally reached.

WHAT EARNS THE MARKS

- **The four invariants named before the ladder.** Schema, assimilation, accommodation, equilibration.
- **Every rung with its age and its defining operation.** Ages without the operation is where the eight marks go.
- **The limit of each stage, not only its gain.**
- **The red strip.** Object permanence and conservation belong to different rungs.

SEE ALSO Slot I - 87 theory of mind and its developmental timetable Slot I - 88 Erikson's theory of child development

SPRINT Day 4, general and developmental psychology

I - 86

Erik H.Erikson - Stages of Life Cycle. [10]

10 ASKED AT 15 MARKS DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

TN-MGRMU, WBUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 The frame	Epigenetic principle, and the crisis as a ratio
6 The eight stages	Age, crisis, virtue, and the pathology if unresolved
2 Clinical use	Where the life cycle enters a formulation

STAGE AND AGE	CRISIS	VIRTUE	IF UNRESOLVED
Infancy, to one year	Trust against mistrust	Hope	Withdrawal, suspicion
Early childhood, one to three	Autonomy against shame and doubt	Will	Compulsion, self doubt
Play age, three to six	Initiative against guilt	Purpose	Inhibition, guilt
School age, six to eleven	Industry against inferiority	Competence	Inertia, inferiority
Adolescence, twelve to eighteen	Identity against role confusion	Fidelity	Identity diffusion, repudiation
Young adulthood	Intimacy against isolation	Love	Exclusivity, isolation
Middle adulthood	Generativity against stagnation	Care	Rejectivity, self absorption
Late adulthood	Ego integrity against despair	Wisdom	Disdain, despair

- **The epigenetic principle.** Erik Homburger Erikson held that development unfolds on a ground plan, each stage arising at its proper time and the whole personality being the sum of all eight. A crisis is resolved as a ratio, never as a victory: enough trust to function alongside enough mistrust to stay safe.
- **Where it is used.** Identity work in adolescent presentations, generativity in midlife depression, life review and integrity in late life. Erikson extended Freud's five psychosexual stages across the whole life span and made the conflict social rather than instinctual, which is the sentence that names his contribution.

WHAT EARNS THE MARKS

- **All eight stages, with the crisis written as a pair of opposites,** which is the form Erikson used.
- **The virtue attached to each resolved crisis.** Most answers give the crisis and stop there.
- **The epigenetic principle named.** It is what makes this a theory rather than a table.

SEE ALSO Slot I - 88 Erikson's theory of child development Slot I - 90 Erikson, the man and the contribution SPRINT Day 4, general and developmental psychology

I - 87

Define "Theory of Mind" (TOM). What is the significance of TOM in normal development of children? Describe its significance in various psychiatric disorders. [1+3+6]

1 + 3 + 6 | DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1 Definition	The capacity, and who named it in which year
3 Normal development	The timetable, drawn, with the task at each point
6 In disorder	Four conditions, each with the specific failure named

THE DEFINITION

The capacity to attribute mental states, beliefs, desires, intentions and knowledge, to oneself and to others, and to grasp that another person's may differ from one's own. Premack and Woodruff named it in 1978.

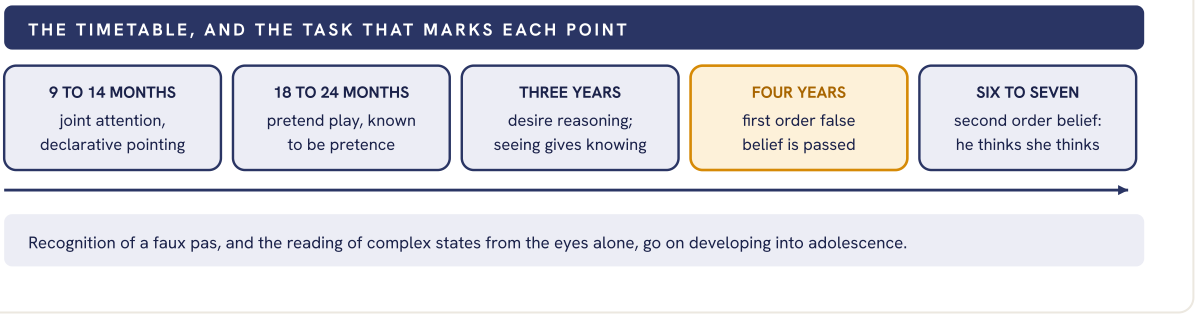


Fig 087.1 The developmental timetable, with the first order false belief task at four years marked in amber because it is the milestone every version of this question turns on.

- **Significance in normal development.** It is what makes deception, persuasion, teaching, keeping a secret, empathy and negotiated friendship possible. The false belief task of Wimmer and Perner is its standard marker, in the Sally-Anne form used by Baron-Cohen, Leslie and Frith.
- **Significance in disorder, named condition by condition.** Autism, where the false belief failure was first shown and framed as mindblindness. Schizophrenia, where mentalising failure tracks persecutory delusions and passivity phenomena. Antisocial personality, where cognitive theory of mind is intact while the affective side is not. It is reported too in attention deficit hyperactivity disorder, in bipolar disorder, in anorexia nervosa and in frontotemporal dementia.

WHAT EARNS THE MARKS

- **The definition in one sentence with its 1978 attribution.** One mark, thirty seconds.
- **The timetable with ages,** and the four year first order task named as the amber milestone.
- **A specific failure per disorder,** not a list of diagnoses said to have a deficit.

SEE ALSO Slot I - 85 Piaget and the stages of cognitive development Slot I - 89 attachment theory **SPRINT** Day 4, general and developmental psychology

I - 88

Erikson's theory of child development. [10]

10 DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The departure	Psychosocial against psychosexual, stated first
6 The childhood crises	Five, each with its age and its Freudian parallel
2 What the child needs	What each stage asks of the people around it

CRISIS	AGE	FREUD'S PARALLEL	SEEN IN CLINIC AS
Trust against mistrust	To one year	Oral	Difficulty trusting any caregiver, later any clinician
Autonomy against shame and doubt	One to three	Anal	Control battles; shame carried into adult self doubt
Initiative against guilt	Three to six	Phallic	Inhibited play and questioning; guilt at wanting
Industry against inferiority	Six to eleven	Latency	School refusal, inferiority, giving up on effort
Identity against role confusion	Twelve to eighteen	Genital	Identity diffusion, and the borderline presentations near it

- **How Erikson departed from Freud.** Freud's stages end at puberty and turn on the site of instinctual gratification. Erikson's run the whole life span and turn on a social task set by the widening circle of people around the child. The ego is autonomous and adaptive, not merely the servant of drive and reality.
- **What each stage asks of the environment.** Consistent responsive care in the first year; graded permission to act and to fail in the next two; tolerance of questions and noisy play in the preschool years; real tasks with real feedback through school; and in adolescence a moratorium in which roles can be tried without permanent cost.

PITFALL

The stem asks for child development, so the three adult stages are context and not content. Listing all eight and describing none of them is the commonest way this answer goes wrong.

WHAT EARNS THE MARKS

- **Psychosocial set against psychosexual in the opening lines.** It frames everything after it.
- **Five childhood crises with ages,** each written as a pair of opposites.
- **What the environment must supply at each stage,** which is what turns the table into developmental psychology.

SEE ALSO Slot I - 86 the eight stages of the life cycle Slot I - 85 Piaget and the stages of cognitive development

SPRINT Day 4, general and developmental psychology

I - 89

Attachment theory. [10]

10 DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The claim	A behavioural system, not a by-product of feeding
3 Development	Four phases, and the separation sequence
3 The patterns	Four classifications, and how each is elicited
2 Clinical use	The disorders, and what a pattern predicts

THE CLAIM

Bowlby held attachment to be a biologically based behavioural system, selected because proximity to a caregiver kept the infant safe. Its set goal is felt security. It is activated by fear, illness and separation, and terminated by contact.

PATTERN	IN THE STRANGE SITUATION	CAREGIVER PATTERN	ADULT INTERVIEW
Secure	Protests separation, is comforted, returns to play	Consistently available and responsive	Autonomous
Insecure avoidant	Little protest, ignores the return, play looks unaffected	Rejecting of distress, pushes independence	Dismissing
Insecure resistant	Intense protest, then angry and clingy, hard to settle	Inconsistent and unpredictable	Preoccupied
Disorganised	Contradictory approach, freezing, apprehension at the caregiver	Frightened or frightening; loss and trauma	Unresolved

- **The developmental frame.** Four phases: preattachment in the first weeks, attachment in the making to about six months, clear-cut attachment with separation protest and stranger wariness from then to two years, and goal corrected partnership after it. Separation runs protest, then despair, then detachment. What is laid down is an internal working model of self and other.
- **Evidence and clinical use.** Harlow's infant monkeys clung to the cloth mother rather than the one that fed them, which separated attachment from feeding. Ainsworth's Strange Situation, eight brief episodes with two separations, is the classification instrument; disorganised was added later by Main and Solomon; the Adult Attachment Interview is the adult counterpart. Clinically: reactive attachment disorder, disinhibited social engagement disorder, separation anxiety, and disorganised attachment as an antecedent of borderline presentations.

WHAT EARNS THE MARKS

- **Attachment named as a system with a set goal,** not as a synonym for a close bond.
- **All four patterns in the grid,** disorganised included, with what elicits each.
- **The instrument named,** Strange Situation for infants, Adult Attachment Interview for adults.
- **The internal working model,** which is what carries the childhood pattern into adult relationships.

SEE ALSO Slot I - 87 theory of mind and its developmental timetable

Slot I - 64 imprinting and the ethological background SPRINT Day 4, general and developmental psychology

I - 90

Eric Erikson. [10]

10 DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The man	Dates, training, and the phrase he gave the language
5 The contribution	Four ideas, each stated in its own terms
2 Standing today	Where it is used, and the criticism it carries

THE MAN

Erik Homburger Erikson, 1902 to 1994. He trained in Vienna in the analysis of children, with Anna Freud as his analyst, and held neither a medical nor a doctoral degree. He gave psychoanalysis the whole life span, gave the ego its own developmental line, and gave the language the phrase identity crisis.

- **The four contributions, named separately.** The epigenetic principle, that development unfolds on a ground plan with each part rising at its proper time. The eight psychosocial stages, which carried Freud's five past puberty to the end of life. Ego psychology, in which the ego has its own energies and adaptive tasks rather than serving drive and reality alone. And the ego virtues, one strength issuing from each crisis resolved.
- **Identity, which is his central idea.** Identity is a sense of sameness and continuity within oneself, matched by the recognition of that sameness by others. Its failure in adolescence is role confusion. He described the psychosocial moratorium, a sanctioned delay in which roles are tried without permanent commitment, and identity diffusion as the outcome when it does not close.
- **Psychohistory, and the criticism.** He applied the stage frame to whole lives, in studies of Luther and of Gandhi, arguing that a personal identity crisis and a historical moment can resolve each other. This is the part of the work most criticised, since a single life cannot be a controlled observation, and the stages themselves are hard to falsify.

WHERE ERIKSON IS STILL USED

Adolescent presentations read as identity work rather than as settled trait pathology. Midlife depression framed as stagnation against generativity. Life review in late life and in palliative care, where integrity against despair is the live question rather than a theoretical one.

WHAT EARNS THE MARKS

- **The epigenetic principle and the eight stages named as two contributions,** because they are two.
- **Identity defined rather than invoked.** Sameness within, recognised from without.
- **One honest criticism.** On a named-person stem it is what separates command from recitation.

SEE ALSO Slot I - 86 the eight stages of the life cycle Slot I - 34 Anna Freud SPRINT
Day 4, general and developmental psychology

AREA 11 OF 12 · P1-A12

Nosology, classification and descriptive psychopathology

6	4.9%	I-91	I-96
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 6

REVISION ORDER

The three phenomenology slots together: they share one definitional grammar and the marks are in the criteria, not in the length. The two classification slots next, the neo-Kraepelinians first because the revision slot argues from them. The spectrum slot last, and give it time: it is three questions in one.

WHAT IS IN THIS AREA

Three phenomenology slots: delusion with the primary forms and the dimensions, consciousness with its dimensions and quantitative changes, and hallucination with the true against pseudo distinction. Three nosology slots: the neo-Kraepelinians and their influence on the current systems, the purpose of revising DSM and ICD and how far that purpose has been met, and the spectrum concept with the mixed episode and the epilepsy spectrum. Every slot in this area was seen once in the record, which is the whole of what its chips claim.

I - 91

Who are the neo-Kraeplineans? How they influence the current classificatory system? [3+7]

3 + 7 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Who they were**	The St Louis school, and why Kraepelin is the ancestor
2	**What they demanded**	Illness, boundaries, criteria, validation
4	**The influence**	Feighner, the RDC, DSM-III, and what ICD took
1	**The honest limit**	Reliability bought, validity still owed

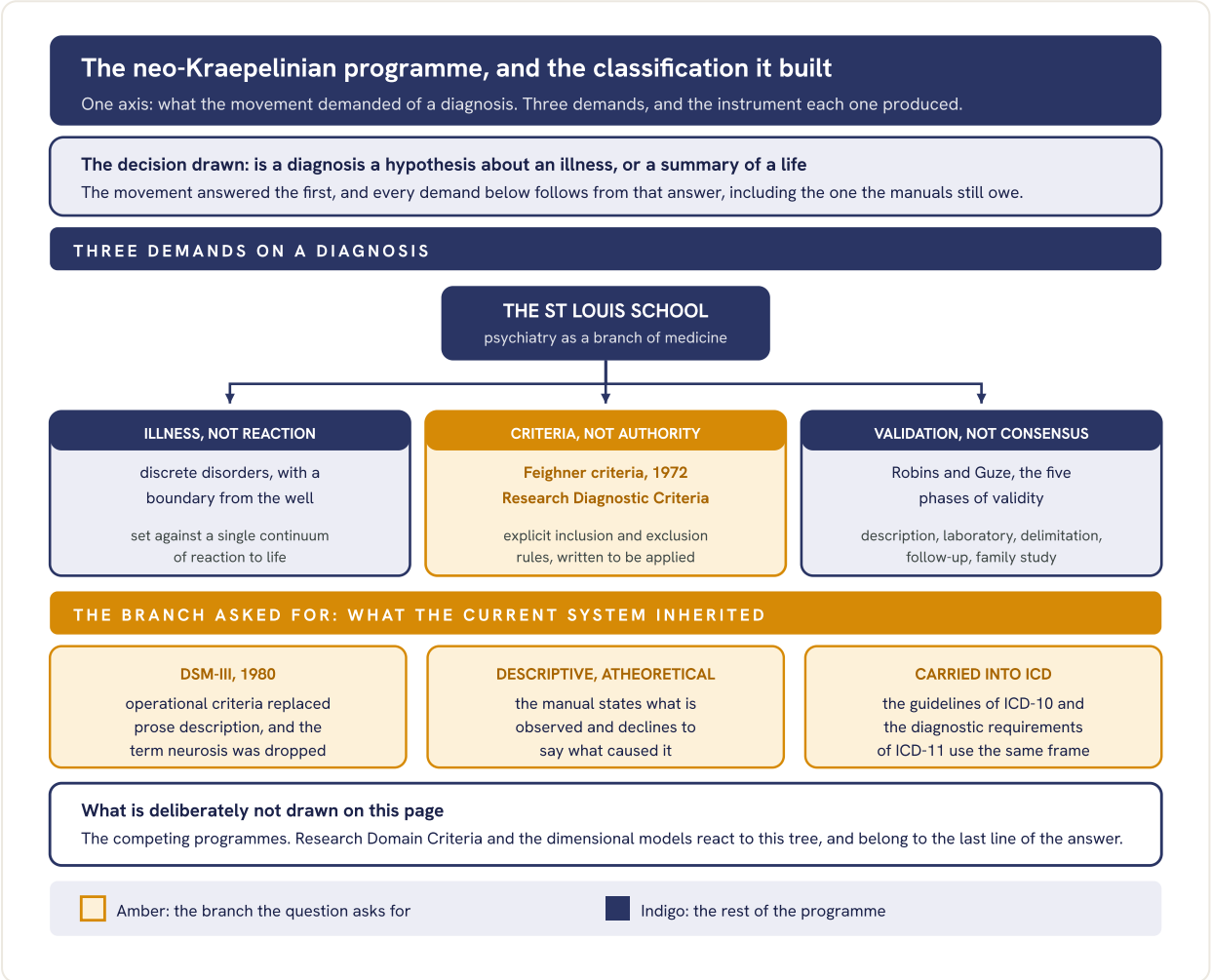


Fig 091.1 The neo-Kraepelinian programme drawn as three demands made on a diagnosis, with the criteria branch opened out into the architecture DSM-III built and both manuals still use.

- **Who they were.** A group working in St Louis from the late 1960s, Eli Robins, Samuel Guze and George Winokur, named neo-Kraepelinian by Gerald Klerman, who set out the movement's credo. Kraepelin is the ancestor because he classified by course and outcome rather than by presumed cause.
- **What it has not delivered.** Reliability improved; validity did not follow. No laboratory finding delimits these categories, comorbidity is the rule rather than the exception, and both manuals have answered by adding dimensions to a frame that stays categorical.

WHAT EARNS THE MARKS

- **The names attached to the movement.** Robins, Guze and Winokur, and Klerman who named it.
- **Operational criteria named as the inheritance,** rather than DSM-III named as an event.
- **The Robins and Guze validators listed.** They are the standard the system is still judged against.
- **The unfinished part stated.** Reliability without validity is the honest close.

SEE ALSO Slot I - 93 the purpose of revising a classification Slot I - 92 spectrum concepts in psychiatry **SPRINT** Day 6, psychometry, assessment and descriptive psychopathology

I - 92

Discuss the concept of spectrum disorder(s) in psychiatry with suitable diagrams. How has current understanding of mixed episode developed? What is epilepsy spectrum disorder? [3+5+2]

3 + 5 + 2 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	What a spectrum is	Two senses, drawn: one graded condition, one family
2	Why it is claimed	Family, genetic and dimensional evidence
3	Mixed episode	Kraepelin, DSM-IV, the DSM-5 specifier, ICD-11
2	Epilepsy spectrum	What the term covers, and where it stands

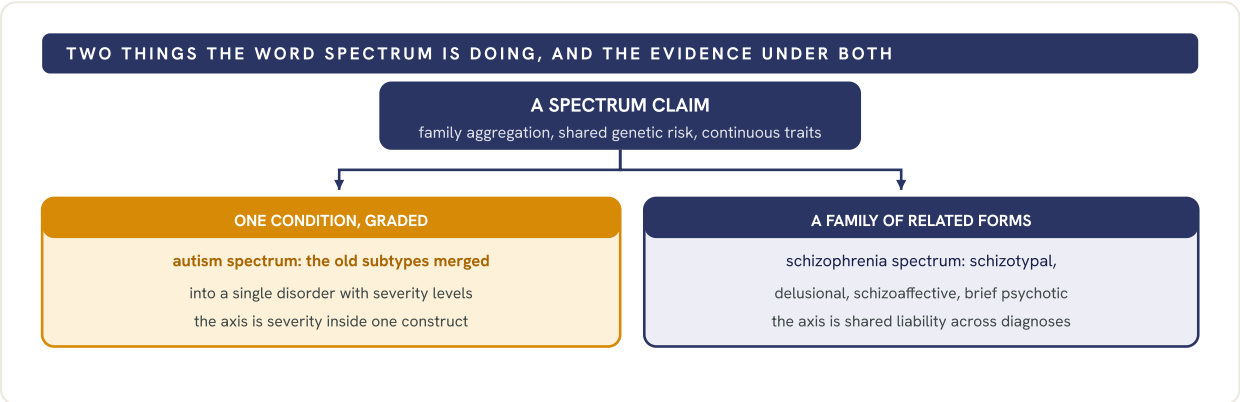


Fig 092.1 The two senses the word spectrum carries in psychiatry, severity graded within one construct on the left and shared liability across neighbouring diagnoses on the right, with the evidence both rest on named at the root.

- **How the mixed episode changed.** Kraepelin drew mixed states from the independent variation of mood, thought and volition. DSM-IV required full criteria for mania and for depression at once, a threshold so rarely met that most mixity went unrecorded.
- **Where the systems now part.** DSM-5-TR carries mixity as a with-mixed-features specifier, three non-overlapping symptoms of the opposite pole. ICD-11 keeps a mixed episode as an episode type. Mixed features predict poorer outcome and higher suicide risk.

WHAT THE THIRD PART IS ACTUALLY ASKING

Epilepsy spectrum disorder names no category in either manual. It is used for the temporolimbic psychiatric picture seen with epilepsy: interictal dysphoric disorder, postictal and interictal psychosis, and the interictal behaviour syndrome of hyperreligiosity, hypergraphia, viscosity and altered sexuality.

WHAT EARNS THE MARKS

- **Both senses of spectrum given, with an example under each.**
- **The mixed episode traced, not merely defined.** Kraepelin, DSM-IV, DSM-5, ICD-11.
- **The specifier stated exactly.** Three non-overlapping symptoms of the opposite pole.
- **The honest line on the third part.** The term names no category in either manual.

SEE ALSO Slot I - 91 the neo-Kraepelinians and the current system Slot I - 93 the purpose of revising a classification

I - 93

There have been many revisions of diagnostic systems (e.g. DSM, ICD) in psychiatry. In your view, what is the purpose of the exercise of revision and to what extent the purpose has been achieved by DSM-5 and ICD-11? [5+5]

5 + 5 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Why a system is revised	Evidence, usability, harm, and the users served
3	What the two did	Regrouping, dimensions, and what was removed
2	Where it was met	Organisation and utility, field tested
2	Where it was not	No validators, comorbidity, threshold creep

PURPOSE OF REVISING	WHAT DSM-5 AND DSM-5-TR DID	WHAT ICD-11 DID	HOW FAR IT WAS MET
Follow the evidence	Chapters regrouped by shared mechanism	The same regrouping; subtypes dropped	**Architecture moved, validators did not**
Raise reliability	Field trials in routine clinics, reported openly	Field studies across many countries	**Measured honestly, and modest**
Serve the user	Kept criteria with counts and durations	Essential features written as prose	**ICD-11 chose the clinician**
Admit dimensions	Severity specifiers; trait model kept apart	Personality rebuilt as severity plus traits	Carried further by ICD-11
Reduce harm	Bereavement exclusion removed	Gender incongruence moved out of the chapter	Met in part, contested in both

- **What a revision is for.** To move the manual closer to nature where evidence allows, to keep it usable by the people who must apply it, and to stop it doing harm. The three pull against each other, so every revision is a compromise.
- **The charge that sticks.** Neither revision produced a diagnostic validator, and comorbidity is still the rule, which is what a valid categorical system should not produce.

PITFALL

Answering with a list of what changed. The stem asks for a purpose and then for a verdict, so every change named has to be tied to the purpose it served and judged against it. A changed manual is not by itself an achieved purpose.

WHAT EARNS THE MARKS

- **Purposes named before any change is described.** The stem asks for a view, and a view needs a criterion.
- **The structural contrast stated,** counted criteria against prose essential features.
- **A verdict per purpose,** rather than one verdict on the whole revision.
- **The missing validator named.** It is the honest answer to the second half.

SEE ALSO Slot I - 91 the neo-Kraepelinians and the current system Slot I - 92 spectrum concepts in psychiatry

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

I - 94

Define delusion. What are primary delusions? Discuss dimensions of delusions. [2+3+5]

2 + 3 + 5 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The definition	Three criteria, stated as criteria
3 Primary delusions	Un-understandable, and the forms it takes
4 The dimensions	Each one varying free of the others
1 Why they matter	Insight, function, and where the risk sits

THE DEFINITION

A false belief held with extraordinary conviction and incomparable subjective certainty, not open to counter-argument or to the evidence of experience, and out of keeping with the person's social, cultural and educational background. Jaspers named three external criteria: certainty, incorrigibility, impossibility of content.

- **Delusional mood.** An oppressive sense that something momentous is under way, before any belief has formed. It settles when the delusion crystallises.
- **Delusional perception.** A normal percept given a private and unmediated significance: two members, the percept and the meaning. A first-rank symptom.
- **Sudden idea, and delusional memory.** A belief arriving fully formed with no trigger, one member only; and a real memory recalled as carrying a meaning it never had.
- **Primary against secondary.** A primary delusion is un-understandable: it cannot be derived from mood, personality or circumstance. A secondary one can, and is better called a delusion-like idea.

DIMENSION	WHAT IT RANGES OVER	WHY IT IS ASKED FOR
Conviction	Passing doubt through to unshakeable certainty	**Tracks insight, and predicts response to treatment**
Extension	One corner of life through to every part of it	Predicts functional impairment
Bizarreness	Culturally possible through to physically impossible	**Rated unreliably, so it no longer stands alone as a criterion**
Disorganisation	A single fragment through to a systematised structure	Systematised or fragmentary, and how far it is elaborated
Pressure	An occasional thought through to constant, driven preoccupation	**Later work added distress and action, the item that carries risk**

WHAT EARNS THE MARKS

- **The three criteria named as criteria.** Stopping at false belief is half a mark.
- **Un-understandability given as what makes a delusion primary.**
- **The five dimensions named as a set:** conviction, extension, bizarreness, disorganisation, pressure.
- **Independence stated.** Total conviction with no distress and no action is common.

SEE ALSO Slot I - 96 true and pseudo hallucinations Slot I - 95 consciousness and its disturbances

I - 95

Define consciousness. Write the dimensions and quantitative changes in consciousness. [2+4+4]

2 + 4 + 4 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The definition	Awareness of self and surroundings, on two axes
3	The dimensions	Level, content, field, and awareness of self
4	Quantitative changes	The ladder, each rung told from the one above
1	How it is scored	Three subscales, and the total they give

THE DEFINITION

Consciousness is awareness of the self and of the environment. Clinically it runs on two axes that vary independently: the level of arousal, a brainstem and thalamic function of the ascending reticular activating system, and the content of awareness, which is cortical.

- **The dimensions.** Level of arousal, the quantitative axis. Content of awareness, the qualitative axis. The field, which narrows in twilight and dissociative states. And awareness of self: Jaspers' activity, unity, identity over time, and self against not-self.
- **How it is scored.** The Glasgow Coma Scale rates eye opening one to four, verbal response one to five and motor response one to six, giving a total of three to fifteen.

LEVEL	AROUSAL AND RESPONSE	WHAT TELLS IT FROM THE RUNG ABOVE
Full alertness	Awake, attention sustained and directable	The baseline the rest are measured against
Clouding	Threshold raised, attention and comprehension fluctuate	**Fluctuation across hours: the core of delirium**
Drowsiness	Rousable by ordinary speech, then drifts back	Rouses without a physical stimulus
Stupor	Responds only to vigorous or painful stimulation	**Reflex and postural responses are kept**
Coma	Unrousable, no purposeful response to anything	**No sleep-wake cycle at all**

THE WORD STUPOR

Psychiatry and neurology use it differently: in psychiatry akinesis and mutism with consciousness preserved, as in catatonia; in neurology a depressed level of arousal. Rate arousal and awareness separately, as the locked-in syndrome shows.

WHAT EARNS THE MARKS

- **Arousal and awareness separated in the definition itself.**
- **Every rung named with what distinguishes it** from the rung above.
- **Jaspers' four aspects of self-awareness listed,** the part most answers omit.

SEE ALSO Slot I - 47 the mini mental state examination Slot I - 94 delusion, defined and dimensioned

I - 96

Define hallucinations. How do true and pseudo hallucinations differ? What is their clinical significance? [2+5+3]

2 + 5 + 3 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Both, defined**	A percept without an object, and the two exclusions
4	**The grid**	Space, quality, insight, and where each is seen
2	**The disagreement**	Two traditions, and they do not overlap
2	**Clinical significance**	Modality points to cause; not all are illness

THE DEFINITION

A hallucination is a percept without an object: a sensory experience arising with no external stimulus, in external objective space, carrying the force and clarity of a true perception. It is not an illusion, which misperceives something real.

DOMAIN	TRUE HALLUCINATION	PSEUDOHALLUCINATION	WHAT SETTLES IT
Where it is located	External objective space	Inner subjective space	**The space it occupies, Jaspers' test**
Sensory quality	Concrete, as a real percept is	Figurative, without substance	**Concreteness, not vividness**
Insight	Usually absent, held as real	Usually present	**Separates them in the British usage only**
Where it is seen	Psychotic and organic states	Dissociative states, bereavement, trauma	Suggestive, never decisive

Amber marks the discriminators. Both are involuntary, so control settles nothing between them.

- **Two traditions, not one.** Jaspers separates the two by sensory quality and by the space they occupy, not by insight. The later British usage separates them by preserved insight. The two do not pick out the same experiences.
- **Modality points to the cause.** Auditory verbal in schizophrenia; visual with fluctuating consciousness in delirium; olfactory and gustatory in temporal lobe epilepsy; tactile in stimulant use and alcohol withdrawal; complex visual with insight kept in Charles Bonnet syndrome.
- **Not all are pathological.** Hypnagogic and hypnopompic hallucinations, those of bereavement and those of sensory deprivation occur in health.

WHAT EARNS THE MARKS

- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **The two traditions named and told apart.** One definition is not enough here.
- **Modality tied to aetiology,** which is most of the significance mark.
- **Command hallucinations flagged for risk,** and the healthy forms kept out.

SEE ALSO Slot I - 94 delusion, defined and dimensioned Slot I - 95 consciousness and its disturbances **SPRINT** Day 6, psychometry, assessment and descriptive psychopathology

AREA 12 OF 12 · P1-A03

Neuroendocrinology and basic psychopharmacology

4	2.8%	I-97	I-100
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 3

REVISION ORDER

The two stress slots together, they are one answer split at the immune limb. The pineal gland next, and it is short. The evidence-based medicine slot last: it is three terms in one question and it rewards precision rather than length.

WHAT IS IN THIS AREA

Stress in two slots, one asking for the neurobiology with a critical evaluation of coping, and one for the types with their effect on the immune system. The pineal gland. And the slot that runs evidence-based medicine into personalised medicine and pharmacogenetics. The registered name of this area carries basic psychopharmacology, and its one pharmacology slot is a pharmacogenetics question rather than a drug-class question. Four slots, and the area is described here by what they hold.

I - 97

Pineal gland. [10]

10 NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What it is	Position, and the two things that make it unusual
3 Light to melatonin	The pathway, then the synthetic chain
3 Receptors and actions	Two receptors, and what each one does
2 Clinical relevance	The drugs, the disorders, the tumour

THE DEFINITION

A small unpaired midline endocrine gland of the epithalamus, sitting behind the third ventricle. Two features carry the answer: it has no direct nerve supply from the brain, and it lies outside the blood brain barrier. It converts a light signal into a hormonal one.

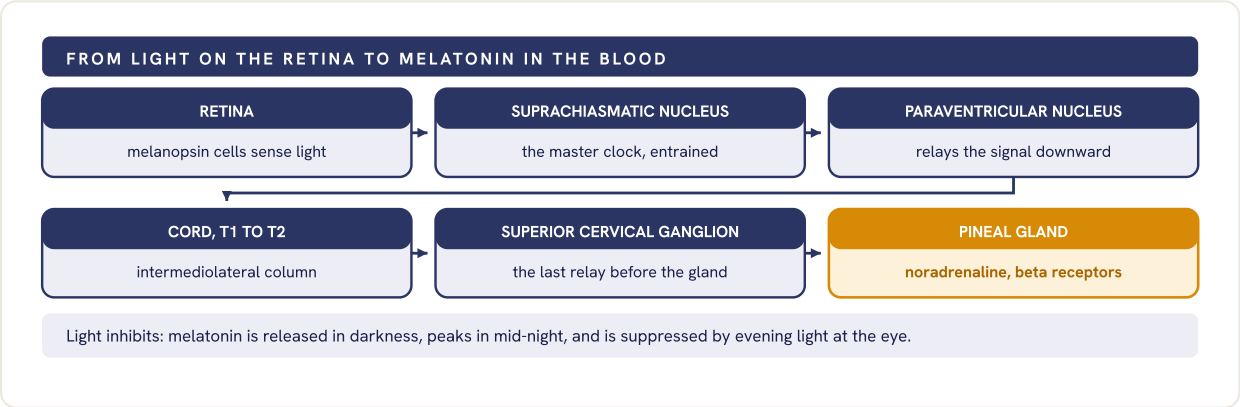


Fig 097.1 A sympathetic pathway with a hypothalamic beginning: the only route from the eye to the gland runs out of the brain, down the cord and back up the neck.

- **Synthesis.** Tryptophan to serotonin, then serotonin to N-acetylserotonin by arylalkylamine N-acetyltransferase, the rate limiting and dark-driven step, then to melatonin. Melatonin is not stored: it diffuses out as it is made.
- **Receptors and actions.** MT1 and MT2, both inhibitory and G protein coupled. MT1 damps the wake signal; MT2 shifts the phase. Melatonin re-times sleep more than it sedates.
- **Where it appears in exams.** Melatonin and ramelteon at MT1 and MT2; agomelatine, an agonist there and a 5-HT2C antagonist. Delayed sleep-wake phase disorder, free-running rhythm in blindness, jet lag. Calcification as a midline marker, and pineal tumours causing Parinaud syndrome.

WHAT EARNS THE MARKS

- **The two unusual features stated early.** No direct central innervation, and outside the blood brain barrier.
- **The whole pathway, eye to gland,** including the trip through the cord and the superior cervical ganglion.
- **The rate limiting enzyme named,** and the point that darkness drives it.
- **Phase shifting separated from sedation.** It is why melatonin is dosed by clock time, not by severity.

SEE ALSO Slot I - 03 neurobiology of sleep and wakefulness Slot I - 09 sleep stages and polysomnography **SPRINT** Day 2, Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology

I - 98

Define stress. Discuss neurobiology of stress. Critically evaluate the efficacy of different coping mechanisms in stress management. [2+3+5]

2 + 3 + 5 | NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	One physiological, one transactional
3 Neurobiology	A fast axis, a slow axis, and the brake on both
5 Coping, evaluated	Match to controllability, then judge the evidence

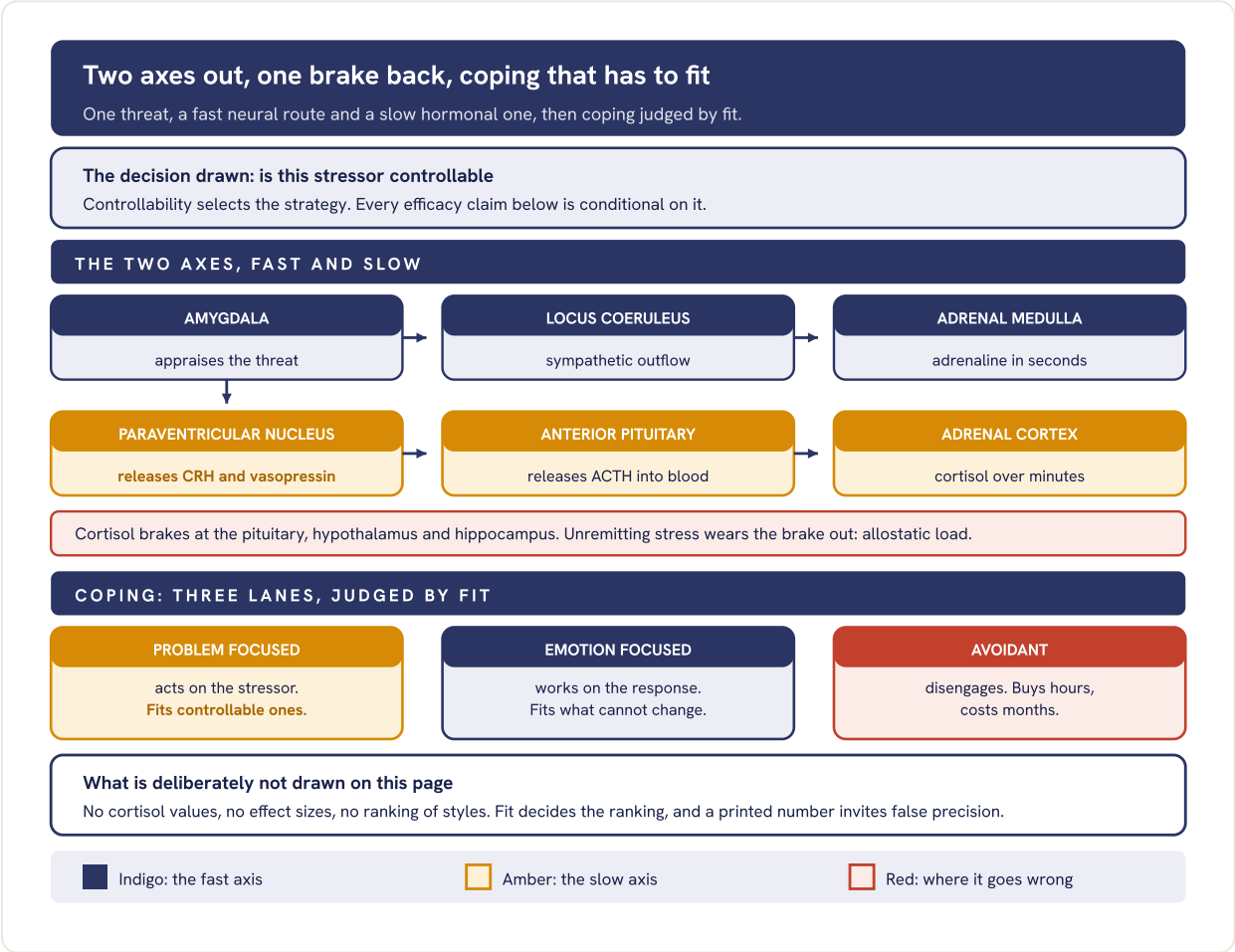


Fig 098.1 The two efferent axes drawn to one width so their timescales compare, with the brake and the coping lanes below.

COPING STYLE	WHAT THE PERSON DOES	WHERE IT WORKS	WHERE IT FAILS
Problem focused	Plans, seeks information, acts	**Controllable stressors: workload, illness self-management**	Effort on what cannot change raises distress
Emotion focused	Reappraises, accepts, relaxes, seeks support	**Uncontrollable ones: bereavement, terminal illness**	When it replaces action that was possible
Meaning focused	Reframes within values and goals	Chronic adversity, caregiving	When it minimises a real threat
Avoidant	Denies, distracts, uses substances	Briefly, in the first hours	**Over time: the most consistent predictor of poor outcome**

The amber cells are the goodness of fit rule. Avoidance reverses: what helps at hour one harms by month three.

- **The two definitions to give.** Physiological: the non-specific response of the body to any demand, through alarm, resistance and exhaustion. Transactional: a relationship appraised as taxing or exceeding the person's resources.
- **What critically evaluating means here.** Most coping research is cross-sectional and self-reported, the categories overlap, and stress management trials report modest, short-lived effects. Then say what stands: exercise, relaxation, mindfulness based stress reduction, stress inoculation.

WHAT EARNS THE MARKS

- **Both definitions.** The response without the appraisal is a half answer.
- **Both axes with their timescales,** and the brake named at all three levels.
- **The goodness of fit rule,** not styles ranked best to worst.
- **One honest limit on the evidence.** The word critically is asking for it.

SEE ALSO Slot I - 100 stress, immunity and psychiatric disorder Slot I - 29 classification of defence mechanisms

SPRINT Day 2, Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology

I - 99

What is evidence based medicine? What is personalized medicine in psychiatry? Discuss pharmacogenetics. [2+3+5]

2 + 3 + 5 | NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Evidence based medicine**	The definition, its three components, the five steps
3	**Personalised medicine**	What it means, and what it is in psychiatry today
3	**Pharmacogenetics that works**	Metabolism genes, and the one safety gene
2	**Where the evidence stops**	Response prediction, and why it is not in clinic

THE DEFINITION TO WRITE FIRST

Evidence based medicine is the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients. Three components, all required: research evidence, clinical expertise, and the patient's values.

GENE	WHAT IT CHANGES	WHAT FOLLOWS CLINICALLY	HOW STRONG THE EVIDENCE IS
CYP2D6	Clearance of risperidone, aripiprazole, venlafaxine, tricyclics	Poor metabolisers accumulate drug; ultrarapid metabolisers are underexposed	**Established, with published dosing guidance**
CYP2C19	Clearance of escitalopram, citalopram, sertraline, some tricyclics	Same direction, so dose is adjusted at the two extremes	**Established, with published dosing guidance**
HLA-B*15:02	Risk of carbamazepine Stevens-Johnson syndrome	Test before starting carbamazepine in people of South and East Asian ancestry	**Established, testing recommended**
SLC6A4, HTR2A	Proposed prediction of antidepressant response	Not usable to choose a drug	Inconsistent across studies

The amber column is the whole answer: pharmacokinetic genes and one safety gene have earned their place, response prediction has not.

- **Evidence based medicine in five steps.** Ask an answerable question, acquire the best evidence, appraise it for validity and effect size, apply it alongside expertise and patient values, and assess the outcome.
- **What personalised medicine is not.** It is not a genetic test that picks the drug. In psychiatry it is mostly clinical: staging, phenotype, previous response, family history, comorbidity and measurement based care, with pharmacogenetics contributing at the metabolic extremes and one pre-treatment safety test.

WHAT EARNS THE MARKS

- **All three components of the definition.** Evidence alone is the commonest half answer.
- **Pharmacokinetic genes separated from pharmacodynamic ones,** and response prediction called not yet usable rather than promising.
- **HLA-B*15:02 before carbamazepine,** named as the one test with a clear ancestry-based indication.

SEE ALSO Slot I - 56 genome wide association studies Slot I - 84 clinical trial registry of India **SPRINT** Day 3, Psychiatric Genetics

I - 100

Enumerate the types of stress and its effect on the immune system and psychiatric disorders. [10]

10 | ASKED AT 15 MARKS | NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The types	Name the axis first: duration, and whether there is an endpoint
3	How stress reaches immunity	Three routes, hormonal, neural and behavioural
3	What changes	Direction depends on the type, and that is the mark
2	Psychiatric consequence	Association, then the one clean causal example

TYPE OF STRESS	WHAT IT LOOKS LIKE	IMMUNE DIRECTION	EXAMPLE
Acute time limited	Minutes, with a clear end	Innate immunity mobilised, natural killer cells rise	A viva, a public talk
Brief naturalistic	Days, a real short stressor	Shift away from cellular toward humoral immunity	Examination week
Event sequences	One event with a train of consequences	Mixed, and it changes over the course	Bereavement, job loss
Chronic	No endpoint in sight, identity altered	**Broad suppression: killer cell cytotoxicity and lymphocyte proliferation fall, wounds heal slowly**	Caregiving, poverty
Distant	Long past, effects persist	**Low grade inflammation with glucocorticoid resistance**	Childhood adversity

The axis is duration and whether the stressor has an endpoint. The amber rows are where the direction reverses, and stating that reversal is what separates this answer from a list. Never write that stress suppresses immunity: it depends on the row.

- **How stress reaches the immune system.** Three routes. Hormonal, through cortisol on lymphocytes. Neural, through sympathetic fibres that directly innervate the spleen, thymus and lymph nodes. Behavioural, because stressed people sleep less, drink more, eat worse and attend less.
- **The paradox worth stating.** Cortisol is anti-inflammatory, so chronic stress ought to damp inflammation. It does the opposite, because immune cells become glucocorticoid resistant and inflammatory signalling comes off the brake.
- **Psychiatric consequence.** Raised C reactive protein and interleukin 6 are associated with depression and with poorer antidepressant response. The cleanest causal evidence is different: interferon alfa given for hepatitis produces depressive illness in previously well people.

WHAT EARNS THE MARKS

- **The types enumerated with a stated axis,** duration and whether the stressor has an end.
- **Both directions given.** Up in acute, down in chronic, is the discriminating sentence.
- **The three routes named,** hormonal, neural and behavioural, not cortisol alone.
- **Association separated from causation,** with interferon alfa offered as the causal case.

SEE ALSO Slot I - 98 neurobiology of stress and coping Slot I - 08 biological markers in psychiatric illness **SPRINT** Day 2, Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology

PAPER I APPENDIX, PAGE ONE OF TWO

Ten sets, ten sittings: Paper I

The same hundred Paper I questions, dealt into ten papers of ten. Each cell gives the slot and the page it is answered on.

Read down a row to sit a paper. The deal is not random: the hundred are ranked by score and dealt one question from each rank decile into each set, so every set holds one of the ten strongest, one of the ten weakest, and one from each band between. That is why the sets are comparable to each other and why none of them is an easy paper.

SET	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10
I-S1	I-85 p 117	I-46 p 68	I-19 p 36	I-21 p 38	I-29 p 47	I-31 p 50	I-94 p 129	I-65 p 91	I-75 p 104	I-89 p 122
I-S2	I-71 p 99	I-15 p 32	I-08 p 22	I-09 p 23	I-10 p 24	I-42 p 63	I-95 p 130	I-24 p 41	I-61 p 86	I-88 p 121
I-S3	I-45 p 67	I-05 p 19	I-55 p 79	I-48 p 70	I-63 p 89	I-64 p 90	I-86 p 119	I-44 p 65	I-51 p 74	I-78 p 107
I-S4	I-36 p 56	I-04 p 18	I-20 p 37	I-39 p 59	I-11 p 25	I-13 p 28	I-43 p 64	I-74 p 103	I-52 p 75	I-70 p 97
I-S5	I-79 p 109	I-18 p 35	I-28 p 46	I-47 p 69	I-23 p 40	I-30 p 49	I-32 p 51	I-67 p 93	I-62 p 87	I-77 p 106
I-S6	I-01 p 15	I-17 p 34	I-98 p 134	I-38 p 58	I-49 p 72	I-12 p 27	I-50 p 73	I-34 p 53	I-53 p 76	I-76 p 105
I-S7	I-02 p 16	I-27 p 45	I-06 p 20	I-72 p 100	I-99 p 136	I-41 p 62	I-56 p 80	I-66 p 92	I-35 p 54	I-87 p 120
I-S8	I-03 p 17	I-16 p 33	I-07 p 21	I-92 p 127	I-81 p 111	I-84 p 115	I-57 p 81	I-59 p 84	I-68 p 94	I-69 p 95
I-S9	I-54 p 78	I-26 p 44	I-91 p 125	I-22 p 39	I-40 p 60	I-83 p 114	I-73 p 102	I-25 p 42	I-60 p 85	I-90 p 123
I-S10	I-14 p 30	I-97 p 133	I-37 p 57	I-80 p 110	I-93 p 128	I-82 p 112	I-33 p 52	I-58 p 83	I-96 p 131	I-100 p 137

No set repeats a question, a merged variant of a question, or a near-duplicate, and no set takes more than three questions from any one area.

PAPER I APPENDIX, PAGE TWO OF TWO

How to sit one of these

SET	MARKS	MEAN SCORE	AREAS	ANCHOR OR HIGH	HEAVIEST AREAS IN THE SET
I-S1	100	0.416	7	2	Developmental psychology 2; Functional neuroanatomy 2; Personality theory 2
I-S2	100	0.410	7	3	Neurochemistry 3; Functional neuroanatomy 2; Biostatistics 1
I-S3	100	0.402	7	3	Psychometry 3; Social 2; Neurochemistry 1
I-S4	100	0.401	6	3	Learning 3; Neurochemistry 3; Functional neuroanatomy 1
I-S5	100	0.402	7	3	Personality theory 3; Functional neuroanatomy 2; Research methodology 1
I-S6	100	0.402	7	4	Psychometry 3; Neurochemistry 2; Functional neuroanatomy 1
I-S7	100	0.402	8	3	Neurochemistry 2; Personality theory 2; Biostatistics 1
I-S8	100	0.402	6	3	Neurochemistry 2; Research methodology 2; Genetics in psychiatry 2
I-S9	100	0.392	8	2	Genetics in psychiatry 2; Functional neuroanatomy 2; Personality theory 1
I-S10	100	0.392	7	3	Neuroendocrinology 2; Research methodology 2; Nosology 2

Mean score is the selection score of the ten questions in that set, on the same scale used across all four books. The spread between the strongest and the weakest set is small by construction.

Three hours, ten questions, one hundred marks. That is the shape of the real paper, so it is the shape of these. Eighteen minutes a question leaves you twenty minutes at the end, which is roughly what you will have and roughly what you will need.

Write the skeleton first, for all ten. Spend the first twenty minutes writing nothing but mark splits and section headings across all ten answers. The map on each page shows what that skeleton should look like, so you can mark your own against it before you have written a line of content. Most marks lost in this examination are lost to allocation, not to ignorance.

Then mark yourself against the anchors. Every map ends with three or four things an examiner ticks. Those are the marking scheme, as close as this book gets to one. Count how many you hit before you read the page, because reading it first will convince you that you knew it.

Sit them in order, or sit them backwards. Set one holds the strongest question in the book and set ten the weakest, but the decile deal keeps the mean scores close, so any set is a fair paper. In order simply means the first one you sit is the one most like the paper you will get.

What a set is not. It is not a mock paper from any board, it is not a leak, and it is not a prediction. It is ten questions a board set, arranged so that ten of them look like a paper. Sit five and you will have written a hundred answers in the shape the examination wants, which is the only preparation that has ever reliably worked.

PART TWO OF FOUR

PAPER II

Psychoses, Mood and the Adult Disorders

The adult clinical paper: schizophrenia and the other psychoses, mood disorders, anxiety and obsessive-compulsive presentations, substance use, the eating, sleep and sexual disorders, and the treatments that answer them.

14	100	78	8	9.7%
AREAS, IN WEIGHT ORDER	ANSWER MAPS	SITTINGS COUNTED	EXAMINING BOARDS	HEAVIEST AREA SHARE

ANCHOR 15 · HIGH 13 · RECURRENT 5 · SINGLE 67

Heaviest area in this paper: Eating, sleep and sexual disorders. The weightage chapter overleaf shows how every area in Paper II was weighted, and what the weight could not promise.

THE WEIGHTAGE CHAPTER, PAGE ONE OF THREE

Where the marks have gone

Nothing in this book is a prediction. It is a count of 78 sittings from 8 examining boards, and a statement of where those boards have put their marks in Paper II.

Fourteen areas, weighted. The university boards are pooled by how much each is trusted, DNB is held out and given a fixed thirty percent, and the resulting weight becomes a share of one hundred and then a whole number of slots. The ledger of which boards were counted, how their papers reached us, and how far each one is trusted, is on the page opposite.

AREA, IN WEIGHT ORDER	UNIV POOL	DNB	WEIGHT	SHARE	SLOTS
01 Eating, sleep and sexual disorders	0.392	0.301	0.365	9.67%	9
02 Substance use: opioids, other drugs and treatment	0.304	0.470	0.354	9.39%	9
03 Anxiety, OCD and related disorders	0.318	0.380	0.337	8.93%	9
04 Schizophrenia: aetiology, phenomenology and course	0.270	0.432	0.318	8.45%	8
05 Trauma, dissociative and somatic symptom disorders	0.282	0.400	0.317	8.42%	8
06 Schizophrenia: treatment and antipsychotics	0.244	0.474	0.313	8.31%	8
07 Mood disorders: pharmacotherapy	0.243	0.377	0.283	7.51%	7
08 Mood disorders: syndromes and nosology	0.280	0.284	0.281	7.45%	7
09 Psychotherapies	0.226	0.344	0.261	6.93%	7
10 Personality disorders	0.243	0.268	0.251	6.65%	7
11 Clinical assessment and cross-cutting nosology	0.209	0.199	0.206	5.46%	6
12 Substance use: general concepts and alcohol	0.164	0.183	0.170	4.50%	5
13 Other psychoses and catatonia	0.203	0.067	0.163	4.31%	5
14 Suicide, self-harm and impulse control	0.166	0.116	0.151	4.01%	5

Univ pool is the trust-weighted verdict of the seven university boards; DNB is the national paper, held out. Weight = 0.70 times the pool plus 0.30 times DNB. Share is that weight as a fraction of Paper II. Slots are the share turned into whole questions by largest remainder, with a floor of two and a cap of twenty; Paper II reached neither.

THE WEIGHTAGE CHAPTER, PAGE TWO OF THREE

How the hundred were picked

BOARD	SITTINGS COUNTED	HOW THE PAPERS REACHED US	TRUST
RGUHS	15	Hand transcription and public mirrors of the printed papers	0.806
KUHS	9	Official university portal PDFs	0.600
TN-MGRMU	35	Public archive of the printed papers, exact sitting dates recovered	0.480
KNRUHS	3	Official university portal PDFs	0.300
MUHS	2	Two watermark-confirmed papers, read by OCR. A third-party recurrence compilation was tested against them, matched none, and was removed from the sitting count	0.267
NTRUHS	2	Mirror text of the printed papers	0.160
WBUHS	2	Public archive of the printed papers	0.180
DNB	10	Official NBE material; 32 of 40 files verified by checksum against the board's own published path	fixed 0.30

Trust is how far a board sets the university pool: its standing, its number of sittings, and the quality of its evidence. DNB sits outside the pool. A third-party compilation claiming thirteen further MUHS sittings was tested against the only confirmed MUHS papers inside its own window, matched none of them, and was removed from this ledger; its topic distribution still contributes to area mass at reduced quality.

1 Count the sittings

78 sittings from 8 boards, each weighted so that it halves roughly every six years and is reduced again when the paper reached us as a mirror, an archive or a transcription rather than an official file.

2 Weight the areas

Each board reads each area twice: breadth, how often it examines there, and mark share, how much of its marks land there. The two are averaged, the boards are pooled by trust, and DNB is held out at a fixed thirty percent.

3 Turn weight into slots

The fourteen weights become shares of one hundred, then whole slots by largest remainder, with a floor of two and a cap of twenty so no area disappears and none swallows the book.

4 Score every eligible question

Four components: weighted recurrence (45 per cent), recency (30), how many boards have asked it (15), and how well it is shaped as a ten-mark question (10). Normalised across all four papers, so one score means one thing everywhere.

5 Fill the slots

The highest scorers in each area, up to that area's slot count, near-duplicates merged first. All 100 were filled from questions actually set: 15 anchor, 13 high, 5 recurrent, 67 single-appearance.

THE WEIGHTAGE CHAPTER, PAGE THREE OF THREE

What this book cannot promise

A frequency count is a strong prior, not a prophecy. Here is exactly what that sentence costs, stated before you rely on it.

It does not predict a paper. Nothing here says what your board will set. It says where the marks have gone across 78 sittings, which is a different claim and a smaller one. A board that changes its examiner, its syllabus or its habits can leave every number in this book behind in one sitting.

Most questions were seen once. Of the 440 selectable Paper II questions in the record, 384 appeared in a single sitting. That is not a defect in the counting, it is what happens when eight boards set different papers: the same TOPIC returns constantly while the same QUESTION rarely does. So the selection is driven by AREA weight, where the signal is dense, and each map prints a band rather than a frequency claim about its own question.

The band on a map is a description, not a forecast. Anchor means this question has come back across boards and years. Single means it was set once in the record we hold. A single-appearance question is here because its AREA is heavy, and it is a fair worked example of that area, which is the only thing it is offered as.

Nothing here is invented. All 100 slots in this book are questions a board actually set. Where a paper in this series cannot fill its slots from observed questions, the shortfall is authored to syllabus and labelled as such on the map. Paper II has no such slots.

The record is uneven, and openly so. Two of the eight boards set papers in a legacy structure that does not map onto the modern four, so their sittings are counted against all of their papers rather than one. Two more contribute only two sittings each. The trust column on the first page of this chapter is where that unevenness is priced in, and it is the number to argue with if you want to argue with the weighting.

No board has endorsed this. Board names appear on every page because a count that hides its sources is not a count. None of them was asked, and none of them is responsible for anything in this book.

AREA 01 OF 14 · P2-A11

Eating, sleep and sexual disorders

9	9.7%	II-01	II-09
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 4 RECURRENT 1 SINGLE 4

REVISION ORDER

The three impotence slots first, and together: they share one differential and one management spine, and the classification slot frames all of them. Anorexia and bulimia next, as one comparison. Gender identity, sex reassignment and the offences slot last.

WHAT IS IN THIS AREA

Seven of the nine slots are sexual: erectile dysfunction and psychogenic impotence across three of them, the classification of male sexual dysfunction, gender identity and gender dysphoria, sex reassignment surgery, and the sexual offences slot. Eating takes the other two, anorexia nervosa and bulimia nervosa. The registered name of this area also carries sleep, and no sleep question survived selection into it. Nothing on the pages that follow is a sleep question, and the area is described here by what it actually holds.

II - 01

Discuss the clinical features and complications of anorexia nervosa. [10]

10 EATING, SLEEP AND SEXUAL DISORDERS

KNRUHS, NTRUHS, WBUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 **Diagnostic core**	Low weight, fear of gain, disturbed body experience
3 **Clinical features**	Behaviour, cognition, and what starvation itself makes
4 **Complications**	System by system, then refeeding
1 **Outcome**	Mortality and the two things that drive it

THE DEFINITION

Restriction of intake producing significantly low weight for age, sex and developmental stage, with fear of weight gain or behaviour that prevents it, and disturbed experience of weight or shape. ICD-11 uses body mass index below 18.5 in adults and has dropped amenorrhoea. DSM-5-TR grades severity on body mass index and names restricting and binge-purge subtypes.

- **Behaviour and cognition.** Dietary rules, driven exercise, purging, food rituals, weighing; overvaluation of shape, denial of seriousness, poor insight.
- **Starvation is a cause, not only an effect.** Low mood, irritability, obsessionality, social withdrawal and preoccupation with food improve with weight restoration.

SYSTEM	WHAT IS FOUND	WHY IT MATTERS
Cardiac	Bradycardia, hypotension, prolonged QT interval, pericardial effusion	**Arrhythmia is a leading cause of death**
Electrolyte	Hypokalaemia; hypochloraemic alkalosis with vomiting; hyponatraemia with water loading	**Drives the arrhythmia risk**
Endocrine	Low triiodothyronine, hypercortisolaemia, hypogonadotropic hypogonadism, osteoporosis	Bone loss may not fully reverse
Gut and liver	Delayed emptying, constipation, raised transaminases, superior mesenteric artery syndrome	Explains bloating on refeeding
Marrow	Anaemia, leucopenia, gelatinous marrow transformation	Reverses with weight gain
Refeeding	Falling phosphate, potassium and magnesium; thiamine depletion; fluid overload	**Iatrogenic, and the one to anticipate**

Amber cells are what an examiner is looking for: the complications that change what you do today, not the ones that merely lengthen the list.

WHAT EARNS THE MARKS

- **Both current criteria named,** with the amenorrhoea point stated as a change rather than assumed.
- **Complications grouped by system,** not listed flat. The grid is the answer.
- **Refeeding syndrome as a separate row,** with phosphate named and thiamine given before calories.
- **Mortality attributed** to cardiac events and to suicide, which is the line most answers omit.

SEE ALSO Slot II - 07 bulimia nervosa SPRINT Day 17, Eating and sleep-wake disorders

II - 02

Management of erectile dysfunction. [10]

10 EATING, SLEEP AND SEXUAL DISORDERS

KUHS, MUHS, NTRUHS, RGUHS, TN-MGRMU - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

3 Assessment	History, vascular and endocrine screen, drug review
5 The ladder	Modify, then oral, then psychosexual, then second line
2 Special situations	Drug induced, and the Indian couple context

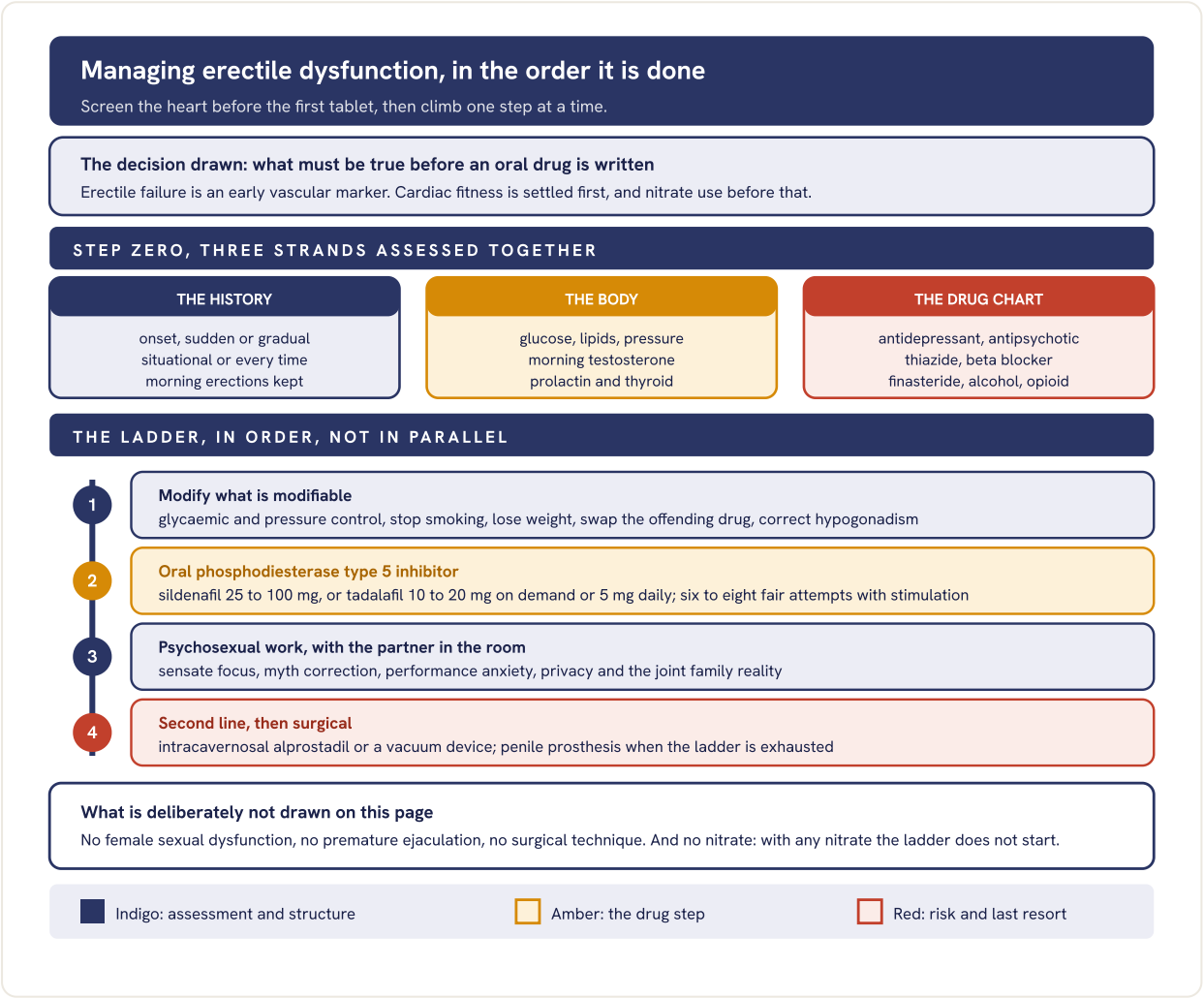


Fig 002.1 Three strands assessed together, then a four-rung ladder climbed in order, the nitrate question settled before rung two.

- **Drug induced.** Reduce the dose, switch to bupropion or mirtazapine, or add an oral inhibitor. For antipsychotics, check prolactin.

PITFALL

Calling the oral drug a failure too early. Most apparent non-response is an inadequate trial: no stimulation, a full stomach with sildenafil, or the lowest dose tried twice.

WHAT EARNS THE MARKS

- **Cardiac fitness and nitrate use settled before the prescription,** written as step zero.
- **The ladder in order,** with a named drug, a dose, and what counts as an adequate trial.
- **The partner in the plan.** Treating the man alone loses the psychosexual marks.

SEE ALSO Slot II - 03 psychogenic impotence Slot II - 09 male sexual dysfunction **SPRINT**

Day 18, Sexual, impulse-control disorders and suicide

II - 03

Define psychogenic impotence as per the current diagnostic systems. Discuss its differential diagnosis. Write in brief about management. [4+2+4]

4 + 2 + 4 | EATING, SLEEP AND SEXUAL DISORDERS | DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

4 **Definition**	Both systems, and the descriptors each attaches
2 **Differential**	Organic causes, and the near neighbours
4 **Management**	Couple work first, the tablet as scaffolding

THE DEFINITION, IN THE WORDS OF THE SYSTEMS

Neither system uses the word impotence. DSM-5-TR erectile disorder requires difficulty obtaining or maintaining an erection, or reduced rigidity, on most occasions for at least six months, with distress, specified as lifelong or acquired and generalised or situational. ICD-11 asks the same descriptors and adds the aetiological contributors, so psychogenic is a descriptor rather than a diagnosis.

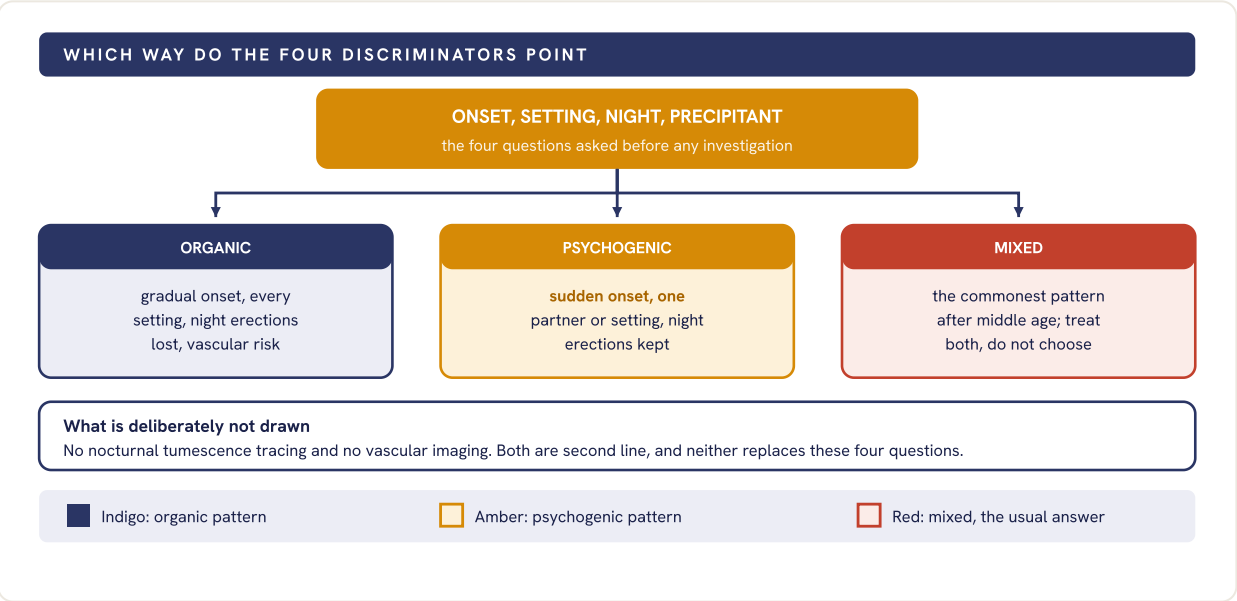


Fig 003.1 The psychogenic label rests on four bedside questions, and the honest third lane is mixed rather than either pure form.

- **Differential.** Vascular, neurogenic, endocrine and drug induced causes; early ejaculation reported as failure; low desire; depressive disorder; relationship discord; semen-loss anxiety.
- **Management.** Correct the myths, sensate focus with the partner, treat depression and anxiety, and use an oral inhibitor as short-term scaffolding.

WHAT EARNS THE MARKS

- **Naming that neither system carries the word impotence,** and giving the descriptor set instead.
- **The four discriminators from the figure,** quoted as the bedside test rather than a laboratory one.
- **Mixed aetiology named as the common answer,** which stops the artificial either or.
- **The partner in the plan,** and the tablet framed as scaffolding, not treatment.

SEE ALSO Slot II - 02 erectile dysfunction Slot II - 06 medico-legal impotence **SPRINT**
Day 18, Sexual, impulse-control disorders and suicide

II - 04

Discuss the indications of sex reassignment surgery. Describe the role of the psychiatrist in this matter. [4+6]

4 + 6 | EATING, SLEEP AND SEXUAL DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The terms	Gender-affirming surgery, and what Indian law now says
2 Indications	Persistence, capacity, age, stability, hormone time
5 The psychiatrist	Stage by stage, and what is documented at each
1 The limit	Surgery is not the gate to legal identity

THE ELIGIBILITY CRITERIA, WORTH WRITING OUT

Persistent and well documented gender incongruence; capacity to give informed consent; age of majority; reasonable control of any coexisting medical or mental health condition; and for genital surgery, a period of continuous hormone therapy. The eighth version of the international standards of care removed the fixed real life experience requirement and the routine two referral rule.

STAGE	WHAT THE PSYCHIATRIST DOES	WHAT IS RECORDED
Assessment	Confirm gender incongruence; exclude psychotic identity disturbance, body dysmorphic disorder and dissociative presentations	**Persistence, onset and course**
Comorbidity	Treat depression, anxiety, substance use and suicidality to stability, not to cure	Response and current risk
Consent	Capacity, realistic expectation, fertility and irreversibility discussed	**A capacity opinion, dated**
Family and work	Family sessions, disclosure planning, school or workplace liaison	Supports actually available
After surgery	Adjustment, pain and body image work, hormone adherence, regret screening	Follow up at fixed intervals

Amber cells are the two documents an examiner expects by name: a statement of persistence and a dated capacity opinion.

- **The Indian frame.** The 2014 Supreme Court judgment recognised the right to a self-identified gender, and the Transgender Persons Act of 2019 gives a certificate of identity through the District Magistrate. Surgery is not a precondition for that recognition.

WHAT EARNS THE MARKS

- **Eligibility written as a list of criteria,** not as a general statement of readiness.
- **The role split by stage,** which is what turns a description into a plan.
- **Stability rather than cure** as the comorbidity standard. The opposite reads as gatekeeping.
- **The statutory line,** that legal identity does not wait on an operation.

SEE ALSO Slot II - 08 gender dysphoria **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

II - 05

What are the sexual offences? Discuss management strategies in a case of pedophilia. Narrate neuropsychological findings in people convicted of sexual offences. [2+5+3]

2 + 5 + 3 | EATING, SLEEP AND SEXUAL DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The offences	Where they now sit in Indian statute
5 Management	Report, assess risk, treat, supervise, review
3 Neuropsychology	What group studies show, and what they cannot do

- **The statutes.** The Bharatiya Nyaya Sanhita of 2023 carries rape, gang rape, assault on modesty, sexual harassment, voyeurism and stalking, replacing the older penal code from July 2024. The Protection of Children from Sexual Offences Act of 2012 covers every sexual offence against a person under eighteen.
- **Pedophilic disorder is the clinical term.** Sustained sexual arousal to prepubescent children over at least six months, in a person aged sixteen or above and at least five years older, either acted upon or causing marked distress.

STEP	WHAT HAPPENS	WHO ACTS	CONDITION OR INSTRUMENT
1	Report an offence against a child	Any professional who learns of it	**Mandatory, without delay**
2	Structured risk assessment	Psychiatrist with the team	Static-99R, Stable-2007
3	Treat comorbidity and substance use	Psychiatrist	Disinhibition raises risk
4	Relapse prevention therapy	Therapist, in a group where possible	Offence chain, victim empathy
5	Drug treatment where urges are intense	Psychiatrist, with consent	**Fluoxetine 40 to 60 mg; antiandrogen with bone and metabolic monitoring**
6	Supervision and scheduled review	Team with probation or family	No unsupervised contact

Order, actor and standard in one view. Reporting duty comes first on the page, as it does in practice.

WHAT THE LAW AND THE TESTS DO NOT COVER

Neuropsychological findings are group level. Convicted samples show frontal and executive weakness, lower measured intelligence in pedophilic samples, raised rates of non-right-handedness, and imaging differences in orbitofrontal, amygdalar and temporal regions. None of it identifies an individual offender. A diagnosis is not a defence, and treatment does not replace the court process.

WHAT EARNS THE MARKS

- **Reporting duty written as step one,** before any clinical formulation.
- **Named risk instruments,** which separate a structured assessment from an impression.
- **Drug treatment placed after therapy and consent,** with the monitoring named.
- **The exclusions strip.** Saying the neuropsychology is group level is the mark most answers give away.

SEE ALSO Slot II - 06 medico-legal impotence **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

II - 06

What is impotence? What are the causes and the medico-legal aspects of impotence? What are its common psychiatric sequelae? [2+4+4]

2 + 4 + 4 | EATING, SLEEP AND SEXUAL DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The two meanings	A clinical diagnosis against a legal word
2 Causes	Four organic groups, drugs, and the psychogenic set
4 Medico-legal	Where impotence decides a marriage or a charge
2 Sequelae	The man, the marriage, and the help-seeking

- **The two meanings.** Clinically the diagnosis is erectile dysfunction. In law impotence is wider: the inability to consummate a marriage, and it is judged in relation to one spouse rather than in general.
- **Causes.** Vasculogenic, neurogenic, endocrine and anatomical; drugs including antidepressants, antipsychotics, thiazides, beta blockers, finasteride, alcohol and opioids; and psychogenic causes led by performance anxiety, depression, discord and semen-loss anxiety.

STATUTE OR SETTING	WHAT IS CLAIMED	WHO DECIDES	THE STANDARD APPLIED
Hindu Marriage Act 1955	Marriage voidable, never consummated	Civil court on medical opinion	**Relative impotence with this spouse is enough**
Special Marriage Act 1954	Same ground for nullity	Civil court	Impotence at marriage and at suit
Dissolution of Muslim Marriages Act 1939	Wife seeks dissolution	Court	Husband allowed one year to show recovery
Divorce Act 1869	Nullity for Christian marriages	Civil court	Impotence at marriage and at suit
Criminal proceedings	Accused pleads incapacity	Medical board on court order	**Establishes present capacity only**

The medico-legal marks live in the last column. Every statute here asks a different question of the same finding.

WHAT A POTENCY EXAMINATION DOES NOT SETTLE

It speaks to capacity today, not to capacity on a past date. A normal finding does not exclude situational psychogenic impotence, and failure under examination conditions is not proof of incapacity. Incapacity for intercourse also does not exclude every sexual offence, because the statutory definitions extend beyond it. The psychiatrist gives an opinion; the court makes the finding.

WHAT EARNS THE MARKS

- **The clinical and the legal meaning separated in the first two lines.**
- **Relativity stated,** that impotence with one spouse is sufficient for nullity.
- **Sequelae named as a list:** depression, anticipatory anxiety, marital discord, alcohol use, semen-loss preoccupation, secrecy and delayed help-seeking.
- **The exclusions strip,** which is what separates a clinician from a reporter of findings.

SEE ALSO Slot II - 03 psychogenic impotence Slot II - 05 sexual offences **SPRINT**

Day 18, Sexual, impulse-control disorders and suicide

II - 07

Clinical features and management of bulimia nervosa. [5+5]

5 + 5 | EATING, SLEEP AND SEXUAL DISORDERS

DNB, RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

3 **The syndrome**	Binge, compensation, and the overvaluation behind both
2 **Signs and labs**	What the body shows while the weight looks normal
4 **Treatment**	Medical risk, then psychological, then the drug
1 **Follow through**	Laxative taper, dental review, comorbidity

- **The syndrome.** Recurrent binges with loss of control, recurrent compensation by vomiting, laxatives, fasting or driven exercise, at least weekly for three months, with self-worth judged on shape and weight. Weight is usually normal, which is why it is missed.
- **Signs and labs.** Knuckle callus, parotid swelling, dental erosion, subconjunctival haemorrhage; hypokalaemia, hypochloraemic alkalosis, raised amylase; rebound oedema when laxatives stop.

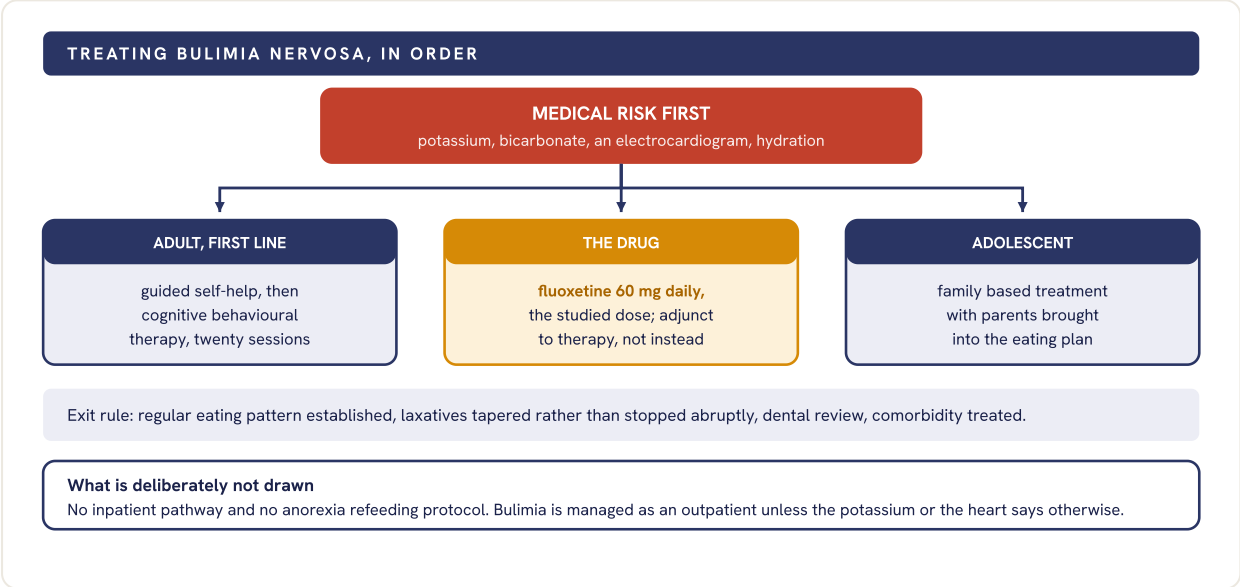


Fig 007.1 Medical risk settled first, then three treatment lanes chosen by age and by role, with the exit rule carried along the foot.

PITFALL

Reaching for bupropion. It lowers the seizure threshold and is avoided where there is purging. The second trap is reassurance about a normal weight, which is exactly what delays the diagnosis.

WHAT EARNS THE MARKS

- **Potassium and the electrocardiogram named before any therapy,** as the first line of management.
- **A named psychological treatment with a dose of sessions,** not the phrase psychotherapy alone.
- **Fluoxetine at 60 mg,** stated as an adjunct rather than a substitute.
- **The laxative taper and the dental review,** which most answers never reach.

SEE ALSO Slot II - 01 anorexia nervosa **SPRINT** Day 17, Eating and sleep-wake disorders

II - 08

What is gender identity? What are the clinical features of gender dysphoria? Briefly discuss its management. [2+3+5]

2 + 3 + 5 | EATING, SLEEP AND SEXUAL DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Gender identity	The term, and the three things it is not
3 Clinical features	Criteria, and how children differ from adults
5 Management	Affirm, assess, then stage the physical steps

THE DEFINITION

Gender identity is a person's internal sense of being male, female, both, neither or elsewhere on a spectrum. It is not the sex recorded at birth, not gender expression, and not sexual orientation. ICD-11 renamed the condition gender incongruence and moved it out of the mental disorders chapter into conditions related to sexual health, keeping a code for access while removing the label of disorder.

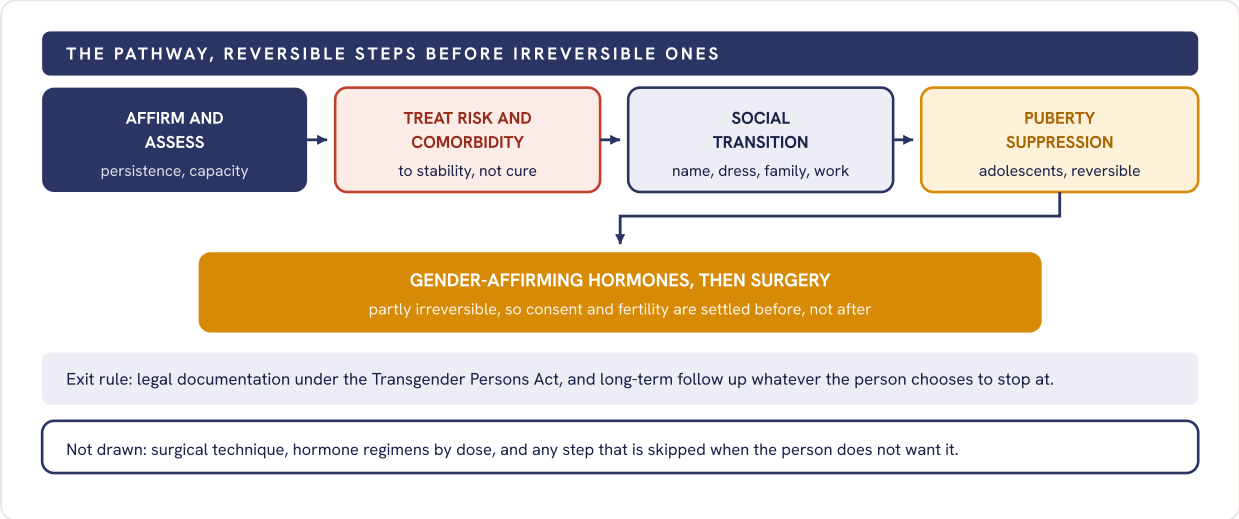


Fig 008.1 Reversible steps precede partly irreversible ones, and the person may stop at any point on the line without the pathway being incomplete.

- **Clinical features.** Marked incongruence between experienced and assigned gender for at least six months, with distress or impairment; in children, insistence on another gender with cross-gender play and aversion to one's own anatomy. Puberty is the point at which persistence becomes clearer.
- **Two things to say.** Distress often reflects minority stress rather than the identity itself. Conversion practices are unethical and have been named professional misconduct by the national regulator.

WHAT EARNS THE MARKS

- **Identity distinguished from expression and orientation** in the first two lines.
- **The ICD-11 relocation named,** which shows the current position rather than the older one.
- **Reversible before irreversible,** read straight off the figure.
- **Minority stress and the ban on conversion practices,** which carry the ethical marks.

SEE ALSO Slot II - 04 gender-affirming surgery **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

II - 09

Describe the classification of male sexual dysfunction. Discuss the approaches to their evaluation and management. [10]

10 | ASKED AT 20 MARKS | EATING, SLEEP AND SEXUAL DISORDERS

RGUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

3 Classification	By phase of the response cycle, plus the descriptors
3 Evaluation	History with the partner, examination, a short lab set
4 Management	Correct the cause, couple work, then the specific drug

PHASE	THE DYSFUNCTION	HOW IT PRESENTS	WHAT SETTLES IT
Desire	Hypoactive sexual desire	Absent or reduced desire, causing distress	Depression and testosterone checked
Arousal	Erectile dysfunction	Cannot attain or keep an erection	**Night erections kept or lost**
Orgasm, early	Early ejaculation	Ejaculation before or soon after entry, with distress	**Lifelong or acquired**
Orgasm, late	Delayed ejaculation, anejaculation	Marked delay or none, often drug related	Drug chart and diabetes
Pain	Sexual pain on penetration	Pain or fear of pain during intercourse	Examination for a local cause
Culture bound	Semen-loss anxiety	Fear of loss of vitality, often with a dysfunction alongside	**Belief, not organ, is the target**

Every diagnosis above also carries descriptors: lifelong or acquired, generalised or situational, and the contributing factors from medical condition, medication, knowledge, relationship and culture.

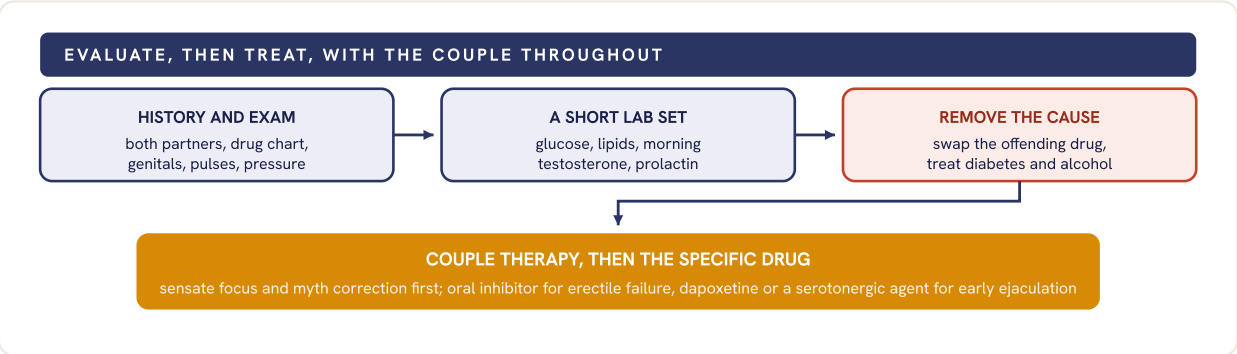


Fig 009.1 Evaluation runs left to right and converges on couple work, with the drug chosen by the phase named in the grid above.

WHAT EARNS THE MARKS

- **The response cycle used as the axis of classification,** stated before the list.
- **The descriptor set,** which is where the current systems differ from the older ones.
- **Semen-loss anxiety included,** because in Indian practice it is common and it changes the target of treatment.
- **The partner in the room,** and the drug named after the psychological step rather than instead of it.

SEE ALSO Slot II - 02 erectile dysfunction Slot II - 03 psychogenic impotence **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

AREA 02 OF 14 · P2-A07

Substance use: opioids, other drugs and treatment

9	9.4%	II-10	II-18
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 8

REVISION ORDER

Take the four opioid slots as one block; they overlap almost completely and only one of them carries the NDPS limb. Tobacco next, as a pair. Motivational enhancement therapy stands alone and is short. Caffeine and the new psychoactive substances last.

WHAT IS IN THIS AREA

Opioids across four slots (the neurobiology and psychosocial factors, substitution therapy with the NDPS Act, the naloxone challenge test with withdrawal management, and naltrexone), tobacco in two (dependence, and a locality tobacco control plan that compares gum with patch), motivational enhancement therapy, caffeine related disorders, and the new psychoactive substances.

II - 10

Describe diagnosis, clinical features and management of caffeine related disorders. [2+3+5]

2 + 3 + 5 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Diagnosis	The recognised entities, and the one still under study
3	Clinical features	The intoxication threshold and the withdrawal clock
5	Management	Taper, treat the syndrome, and the overdose lane

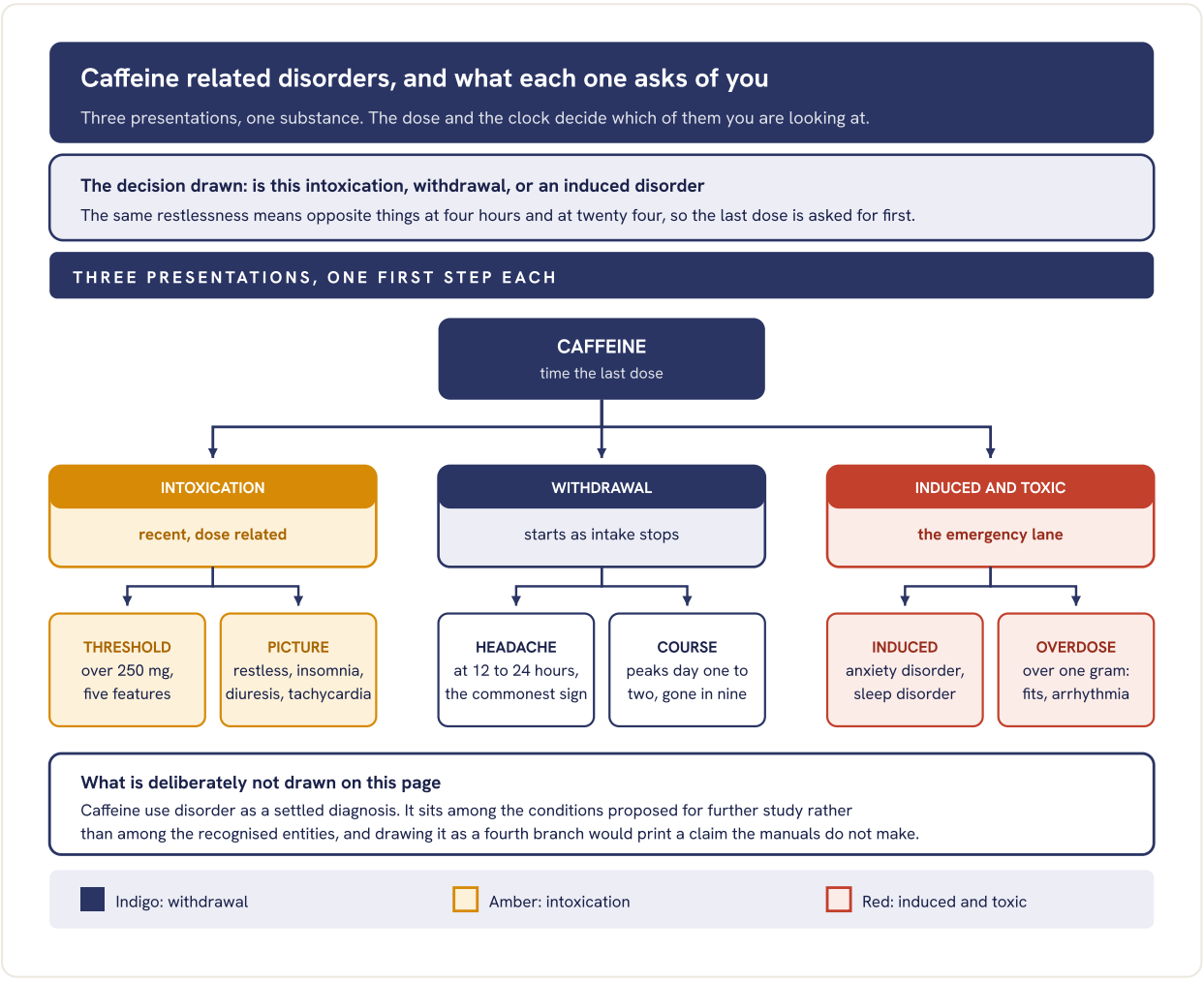


Fig 010.1 The three presentations set against the clock since the last dose, with the toxic lane kept separate because it is the only one that is managed in a resuscitation bay.

- **Management.** Withdrawal is prevented, not treated: taper by about a quarter every few days, substitute decaffeinated drinks, treat the headache simply. Intoxication needs reassurance and a stopped intake. Induced anxiety and insomnia usually clear on abstinence alone.
- **The hidden sources.** Energy drinks, colas, green tea, and combined analgesic and cold preparations. A taper planned on cups of coffee alone fails because the intake was never fully counted.

PITFALL

Do not diagnose an anxiety disorder in a heavy user without first timing the caffeine. Panic attacks are readily provoked at high intake, and the treatment is a taper, not an antidepressant.

WHAT EARNS THE MARKS

- **The recognised entities named,** with caffeine use disorder honestly placed among the conditions for further study.
- **Numbers on the page:** the 250 mg threshold, the headache at 12 to 24 hours, the gram that turns it into an emergency.
- **A taper rather than an abrupt stop,** written as the management of withdrawal.
- **Hidden sources counted,** which is what makes the plan workable.

SEE ALSO Slot II - 17 new psychoactive substances Slot II - 18 tobacco dependence **SPRINT** Day 13, Substance use disorders

II - 11

Describe the neurobiology and psychosocial factors of opioid use disorder. [4+6]

4 + 6 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4 Neurobiology	The receptor, the reward surge, the adapted state
4 Psychosocial factors	Availability, family, adversity, comorbidity
2 Where they meet	Why neither account alone predicts who becomes dependent

THE RECEPTOR, IN ONE LINE

Opioids act at the mu receptor, a G protein coupled receptor that inhibits adenylyl cyclase. In the ventral tegmental area this inhibits GABA interneurons, which disinhibits dopamine neurons and produces the accumbens surge. Everything that follows is the brain adapting to that surge.

STAGE	CIRCUIT	NEUROADAPTATION	WHAT THE PATIENT REPORTS
Intoxication	Ventral tegmental to accumbens	Dopamine surge, positive reinforcement	The rush, then warmth
Withdrawal	Extended amygdala, locus coeruleus	**Cyclic AMP pathway upregulated; dynorphin and CRF recruited**	Dysphoria, pain, autonomic storm
Preoccupation	Prefrontal cortex to striatum	Weak top down control, habit takes over	Craving on seeing a cue
Tolerance	Receptor level	**Desensitisation and internalisation of mu receptors**	The old dose does nothing

The two amber rows explain treatment: alpha two agonists work because the locus coeruleus is disinhibited, and lost tolerance is why relapse after abstinence kills.

- **Psychosocial factors.** Availability and low price, an using peer group, parental substance use with poor supervision, childhood adversity and neglect, school dropout and unemployment, and locally concentrated epidemics that make first exposure ordinary rather than deviant.
- **Comorbidity and self medication.** Conduct disorder, adult attention deficit, depression and post traumatic stress all raise risk, and iatrogenic exposure through prescribed analgesia is a route that clinicians control.
- **Where the two meet.** Most people exposed to opioids do not become dependent. Neurobiology explains what happens once use is repeated; the psychosocial field explains who repeats it.

WHAT EARNS THE MARKS

- **The disinhibition mechanism stated,** not just the phrase dopamine reward.
- **The withdrawal row tied to a drug,** so noradrenergic overactivity explains clonidine.
- **Psychosocial factors given equal weight,** which is what the six marks are for.
- **The integration line.** Answers that give only biology read as incomplete.

SEE ALSO Slot II - 16 opioid withdrawal Slot II - 15 opioid substitution therapy **SPRINT** Day 13, Substance use disorders

II - 12

Naltrexone. [10]

10 SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What it is	Pure antagonist, and how that differs from substitution
3	Indications and dose	Two licensed uses, the dose, the evidence
3	Before you start	The opioid free interval and the challenge test
2	Adverse effects	The liver, and the overdose risk after stopping

THE DEFINITION

A competitive opioid receptor antagonist with highest affinity at the mu receptor and no agonist activity. It is orally active, long acting, has no abuse liability and no street value, and it blocks the reinforcing effect of an opioid or a drink rather than replacing it.

USE	DOSE	WHAT IT ACHIEVES	THE CAUTION
Alcohol dependence	50 mg daily, orally	Fewer heavy drinking days, less craving	Works best with adherence support
Opioid relapse prevention	50 mg daily after detoxification	Blocks euphoria if a lapse occurs	**Requires full detoxification first**
Impulse control problems	50 mg daily, off label	Some benefit in gambling and self injury	Evidence thinner than for alcohol
Long acting form	Monthly injection	Removes the daily adherence decision	Limited availability in India

The amber cell is the one that turns a good answer into a safe one. Given to a dependent user it precipitates immediate withdrawal.

- **Before you start.** Opioid free for seven to ten days after a short acting opioid, and ten to fourteen after a long acting one. Confirm with a negative naloxone challenge and a urine screen. Take baseline liver function tests.
- **Adverse effects.** Nausea, headache, dizziness, abdominal pain, sleep disturbance and dysphoria; transaminase rise at high doses, so avoid in acute hepatitis or liver failure. It also blocks opioid analgesia, which matters before surgery.
- **The risk on stopping.** Tolerance is lost while the blockade is on. A patient who resumes his old dose after discontinuing is at high risk of fatal respiratory depression, and he must be told so.

WHAT EARNS THE MARKS

- **Antagonist contrasted with substitution** in the first two lines, which frames everything else.
- **The opioid free interval with its numbers,** plus the challenge test named.
- **The loss of tolerance warning.** Most answers stop at hepatotoxicity and miss the fatal risk.

SEE ALSO Slot II - 16 naloxone challenge test Slot II - 15 opioid substitution therapy **SPRINT** Day 13, Substance use disorders

II - 13

What is motivational enhancement therapy? What are the indications of motivational enhancement therapy in psychiatry? Discuss the principles of motivational enhancement therapy. [3+3+4]

3 + 3 + 4 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 What it is	Manualised motivational interviewing plus structured feedback
3 Indications	Where in psychiatry it is used, and the trial behind it
4 The principles	Four, each with the therapist behaviour it demands

THE DEFINITION

A brief, structured psychotherapy, usually four sessions, that applies motivational interviewing and adds personalised feedback from a formal assessment. It is client centred in style and directive in aim: the therapist does not install motivation, he draws out the patient's own reasons to change.

PRINCIPLE	WHAT IT MEANS	THERAPIST BEHAVIOUR	THE FAILURE MODE
Express empathy	Accurate understanding without approval or judgement	Reflective listening	Sympathy that colludes
Develop discrepancy	Widen the gap between present use and stated values	Elicit change talk	**Supplying the discrepancy yourself**
Roll with resistance	Resistance is a signal to change approach, not to push	Reframe, shift focus	**Arguing for change**
Support self efficacy	Belief that change is possible is itself predictive	Affirm past successes	Reassurance without evidence

The amber cells are the two errors that turn this into ordinary advice giving, and naming them is worth more than listing the principles alone.

- **Indications.** Alcohol and other substance use disorders, engagement of the ambivalent or poorly motivated patient, medication adherence in bipolar disorder and schizophrenia, tobacco cessation, gambling, eating disorders, and adherence to long term physical treatment.
- **The evidence.** In the large matching trial four sessions of this approach performed about as well as twelve sessions of cognitive behavioural or twelve step facilitation therapy, which is why it is the pragmatic first offer in a busy service.

WHAT EARNS THE MARKS

- **The distinction from motivational interviewing:** the added structured feedback and the fixed brief format.
- **All four principles named,** each with the therapist behaviour beside it in the grid.
- **Indications reaching beyond addiction,** since the question asks about psychiatry as a whole.
- **The matching trial cited,** which is what turns an assertion of efficacy into an answer.

SEE ALSO Slot II - 89 relapse prevention without drugs Slot II - 18 tobacco dependence

II - 14

How can you plan for tobacco control in your locality? What type of assistance would you want from the local Government? Compare nicotine chewing gum and nicotine patches. [5+3+2]

5 + 3 + 2 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

5 The locality plan	Map, protect, enforce, treat, educate
3 Government assistance	What only the administration can supply
2 Gum against patch	The grid, and why the answer is usually both

THE FRAMEWORK TO NAME FIRST

Build the local plan on the national tobacco control law of 2003 and the National Tobacco Control Programme: smoke free public places, no sale to minors or near schools, no advertising, pictorial warnings, and a cessation service in the district hospital.

DOMAIN	CHEWING GUM	TRANSDERMAL PATCH	WHAT SETTLES IT
Delivery	Intermittent, buccal, user controlled	Continuous, transdermal, fixed	Who controls the dose
Onset	Minutes, peaks within half an hour	Hours, then a steady level	**Only gum treats a craving as it happens**
Dosing	2 mg, or 4 mg if he smokes within half an hour of waking	Stepped strengths tapered over weeks	Time to first cigarette
Adherence	Frequent dosing, technique dependent	**Once daily, the best adherence of the group**	Number of decisions per day
Adverse effects	Jaw ache, hiccups, dyspepsia	Skin irritation, vivid dreams overnight	Dentition against skin

Chew and park, do not chew continuously: swallowed nicotine is not absorbed and causes the dyspepsia.

- **The locality plan.** Survey the burden and the vendors, declare schools and health facilities tobacco free, support enforcement of the public place ban, run a cessation clinic with trained counsellors, and take school and workplace education to where the young start.
- **What to ask the administration for.** Enforcement machinery and a designated officer, vendor licensing and action on sales near schools, programme funds for replacement therapy, health worker training, and the education department's cooperation for school work.
- **Combination is the answer.** A patch for the background plus gum for breakthrough craving beats either alone, so the comparison ends in a recommendation rather than a draw.

WHAT EARNS THE MARKS

- **The named law and programme,** before any local activity is described.
- **A plan divided into what you do and what you request,** as the question splits it.
- **The two amber cells:** gum for acute craving, patch for adherence.
- **The combination recommendation,** which is the point of comparing them.

SEE ALSO Slot II - 18 tobacco dependence Slot II - 86 reducing alcohol use in your area **SPRINT** Day 13, Substance use disorders

II - 15

Describe opioid substitution therapy. Mention the psychological and pharmacological principles underlying it. Discuss the NDPS Act in relation to it. [3+3+4]

3 + 3 + 4 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	What it is	The agents, the setting, and the stated goal
3	The principles	Cross tolerance below, harm reduction above
4	The law	What the Act enables, and what it leaves out

STEP	WHAT HAPPENS	WHO ACTS	CONDITION OR INTERVAL
1	Confirm opioid dependence and score withdrawal	Treating psychiatrist	Objective signs required
2	Induction on buprenorphine, or on methadone	Licensed centre staff	Only in mild withdrawal
3	Titrate to the dose that abolishes craving	Prescriber, daily review	Usually the first two weeks
4	Maintenance with directly observed dosing	Dispensing centre	Take home doses on stability
5	Psychosocial work, then a planned taper or continued maintenance	Team and patient together	Retention is the outcome

Induction into a patient who is not yet in withdrawal precipitates it. Order, actor and interval are exactly what this archetype is marked on.

- **Pharmacological principle.** A long acting agonist occupies the receptor, so the plasma level stays flat, withdrawal is abolished, cross tolerance blunts the euphoria of any illicit dose, and the peak and trough cycle that drives injecting disappears. Buprenorphine is a partial agonist with a ceiling on respiratory depression.
- **Psychological principle.** Harm reduction. Removing the daily hunt for money and drug frees attention for work, family and treatment, and retention in care is the measure by which the approach is judged.

WHAT THE ACT DOES NOT COVER

The Act licenses and supplies; it does not fund a service, train a team or create demand reduction on its own. It does not touch alcohol or tobacco. Consumption of a narcotic drug remains an offence, and the immunity for an addict who volunteers for treatment is conditional, not a general decriminalisation.

WHAT EARNS THE MARKS

- **Steps in order with the actor named at each,** and the mild withdrawal condition on induction.
- **Both principles answered separately,** pharmacological and psychological, because the question splits them.
- **The 2014 amendment named:** essential narcotic drugs brought under central rules, which is what made this therapy practicable.
- **The exclusions strip.** Listing provisions without their boundary reads as recall from a summary.

SEE ALSO Slot II - 16 opioid withdrawal Slot II - 12 naltrexone

II - 16

What is naloxone challenge test? Discuss management of opioid withdrawal with specific mention of non-opioid pharmacological strategies. [3+7]

3 + 7 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The challenge test	Purpose, dose, observation period, how it is read
4	Assess the withdrawal	The rating scale, the clock, the severity call
3	Non-opioid strategies	Alpha two agonist first, then symptom by symptom

THE NALOXONE CHALLENGE TEST

A safety test done before starting naltrexone, to prove that no physiological opioid dependence remains. Naloxone 0.8 mg is given, part intravenously and the remainder after observation, and the patient is watched for twenty to forty five minutes. Any withdrawal sign is a positive test: defer, and repeat later.

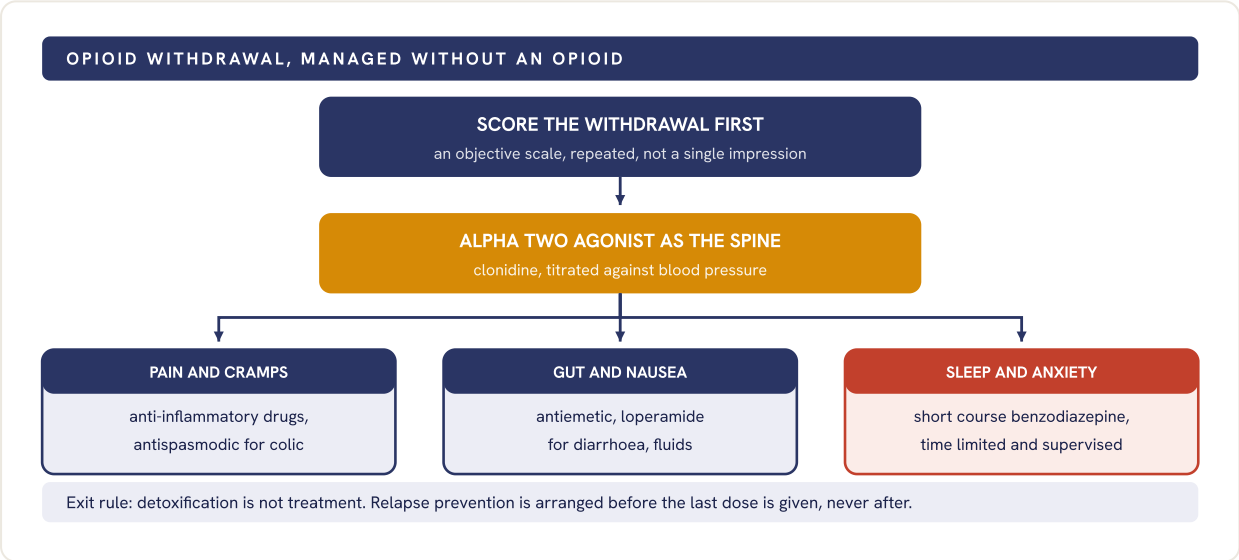


Fig 016.1 The alpha two agonist carries the syndrome and the three lanes mop up what it leaves, with the red lane marked because that is where iatrogenic dependence begins.

- **Why clonidine works.** Chronic opioid use disinhibits the locus coeruleus, and withdrawal releases a noradrenergic storm. An alpha two agonist suppresses it directly, which is why it treats sweating, cramps and restlessness but does very little for craving.

WHAT EARNS THE MARKS

- **The challenge test written with its numbers,** dose and observation period, and a stated reading rule.
- **An objective withdrawal score placed before any drug,** as the first box of the figure.
- **Clonidine explained by the locus coeruleus,** not merely listed.
- **The exit rule,** that detoxification without relapse prevention is an incomplete plan.

SEE ALSO Slot II - 12 naltrexone Slot II - 11 neurobiology of opioid use disorder **SPRINT** Day 13, Substance use disorders

II - 17

Define new psychoactive substances. How do you classify them? Write the clinical features related to use of any one of them. [2+4+4]

2 + 4 + 4 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The definition	What new means here, and what not controlled means
4 The classification	Families by mechanism, with an example in each
4 One family in detail	Synthetic cannabinoids, features and management

THE DEFINITION

Substances of abuse, pure or in preparation, not controlled by the 1961 or 1971 drug conventions but posing a public health threat. New means newly available, not newly synthesised.

FAMILY	EXAMPLE	MECHANISM	SIGNATURE EFFECT
Synthetic cannabinoids	The JWH and AM series	**Full agonist at CB1, unlike the partial agonism of cannabis**	Agitation, psychosis, seizures
Synthetic cathinones	Mephedrone	Monoamine releaser and re-uptake blocker	Stimulant, then paranoia
Phenethylamines	The 2C and NBOMe series	Serotonin 2A agonism	Hallucinations, hyperthermia
Synthetic opioids	Fentanyl analogues	Potent mu agonism	**Respiratory arrest at microgram doses**
Plant based and sedative	Kratom, etizolam	Mixed opioid, or benzodi-azepine like	Dependence, withdrawal fits

Ketamine type dissociatives form a sixth family. Classify by mechanism: street names change with every batch, receptors do not.

- **Synthetic cannabinoids in detail.** Full CB1 agonism without the cannabidiol that buffers herbal cannabis. Severe agitation, acute psychosis, catatonia, seizures, vomiting, tachycardia and hypertension, with reported acute kidney injury and myocardial ischaemia. Far more toxic than the plant it imitates.
- **The laboratory trap.** A routine urine cannabinoid immunoassay does not detect them. A negative screen in an agitated young man who says he smoked something does not exclude the diagnosis.
- **Management.** Supportive care in a quiet monitored area, benzodiazepines first for agitation and seizure risk, fluids, and watch renal function and creatine kinase.

WHAT EARNS THE MARKS

- **Both conventions named in the definition,** and new correctly glossed as newly available.
- **A classification by mechanism** with one example in each family, rather than a list of street names.
- **Full against partial CB1 agonism,** the amber cell that explains the whole clinical picture, and the negative urine screen that follows from it.

SEE ALSO Slot II - 10 caffeine related disorders Slot II - 90 dependence, abuse and harmful use SPRINT Day 13, Substance use disorders

II - 18

Discuss the clinical features, pharmacological and non-pharmacological management of tobacco dependence. [3+4+3]

3 + 4 + 3 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Clinical features	Dependence criteria, severity marker, withdrawal course
4 Pharmacological	Three first line agents, each with its rule
3 Non-pharmacological	The five step framework and what follows a lapse



Fig 018.1 The five step framework drawn as one sorting question, with the amber severity marker placed where it actually changes the prescription.

- **Clinical features.** The dependence criteria applied to nicotine, with withdrawal starting within a day of stopping: irritability, anxiety, poor concentration, restlessness, low mood, insomnia and increased appetite. Most of it settles in two to four weeks; craving outlasts it.
- **Pharmacological.** Replacement therapy, best as a patch plus a rescue form; varenicline, a partial agonist at the nicotinic receptor, titrated over the first week and continued twelve weeks; bupropion, avoided where there is any seizure risk or an eating disorder.
- **Smokeless tobacco.** In Indian practice much of the burden is chewed, not smoked. Ask about it by name, examine the mouth for submucous fibrosis, and remember that replacement therapy still applies.

WHAT EARNS THE MARKS

- **Time to first cigarette used as the severity marker,** as the amber box does, not the number smoked alone.
- **Three first line agents named with a rule each,** rather than the word counselling on its own.
- **The unwilling patient given a plan,** which is the lane most answers leave empty.
- **Smokeless tobacco asked about,** the Indian half of the question.

SEE ALSO Slot II - 14 tobacco control and replacement therapy Slot II - 13 motivational enhancement therapy

SPRINT Day 13, Substance use disorders

AREA 03 OF 14 · P2-A08

Anxiety, OCD and related disorders

9	8.9%	II-19	II-27
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 HIGH 3 SINGLE 5

REVISION ORDER

Build one OCD answer properly and five of the nine slots open. Panic and agoraphobia next, because they share a structure. Generalised anxiety disorder and hoarding are independent and both are short.

WHAT IS IN THIS AREA

Obsessive-compulsive disorder in five of the nine slots: the definition with severe-case management, the aetiopathology, the non-pharmacological management with the course studies, treatment resistance, and the psychological-theory slot it shares with depression. Panic disorder, generalised anxiety disorder, agoraphobia and hoarding disorder take the rest.

II - 19

Discuss non-pharmacological management of OCD. Describe studies on the course of OCD. [6+4]

6 + 4 | ANXIETY, OCD AND RELATED DISORDERS

DNB, WBUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	The first line	Exposure with response prevention, and why it works
3	What is added	Cognitive work, the family, and the refractory lane
4	Course studies	What the long follow ups actually found

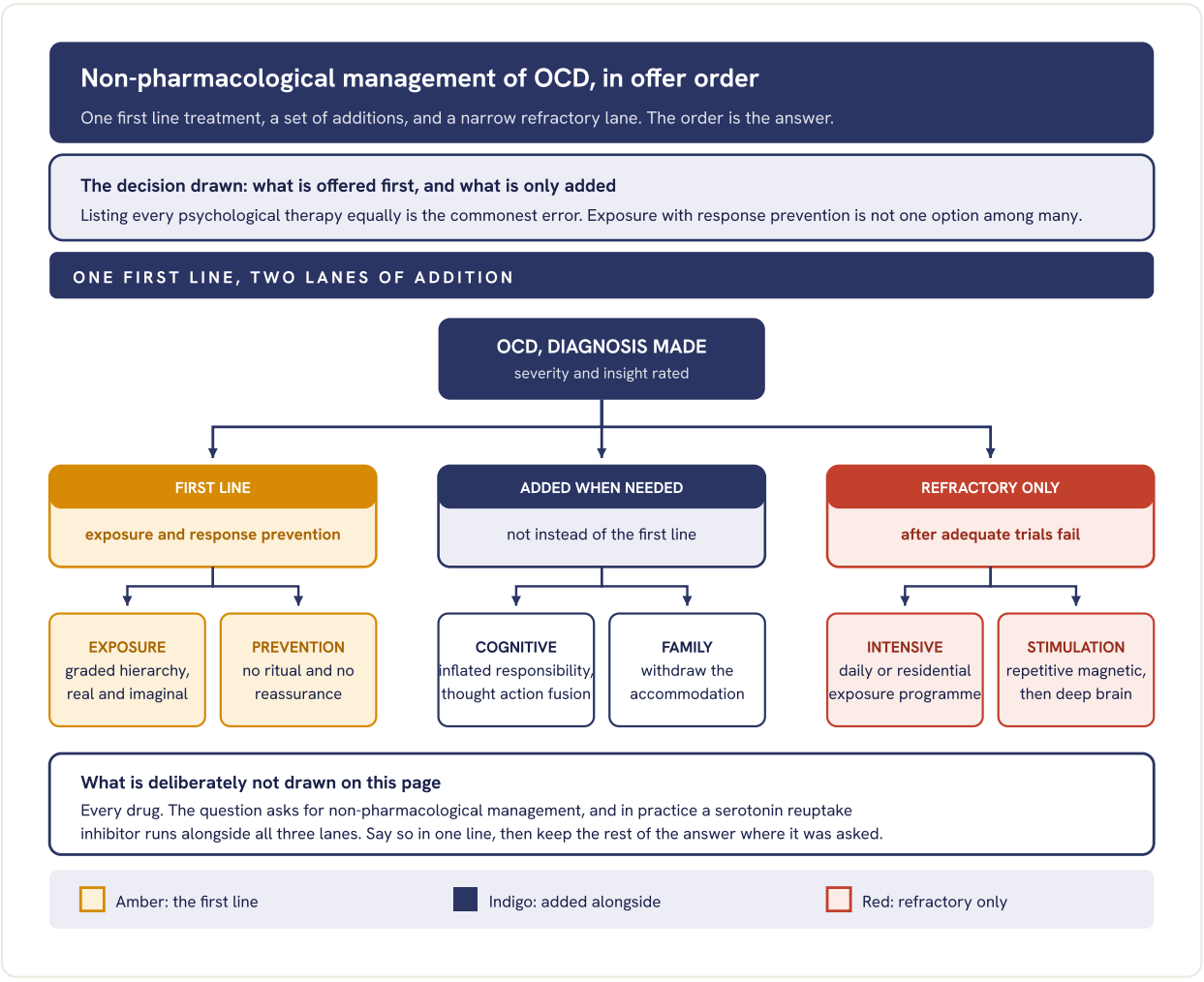


Fig 019.1 Exposure with response prevention drawn as the trunk rather than as one option among six, with the refractory lane kept visibly separate.

- **How exposure is delivered.** A shared hierarchy, prolonged exposure until anxiety falls without a ritual, homework between sessions, and no reassurance from staff or family. Response prevention is the active ingredient; exposure without it is distress with no learning.

COURSE STUDIES: WHAT THEY FOUND

Long term follow up finds a mainly chronic, fluctuating course. In the largest forty year study most patients improved and about a fifth recovered fully, while around half still met criteria at follow up. Later prospective cohorts confirm low full remission and frequent partial response. Early onset, poor insight, hoarding and long duration before treatment predict worse outcome.

WHAT EARNS THE MARKS

- **Response prevention named as the active ingredient,** the second amber leaf.
- **Family accommodation addressed,** which most answers never mention.
- **Course described with numbers,** chronic with partial remission rather than the word variable.
- **Predictors of poor outcome listed,** which is what makes it a study answer.

SEE ALSO Slot II - 22 severe OCD Slot II - 25 treatment resistant OCD **SPRINT**

Day 14, Anxiety, OCD and trauma-related disorders

II - 20

Management of panic disorder. [10]

10 ANXIETY, OCD AND RELATED DISORDERS

KNRUHS, MUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	**Confirm and exclude**	The medical and substance causes that mimic it
2	**The acute attack**	What is done in the room, and what is not
4	**Definitive treatment**	Cognitive behavioural therapy and the drug, in parallel
2	**Continuation**	How long, and how it is stopped

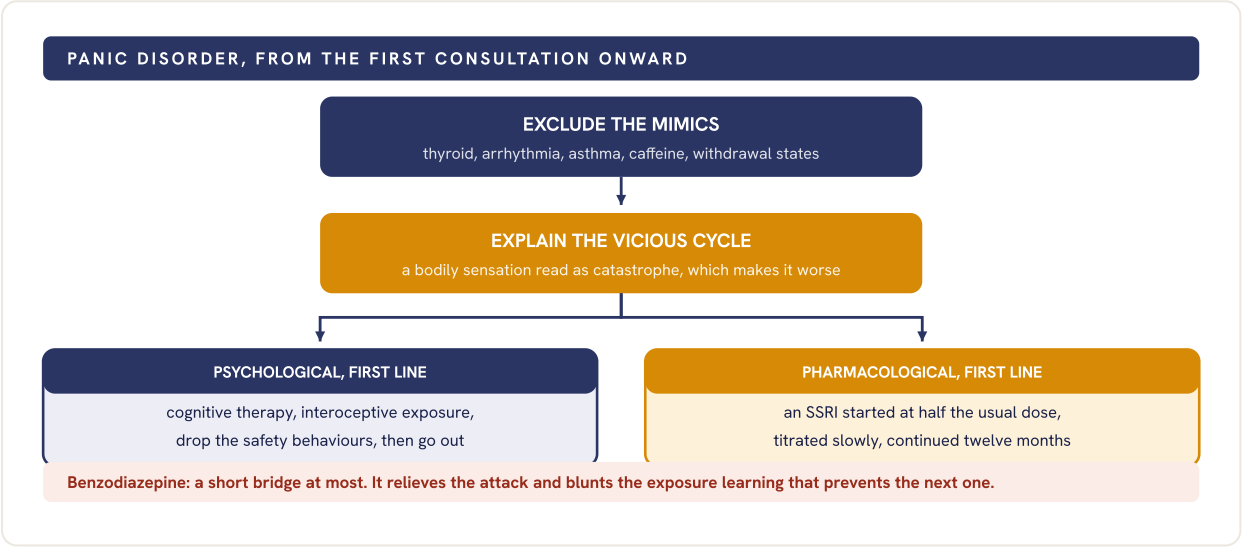


Fig 020.1 The two first line treatments drawn side by side rather than in sequence, with the benzodiazepine caution carried as a red strip along the foot.

- **In the acute attack.** Stay, speak calmly, name what is happening, and slow the breathing. Do not investigate again in a patient already worked up, because repeated tests confirm his belief that something has been missed.
- **The cognitive model.** Panic is the catastrophic misinterpretation of a benign bodily sensation. Treatment tests that interpretation directly, by inducing the sensation in the room and letting nothing be done about it.
- **Stopping.** Continue for at least a year after full response, then taper slowly. Early stopping and abrupt cessation are the two commonest reasons a treated patient relapses.

WHAT EARNS THE MARKS

- **Medical mimics excluded first,** as the top box of the figure does.
- **Both first line treatments named as first line,** not therapy offered only after drugs fail.
- **Interoceptive exposure and safety behaviours,** the specific techniques rather than the words cognitive therapy.
- **The benzodiazepine caution with its reason,** the red strip.

SEE ALSO Slot II - 26 agoraphobia Slot II - 23 generalised anxiety disorder SPRINT

Day 14, Anxiety, OCD and trauma-related disorders

II - 21

Discuss the etiopathology of obsessive-compulsive disorder. [10]

10 ANXIETY, OCD AND RELATED DISORDERS

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **The axis**	One disorder read at five levels, not five theories
4 **The biological levels**	Genes, transmitters, the circuit, the immune route
2 **The psychological level**	Appraisal and learning, from normal intrusions
2 **What ties them**	The evidence each level rests on, named

STATE THE AXIS BEFORE THE LIST

No single cause is established. The defensible answer reads one disorder at five levels, genetic, neurochemical, circuit, immunological and psychological, and names the observation each level rests on.

LEVEL	THE PROPOSAL	THE EVIDENCE IT RESTS ON	WHERE IT IS WEAK
Genetic	Moderate heritability, higher in early onset	Twin and family studies; glutamate transporter genes	No replicated single gene
Neurochemical	Serotonin dysfunction, with glutamate excess	**Selective response to serotonergic drugs alone**	Response is not proof of cause
Circuit	Overactive cortico-striato-thalamo-cortical loop	**Orbitofrontal and caudate overactivity that normalises with treatment**	Also seen in other conditions
Immunological	Post-streptococcal autoimmunity in a subgroup	Abrupt paediatric onset with tics after infection	A small and contested subgroup
Psychological	Normal intrusions appraised as personal responsibility	Intrusive thoughts are near universal in healthy people	Does not explain onset

The amber cells are the two findings an examiner expects by name, and both are pharmacological or imaging evidence rather than theory.

- **The psychological account, in detail.** Intrusions are universal. What makes them clinical is the appraisal: inflated responsibility for harm, and thought action fusion, the belief that thinking something makes it likelier or is morally equivalent to doing it. The compulsion relieves anxiety and is negatively reinforced, so it recurs.
- **The honest limit.** Every level above describes a correlate. None of them predicts who develops the disorder, and treatment response at one level does not establish causation at that level.

WHAT EARNS THE MARKS

- **The axis stated before the list,** which turns five theories into one structured account.
- **The loop named in full,** cortico-striato-thalamo-cortical, with the regions.
- **Universal intrusions as the starting point,** the finding that makes the appraisal model plausible.
- **The weakness column.** Naming what each level cannot explain reads as command.

SEE ALSO Slot II - 22 severe OCD Slot II - 24 psychological theories and management SPRINT

Day 14, Anxiety, OCD and trauma-related disorders

II - 22

Define obsessive-compulsive disorder. Discuss the management of a patient with severe OCD. [3+7]

3 + 7 | ANXIETY, OCD AND RELATED DISORDERS

DNB, KUHS, NTRUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

3 **Definition**	Obsession and compulsion defined, then the disorder
2 **Assess severity**	Rate it, check insight, risk and where he is treated
4 **Combined treatment**	High dose serotonergic drug plus intensive exposure
1 **If no response**	The augmentation step, and what precedes it

THE DEFINITION

Recurrent obsessions, which are intrusive unwanted thoughts, images or impulses recognised as one's own and resisted, or compulsions, which are repetitive acts performed to reduce the distress they cause. They are time consuming or distressing, and not pleasurable in themselves.

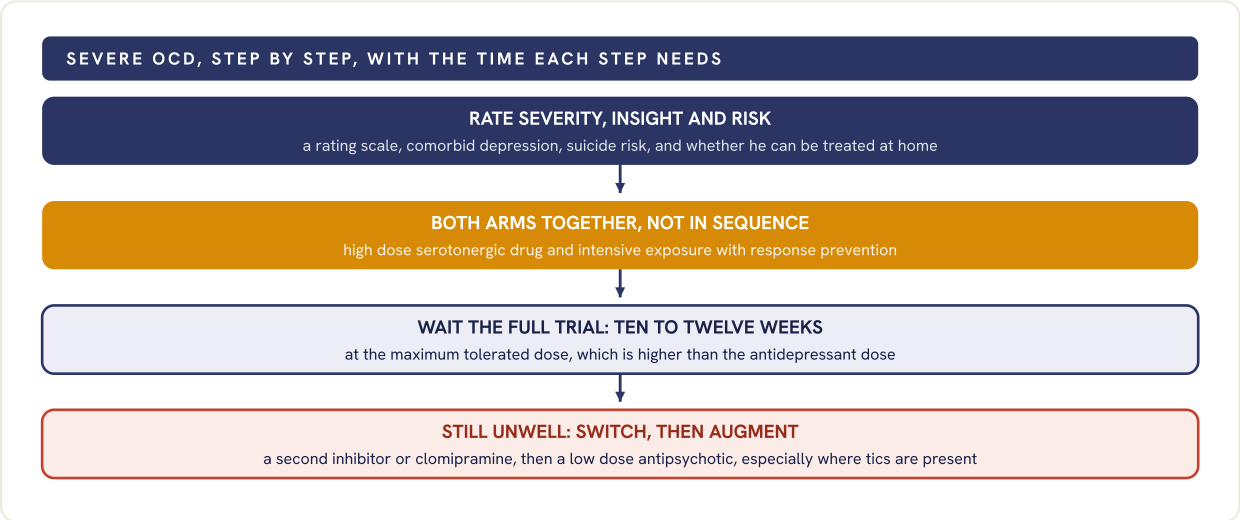


Fig 022.1 The ladder with the waiting time written into each step.

- **Dosing.** Serotonin reuptake inhibitors are used at the top of their range in this disorder, well above the usual antidepressant dose, and the response is slower. Clomipramine remains the reference agent, with an electrocardiogram before and during treatment.
- **Before calling it resistant.** Confirm the drug was taken at that dose for that long, that exposure was actually delivered with response prevention, and that the family have stopped providing reassurance. Most apparent resistance is one of these three.

WHAT EARNS THE MARKS

- **Obsession and compulsion defined separately** before the disorder is defined.
- **Drug and exposure started together,** the amber step, not therapy held back as a second line.
- **The ten to twelve week trial at high dose,** written as a number, and the three checks before resistance is declared.

SEE ALSO Slot II - 25 treatment resistant OCD Slot II - 19 non-pharmacological management of OCD **SPRINT** Day 14, Anxiety, OCD and trauma-related disorders

II - 23

Describe the clinical management of generalised anxiety disorder. [10]

10 ANXIETY, OCD AND RELATED DISORDERS KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Confirm the diagnosis**	Six months of uncontrollable worry, and what mimics it
2	**Step one and two**	Education, monitoring, guided self help
4	**Step three**	Therapy or drug, both first line, patient's choice
2	**Step four**	The refractory options, and what is avoided throughout

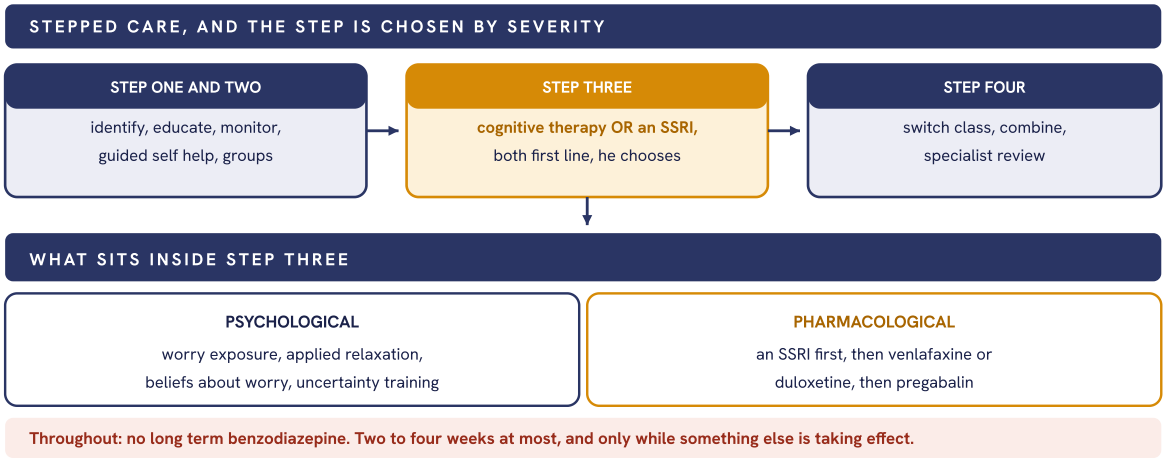


Fig 023.1 Four steps across the top, then the contents of the step that matters opened out below it.

- **Confirm first.** Six months of excessive, uncontrollable worry across several domains with restlessness, fatigue, poor concentration, irritability, muscle tension or disturbed sleep. Exclude thyroid disease, caffeine, withdrawal states and a primary depressive episode.
- **The models worth naming.** Worry as avoidance of worse imagery, positive and negative beliefs about worrying itself, and intolerance of uncertainty. Each generates a technique, which is why naming them is not decoration.
- **Duration.** Continue the drug for at least twelve months after response and taper slowly. This is a chronic relapsing condition, and stopping at three months is why so many return.

WHAT EARNS THE MARKS

- **Stepped care named as a structure,** so the answer is organised by severity rather than by treatment type.
- **Therapy and drug both marked first line,** the amber box.
- **The benzodiazepine limit stated in weeks,** the red strip.
- **Twelve months of continuation,** which most answers omit entirely.

SEE ALSO Slot II - 20 panic disorder Slot II - 26 agoraphobia SPRINT
Day 14, Anxiety, OCD and trauma-related disorders

II - 24

Discuss different psychological theories of depression and OCD. Describe how these theories can help in their management. [5+5]

5 + 5 | ANXIETY, OCD AND RELATED DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Depression theories	Cognitive, behavioural, interpersonal, psychodynamic
2 OCD theories	Learning and appraisal, from normal intrusions
3 Theory into technique	Each model names the intervention it generates
2 Choosing	By formulation, not by habit or availability

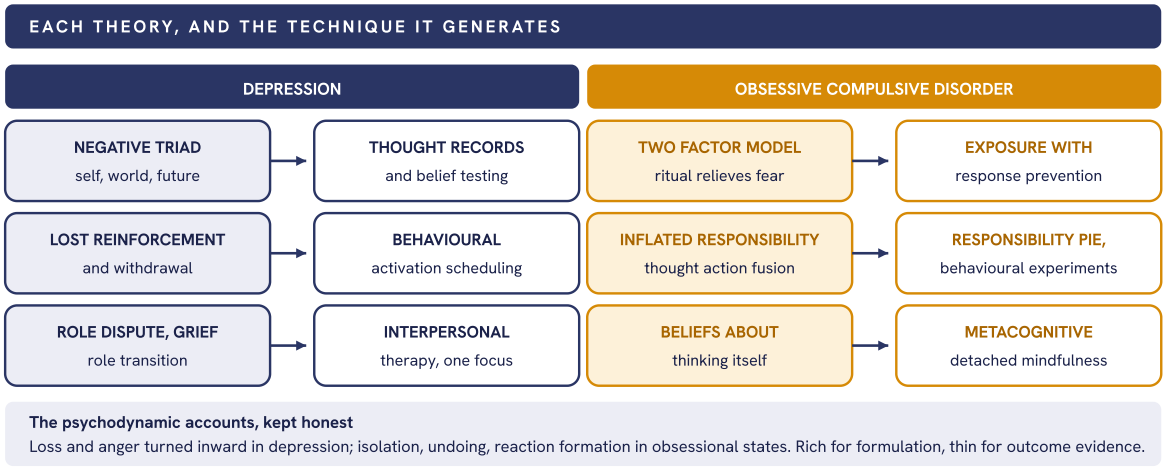


Fig 024.1 Each row reads left to right as one claim and the technique it licenses, which is exactly what the second half of the question asks for.

- **Choosing between them.** Take the model that fits the formulation. Withdrawal and inactivity call for activation, a dispute or a bereavement for interpersonal work, ritual maintained by relief for exposure, and a patient tormented by the meaning of his thoughts for the appraisal route.

WHAT EARNS THE MARKS

- **Every theory paired with its technique on the same line,** as each row of the figure does.
- **Both disorders answered,** because half the marks sit in the column most answers neglect.
- **Normal intrusions as the starting point of the OCD models,** which is what makes them treatments and not descriptions.
- **The psychodynamic strip kept honest,** useful for formulation, weak on outcome evidence.

SEE ALSO Slot II - 21 etiopathology of OCD Slot II - 19 non-pharmacological management of OCD

II - 25

What is treatment resistant OCD? What are the proposed neurobiology of such resistance? Discuss the treatment options for such a case. [2+3+5]

2 + 3 + 5 | ANXIETY, OCD AND RELATED DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The definition	What counts as adequate, and the staging above it
3 Proposed neurobiology	Circuit, glutamate, and the phenotypes that resist
5 Treatment options	Optimise, augment, then the neuromodulation lane

THE DEFINITION

Failure to achieve a meaningful fall in symptom score, conventionally under twenty five to thirty five per cent on a standard rating scale, after two adequate trials of a serotonin reuptake inhibitor at maximum tolerated dose for ten to twelve weeks each, together with an adequate trial of exposure with response prevention. Staged from non-response through refractory to intractable.

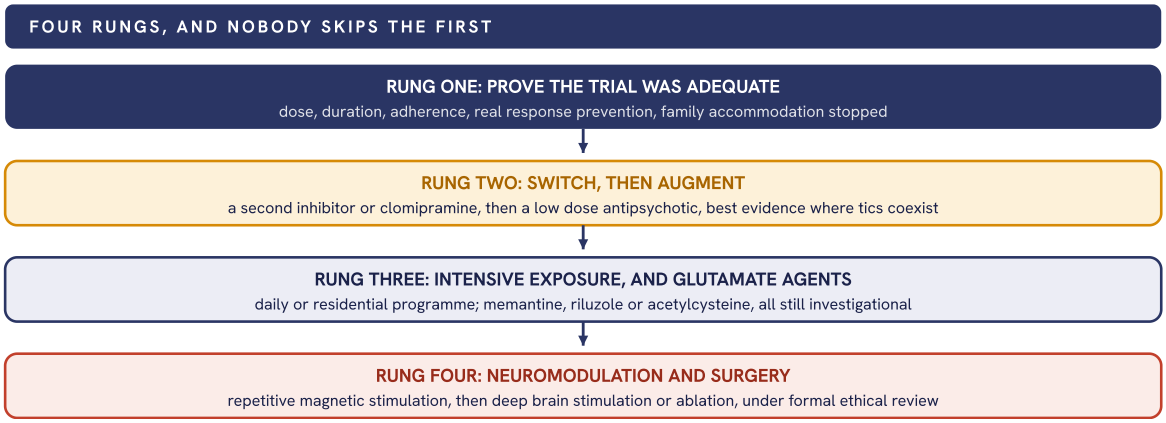


Fig 025.1 The ladder for resistant disease, with the audit of the first trial drawn as a rung rather than assumed, because most resistance is found there.

- **The proposed neurobiology.** Persistent overactivity in the orbitofrontal to caudate loop that does not normalise with treatment; raised glutamate in the caudate and cerebrospinal fluid with reduced inhibitory tone; and dopaminergic involvement, which is why antipsychotic augmentation helps where there are tics.
- **The phenotypes that resist.** Early onset, poor or absent insight, hoarding symptoms, prominent tics, and long duration before any treatment. Naming these is what turns a list of drugs into a clinical answer.

WHAT EARNS THE MARKS

- **The definition with its numbers,** two trials, ten to twelve weeks, a stated response threshold.
- **The first rung, auditing the trial,** before any new drug is proposed.
- **Glutamate named alongside serotonin,** which is the current part of the answer.
- **Surgery placed last and gated by ethical review,** not offered as one option among many.

SEE ALSO Slot II - 22 severe OCD Slot II - 21 etiopathology of OCD SPRINT
Day 37, Psychopharmacology II: resistance and neurostimulation

II - 26

What is agoraphobia? Discuss diagnostic features, differential diagnosis and management of agoraphobia. [2+3+2+3]

2 + 3 + 2 + 3 | ANXIETY, OCD AND RELATED DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	The fear is of escape being blocked, not of the place
3 Diagnostic features	Two of five situations, avoidance, six months
2 Differential diagnosis	Sorted by what he is actually afraid of
3 Management	Graded exposure, the drug, and the family rule

CONDITION	WHAT HE IS AFRAID OF	WHAT SETTLES IT
Agoraphobia	Being trapped or helpless if symptoms come	**Fear of the escape route, not of the place**
Social anxiety disorder	Being watched and judged	The fear goes when nobody can observe
Specific phobia	The situation itself	One circumscribed trigger, no panic elsewhere
Psychotic disorder	Being followed or harmed	**A delusional reason for staying in**

Ask what he expects to happen if he goes out. That one question does most of this table's work, and it also separates depression, where there is simply no drive to go.

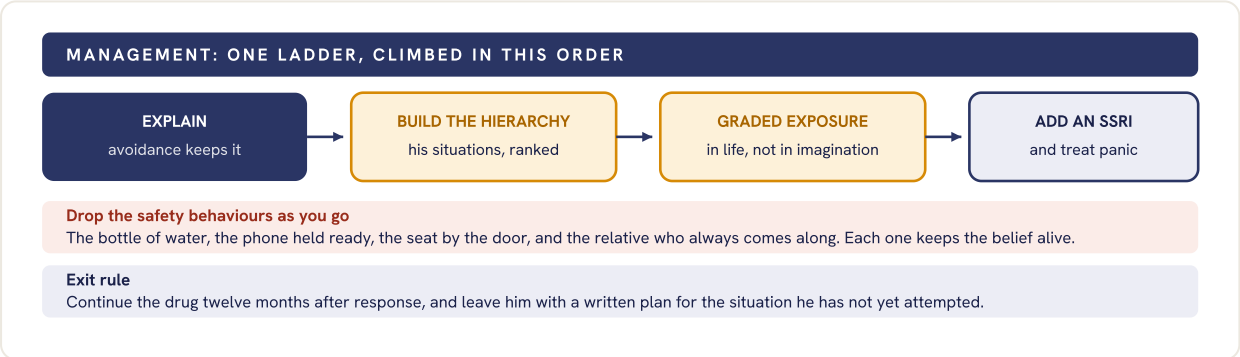


Fig 026.1 Exposure drawn as the spine of treatment with the drug alongside it, and the safety behaviours carried as a red strip because they are what quietly defeats the ladder.

- **Diagnostic features.** Marked fear in at least two of five settings: public transport, open spaces, enclosed places, queues or crowds, and being outside the home alone. Avoided, endured with dread, or requiring a companion; out of proportion; six months.

WHAT EARNS THE MARKS

- **The definition built on escape and help,** not on a list of feared places.
- **Two of five situations and the six month rule,** stated as criteria.
- **The differential sorted by the feared outcome,** which is the column that does the work.
- **The companion named as a safety behaviour,** the line most answers miss.

SEE ALSO Slot II - 20 panic disorder Slot II - 23 generalised anxiety disorder **SPRINT**

Day 14, Anxiety, OCD and trauma-related disorders

II - 27

Discuss the clinical features and differential diagnosis of hoarding disorder. [5+5]

5 + 5 | ANXIETY, OCD AND RELATED DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	Difficulty discarding, and the clutter that follows
3	Clinical features	Acquisition, insight, course, and the safety risk
3	The differential	Sorted by why the objects are kept
2	Comorbidity	What travels with it and changes the plan

THE DEFINITION

Persistent difficulty parting with possessions regardless of their actual value, driven by a perceived need to save them and by distress on discarding. The accumulation congests living areas so they cannot be used as intended, and it causes impairment or an unsafe home.

CONDITION	WHY THE OBJECTS ARE KEPT	WHAT SETTLES IT
Hoarding disorder	Attachment and a felt need to save	**Distress on discarding, comfort in keeping**
Obsessive compulsive disorder	An obsession, often contamination or harm	**Keeping is unwanted and brings no pleasure**
Depression	Nothing is kept deliberately; nothing is cleared	Accumulation is passive, follows low mood
Schizophrenia	A delusional reason for saving	The stated reason is bizarre
Frontal or degenerative disease	Loss of judgement and disinhibition	Late onset, with squalor and self neglect
Normal collecting	Interest and pride in the collection	Organised, displayed, no impairment

Two questions separate almost the whole table: why is it kept, and how does he feel while keeping it.

- **Clinical features.** Excessive acquisition in most cases, by buying or by collecting free things. Insight ranges from fair to absent, and the specifier is recorded. Onset is in adolescence, severity grows each decade, and the course is chronic without treatment.
- **The risk that must be assessed.** Fire load, blocked exits, falls, infestation, and the safety of children or dependent elders in the home. Public health or fire services may already be involved, and their involvement changes the treatment plan.

WHAT EARNS THE MARKS

- **Difficulty discarding named as the core,** not the clutter, which is only its consequence.
- **Acquisition and insight recorded as specifiers,** which shows the current classification is known.
- **The differential organised by motive,** the amber pair separating this from obsessive compulsive disorder.
- **The safety assessment,** which is what makes it a clinical answer rather than a description.

SEE ALSO Slot II - 21 etiopathology of OCD Slot II - 22 severe OCD SPRINT

Day 14, Anxiety, OCD and trauma-related disorders

AREA 04 OF 14 · P2-A01

Schizophrenia: aetiology, phenomenology and course

8	8.4%	II-28	II-35
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 · HIGH 1 · RECURRENT 1 · SINGLE 4

REVISION ORDER

Course and outcome first, and negative symptoms with it: those two carry the anchor weight here. Psychosocial management and the chronic-care slot next, they share their content. Early psychosis after that. Endophenotype and expressed emotion last, both short and both critical-evaluation shapes.

WHAT IS IN THIS AREA

Course and outcome, negative symptoms and their management, early psychosis and its prevention, psychosocial management, childhood psychosis, the chronic-care slot that asks what India actually provides and how family functioning is assessed, endophenotypes and biomarkers, and expressed emotion as a critical evaluation.

II - 28

Course and outcome of schizophrenia. [10]

10 SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB, KNRUHS, RGUHS, TN-MGRMU - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2 **Two questions**	Course is the shape over time, outcome the state at follow-up
2 **Course patterns**	Prodrome, episode structure, the ICD-11 course specifiers
3 **Outcome at follow-up**	The long cohorts, and what they actually measured
3 **Predictors and mortality**	Prognostic factors, the mortality gap, what is modifiable

SEPARATE THE TWO WORDS FIRST

Course is the longitudinal shape: prodrome, episodes, remissions, residual state. Outcome is the position at a stated follow-up point, on three separable axes, symptomatic, functional and personal. ICD-11 asks for both, an episode pattern plus a remission qualifier.

DOMAIN	POINTS TO BETTER OUTCOME	POINTS TO WORSE OUTCOME
Onset	Acute, later, clear precipitant	Insidious, before twenty, no precipitant
Premorbid	Good work and social record, married	Schizoid traits, poor school record
Symptoms	Positive and affective symptoms lead	Negative symptoms, early cognitive loss
Modifiable	Short untreated psychosis, adherence kept	Long untreated psychosis, substance use

Only the last row can be moved by a clinician. High expressed emotion and absent carer support belong with the right-hand column.

- **Course.** A premorbid phase, a prodrome of one to five years with attenuated symptoms and functional slippage, then the first episode. Deterioration is steepest in the first five years and then largely plateaus.
- **Outcome.** Roughly a third do well, a third relapse with residual disability, a third deteriorate. The WHO cohorts, IPSS, DOSMeD and ISoS, reported better outcome in Indian and other low-income centres, and Indian long-term follow-up supports it.
- **Qualify it.** That advantage is contested: differential attrition, milder acute-onset cases entering the cohorts, and outcome scored as social role rather than symptom count.
- **Mortality.** Life expectancy is fifteen to twenty years shorter, mostly cardiovascular, not suicide. Lifetime suicide is about five per cent, concentrated early and after discharge.

WHAT EARNS THE MARKS

- **Course and outcome separated in the first two lines.** Most answers merge them and then repeat themselves.
- **The prognostic grid, both columns,** with the modifiable row identified as modifiable.
- **The named cohorts with their caveat,** and the mortality gap named as cardiovascular.

SEE ALSO Slot II - 29 negative symptoms Slot II - 35 expressed emotion SPRINT

Day 8, Schizophrenia: aetiology, phenomenology, classification and course

II - 29

Describe negative symptoms in schizophrenia and discuss about the management. [5+5]

5 + 5 | SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB, KUHS, NTRUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2	The two factors	Diminished expression and avolition, five symptoms between them
3	Primary or secondary	Deficit syndrome, and the four causes that mimic it
3	Drug treatment	Lower the dose first, then switch, and monitor
2	Psychosocial	Skills, remediation and employment carry the function

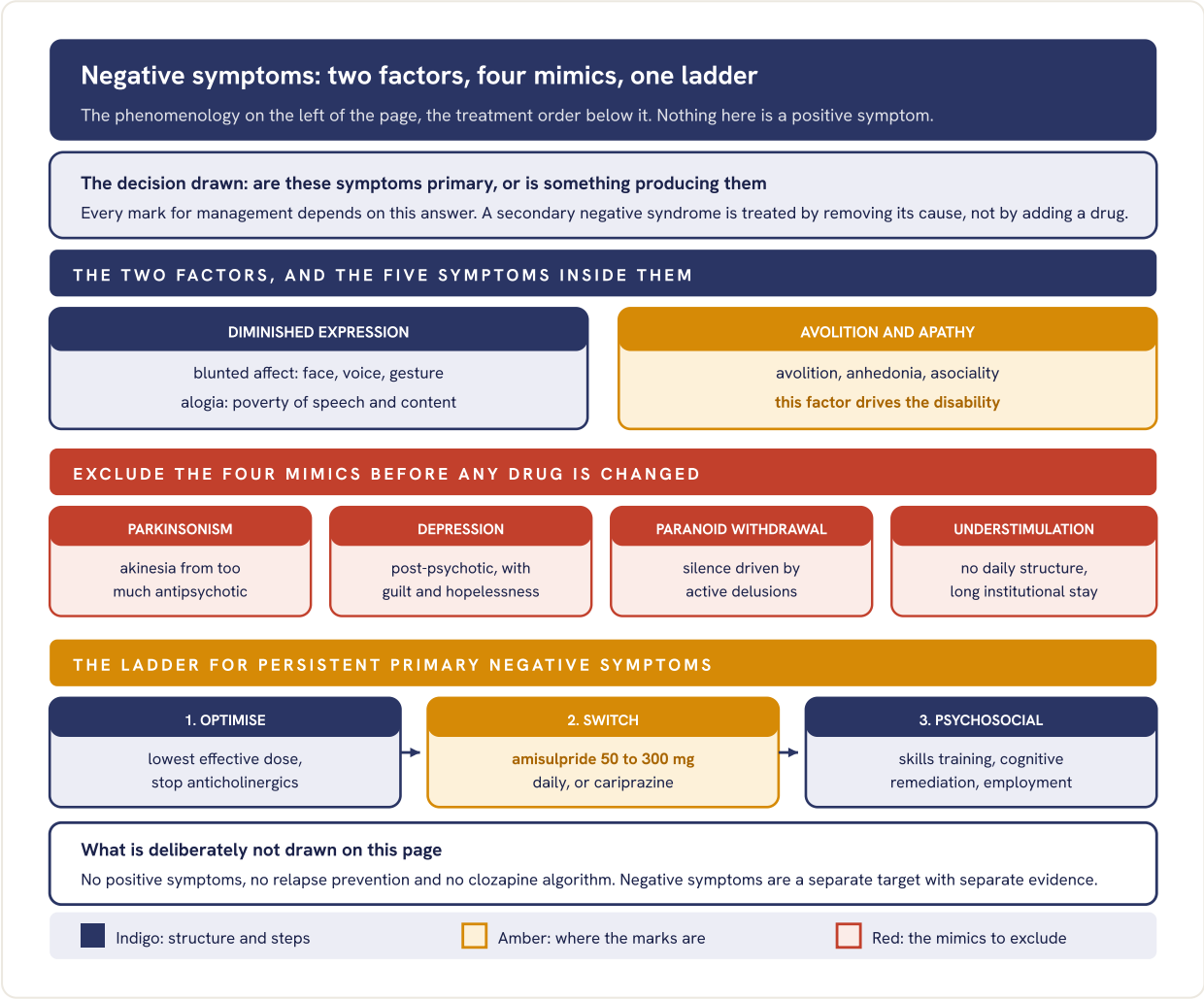


Fig 029.1 The two-factor structure of negative symptoms, the four secondary causes that must be excluded before the syndrome is called primary, and the three-step treatment ladder that follows.

- **The five symptoms.** Blunted affect and alogia make up diminished expression. Avolition, anhedonia and asociality make up the motivational factor. Attention impairment is described alongside them but loads separately.
- **Deficit syndrome.** Negative symptoms that are primary, enduring for at least a year, and present during clinical stability. It carries a poorer functional trajectory and is the population the drug trials target.

PITFALL

Clozapine is not the answer to this question. Its evidence is for positive symptom resistance; for primary negative symptoms it performs no better than other second generation agents. Low-dose amisulpride and cariprazine carry the dedicated trials.

WHAT EARNS THE MARKS

- **The two-factor structure named, with all five symptoms placed.** A flat list of six A words is a weaker answer.
- **The four mimics excluded before treatment is discussed.** This is the sequence the second half of the question is testing.
- **Dose reduction written as step one.** Adding a drug to treat drug-induced akinesia is the commonest error here.
- **Psychosocial treatment given its own line.** Function does not recover on pharmacology alone.

SEE ALSO Slot II - 28 course and outcome Slot II - 48 cognitive remediation **SPRINT**

Day 8, Schizophrenia: aetiology, phenomenology, classification and course

II - 30

What do you understand by the concept of early psychosis? Discuss the prevention and treatment of early psychosis. [4+3+3]

4 + 3 + 3 | SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

4 The concept	Clinical staging, the at-risk mental state, the critical period
3 Prevention	Indicated prevention only, and what is not recommended
3 Treatment	Shorten the untreated period, low dose, family and work

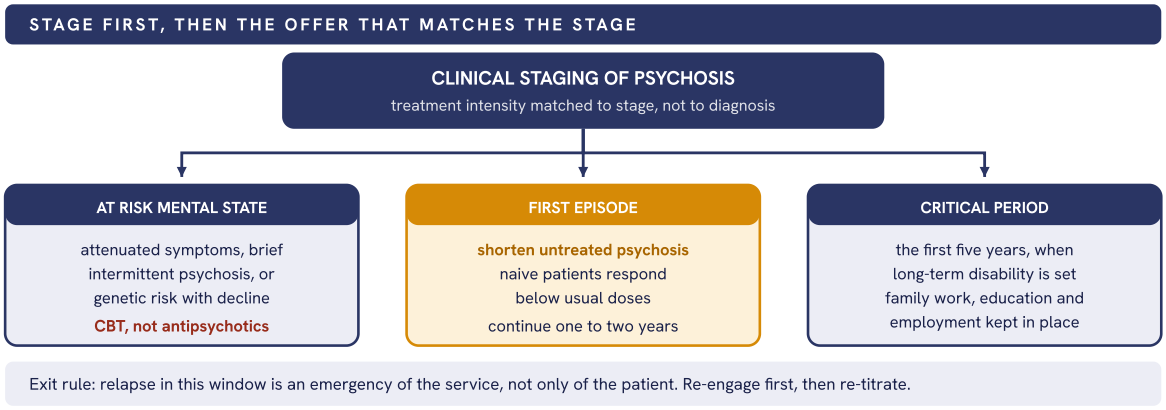


Fig 030.1 Early psychosis as three stages, each with the offer that belongs to it, and the first five years marked as the window where effort pays.

COLOUR CODE stage and structure | where treatment changes outcome | what is not recommended

- **The concept.** Psychosis is staged rather than only diagnosed: asymptomatic risk, undifferentiated distress, at-risk mental state, first episode, relapse, persistent illness.
- **Prevention.** Only indicated prevention is workable. Cognitive behavioural therapy reduces transition modestly. Universal screening is not justified: most at-risk cases never convert.
- **The Indian problem.** Untreated psychosis commonly runs beyond a year, through faith healing pathways and thin services. The district programme is the realistic vehicle.

PITFALL

Prescribing an antipsychotic for an at-risk mental state. Benefit is unproven, most of those treated would never have converted, and the metabolic and motor harms are certain.

WHAT EARNS THE MARKS

- **Staging stated as the organising idea,** not just the phrase "early intervention".
- **The three at-risk groups named.** They are the operational definition being asked for.
- **Untreated psychosis identified as the modifiable variable,** and the critical period given as five years.

SEE ALSO Slot II - 45 first episode pharmacology Slot II - 32 childhood psychosis

II - 31

Discuss psychosocial management in a case of schizophrenia. [10]

10 SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Principles	Alongside medication, phase matched, family as the unit
3 Family work	Psychoeducation, expressed emotion, a defined session count
3 Individual work	Adherence, cognitive therapy, skills, cognitive remediation
2 Rehabilitation	Supported employment, day care, disability entitlements

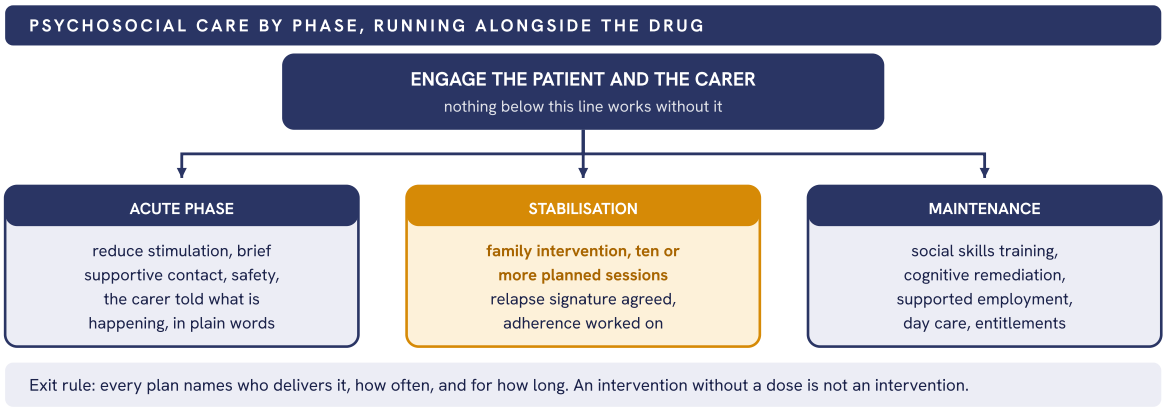


Fig 031.1 Psychosocial management by phase of illness, with the interventions that carry trial evidence placed where they are actually delivered.

- **Family intervention.** The strongest psychosocial effect on relapse: psychoeducation, communication and problem-solving training, and work on criticism and overinvolvement, over at least ten planned sessions.
- **Individual work.** Cognitive behavioural therapy for persisting positive symptoms, social skills training, cognitive remediation for attention and working memory, and adherence work in a motivational rather than a warning stance.
- **Work and entitlements.** Supported employment, placed first and trained on the job, outperforms prevocational training. Disability certification opens pension, concession and reservation.

PITFALL

Writing "counselling and rehabilitation" and stopping. An answer that cannot say how many sessions of what, delivered by whom, has described a sentiment rather than a plan.

WHAT EARNS THE MARKS

- **Phase-matched structure.** Acute, stabilisation and maintenance ask for different things.
- **Family intervention named with its session count.** It is the highest-yield line on the page.
- **Supported employment preferred to prevocational training,** with entitlements and carer burden included.

SEE ALSO Slot II - 33 chronic care in India Slot II - 35 expressed emotion

II - 32

Discuss the concept of childhood psychosis and its management. [10]

10 SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The concept	How it separated from autism, and the terms used now
3	What must be excluded	Organic causes, and the phenomena that only look psychotic
3	Drug treatment	Agent, low start, and the monitoring children need more of
1	Around the child	School, family and development kept moving

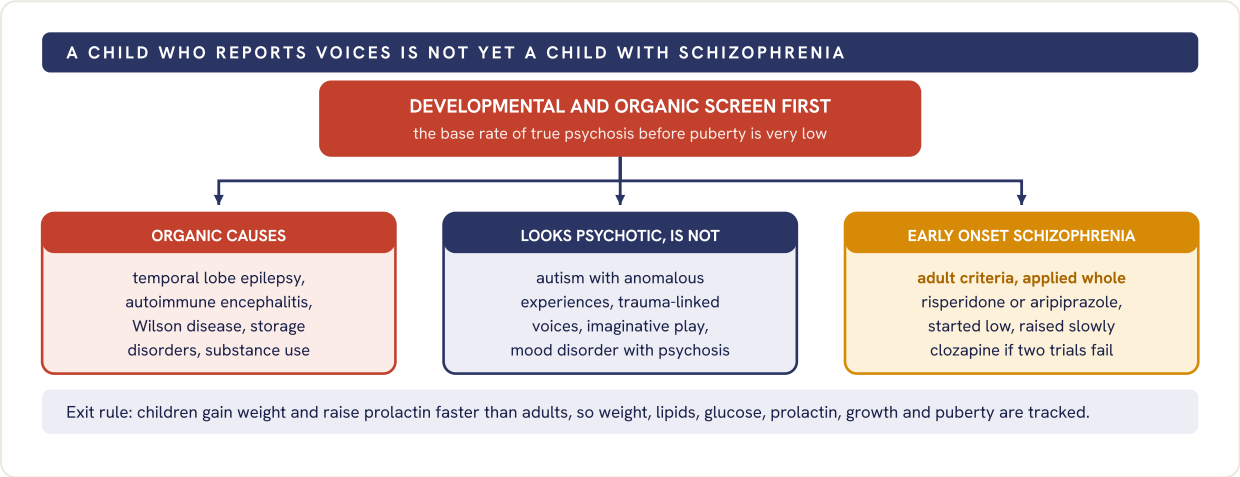


Fig 032.1 The order of work in a child with apparent psychosis: exclude the organic causes, separate the mimics, then treat as early onset schizophrenia.

- **The concept.** Until the 1970s every severe childhood disturbance was called childhood schizophrenia. Kolvin and Rutter separated it by age of onset, phenomenology, family history and course. The split has held.
- **The terms now.** Childhood onset means before thirteen and is rare, of the order of one in ten thousand. Early onset means before eighteen. Criteria are the adult criteria, unchanged.
- **What is different.** More premorbid language, motor and social delay, more insidious onset, more negative symptoms, a higher yield from genetic testing including the 22q11.2 deletion, poorer outcome.

PITFALL

Treating a reported voice as sufficient. Non-psychotic hallucinatory experiences are common in anxious, grieving and maltreated children and mostly remit.

WHAT EARNS THE MARKS

- **The historical separation from autism, with its basis.** The word "concept" in the stem asks for exactly this.
- **The organic screen written before any drug.** In this age group it separates a diagnosis from a mistake.
- **Monitoring described as heavier than in adults,** with the parameters named.

SEE ALSO Slot II - 30 early psychosis Slot II - 45 first episode pharmacology

II - 33

What are the options available in India for chronic care of longstanding schizophrenic patients? Discuss different government facilities a chronic schizophrenia patient can avail. How to assess family functioning in a case of schizophrenia? [2+4+4]

2 + 4 + 4 | SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What chronic care asks	Where the person lives, who supervises, what fills the day
4	Government routes	The programme, the Act, the certificate, the entitlements
2	Other options	Day care, halfway homes, long stay, carer-led groups
2	Family functioning	Four domains, and the instrument for each of them

WHAT CHRONIC CARE ACTUALLY DECIDES

Three questions, and every option below answers one of them: where the person lives, who supervises treatment, and what occupies the day. Cure is not on the list.

ROUTE	WHAT IT PROVIDES	HOW IT IS REACHED
District programme	Free outpatient care, medicines, outreach to the taluk	District hospital or outreach camp
Mental Healthcare Act 2017	Community living, less restrictive care, state duty to provide halfway homes	State authority, review board on appeal
Disability certificate	Benchmark disability at forty per cent on the notified scale	Medical board at the district hospital
What follows it	Pension, travel concession, employment reservation, carer tax relief	Certificate plus the disability identity card

Insurance for mental illness on the same terms as physical illness follows from section 21(4). Non-government day care, halfway homes and family associations sit alongside all of this.

- **Family functioning, four domains.** Burden, on the Family Burden Interview Schedule of Pai and Kapur, which separates objective from subjective. Expressed emotion, on the Camberwell Family Interview or the five minute speech sample. Knowledge and attitudes, on the Family Interview Schedule. General functioning, on the Family Assessment Device.
- **What it is for.** It answers whether this family can supervise medication and absorb a relapse. That decides home-based against residential care, and it is reassessed.
- **The line most answers miss.** The commonest crisis is the ageing or death of the parent carer. A named second carer, a nominated representative and a guardianship route belong in the plan.

WHAT EARNS THE MARKS

- **The three questions chronic care answers,** stated before any list of facilities.
- **Each government route with its access path.** Naming a scheme without saying how a patient reaches it is half a mark.
- **Named instruments for family functioning, including an Indian one,** with the domain each covers.

SEE ALSO Slot II - 31 psychosocial management Slot II - 35 expressed emotion

II - 34

What is endophenotype? Discuss schizophrenia endophenotype biomarkers. [3+7]

3 + 7 | SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 **Definition and criteria**	The five requirements, and that all five are needed
4 **The candidates**	Electrophysiology, eye movement, cognition, imaging
2 **Why they were wanted**	A simpler genetic target than the whole syndrome
1 **Where it stands**	One honest sentence on what the programme delivered

THE DEFINITION, WITH ITS FIVE CRITERIA

A measurable, heritable trait on the causal path between genotype and clinical syndrome. Gottesman and Gould require five things: association with the illness in the population, heritability, state independence, co-segregation within families, and presence in unaffected first degree relatives above the population rate.

CANDIDATE	WHAT IS MEASURED	STANDING
P50 gating	Suppression of the response to the second of two clicks	Deficient; linked to the alpha-7 nicotinic locus
Prepulse inhibition	Startle reduced by a weak preceding stimulus	Reduced in patients and relatives
Mismatch negativity	Automatic detection of an auditory change	Amplitude reduced; the most reproducible
Eye movement	Smooth pursuit tracking and antisaccade errors	Impaired in patients and many relatives
Neurocognition	Working memory, sustained attention, verbal memory	Impaired, heritable, state independent

Structural imaging findings, neurological soft signs, dermatoglyphic asymmetry and the blunted niacin skin flush are described alongside but meet fewer of the five criteria.

- **Why they were wanted.** The syndrome is heterogeneous and polygenic. A trait nearer gene action was expected to be genetically simpler, mappable in smaller samples.
- **What happened.** The candidates proved as polygenic as the syndrome, and none has shown larger genetic effect sizes. The promised shortcut did not appear.
- **Where it stands.** They survive as vulnerability markers, as a way of stratifying trial samples, and as objective endpoints. The ambition was absorbed into dimensional frameworks cutting across diagnoses.

WHAT EARNS THE MARKS

- **All five criteria, listed as criteria.** Three of five is the commonest way the first three marks are lost.
- **Candidates grouped by modality,** not offered as an undifferentiated list.
- **The relative finding stated,** since presence in unaffected relatives is what makes a trait an endophenotype at all.

SEE ALSO Slot II - 80 functional imaging Slot II - 81 electroencephalography

II - 35

Expressed emotion in psychiatry: critically evaluate. [10]

10 SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it is	Five components, rated from the relative, with thresholds
3	The case for it	Relapse prediction, holding across several diagnoses
3	The case against	Culture, direction of causation, cost of measuring
2	The verdict	Modifiable, so worth measuring; never a family blame

THE CONSTRUCT, STATED PRECISELY

The emotional climate of the household toward the patient, rated from a semi-structured interview with the relative. A property of a relationship at a point in time, not a personality of the family.

COMPONENT	WHAT IS RATED	THRESHOLD FOR HIGH
Critical comments	Count of critical remarks about the patient	Six or more
Hostility	Generalised criticism or rejection of the person	Any
Overinvolvement	Overprotection, self-sacrifice, dramatic distress	Rating of three or more
Warmth	Sympathy, concern, interest in the person	Not part of the definition
Positive remarks	Praise and appreciation	Not part of the definition

Rated by the Camberwell Family Interview, which runs over an hour and needs training. The five minute speech sample and self-report questionnaires are shorter proxies with lower sensitivity.

- **The case for it.** High expressed emotion roughly doubles nine to twelve month relapse risk in schizophrenia, and the association holds in mood disorders, eating disorders and substance use.
- **The cultural objection.** Indian samples show lower rates, and overinvolvement means something different where multigenerational co-residence and close supervision are normative.
- **The causal objection.** Criticism rises with the patient's disturbance, so part of the association runs from illness to relative. The relationship is bidirectional.
- **The verdict.** Worth measuring because it is modifiable: family intervention that lowers criticism lowers relapse. That is also why it is never presented to a family as a fault.

WHAT EARNS THE MARKS

- **All five components, with the three that define high expressed emotion marked.** Warmth and positive remarks are rated but excluded.
- **Both objections argued rather than named.** The stem says critically evaluate.
- **A verdict at the end.** A critical evaluation that never lands on a position is an essay, not an answer.

SEE ALSO Slot II - 28 course and outcome Slot II - 31 psychosocial management

AREA 05 OF 14 · P2-A09

Trauma, dissociative and somatic symptom disorders

8	8.4%	II-36	II-43
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 3 · HIGH 1 · SINGLE 4

REVISION ORDER

The two dissociative slots together: one asks management and one asks phenomenology, and they are one answer split. PTSD and pathological grief next. The somatoform and abnormal illness behaviour pair after that. The rape slot and the depression scales slot are independent.

WHAT IS IN THIS AREA

Dissociative and conversion disorder in two slots, post-traumatic stress disorder, pathological grief, the somatoform slot with chronic pain, abnormal illness behaviour, and the rape slot with marital rape and survivor coping. One slot here asks for the rating scales of depression with one described in full. That is an assessment question the boards set inside a trauma area, and it is answered on its page as it was set.

II - 36

Management of dissociative disorders. [10]

10 TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

KNRUHS, RGUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

3 **Diagnose positively**	Positive signs, not a diagnosis of exclusion
4 **The core treatment**	Explanation, rehabilitation, then the psychological work
3 **Traps and prognosis**	What harms, and what predicts recovery

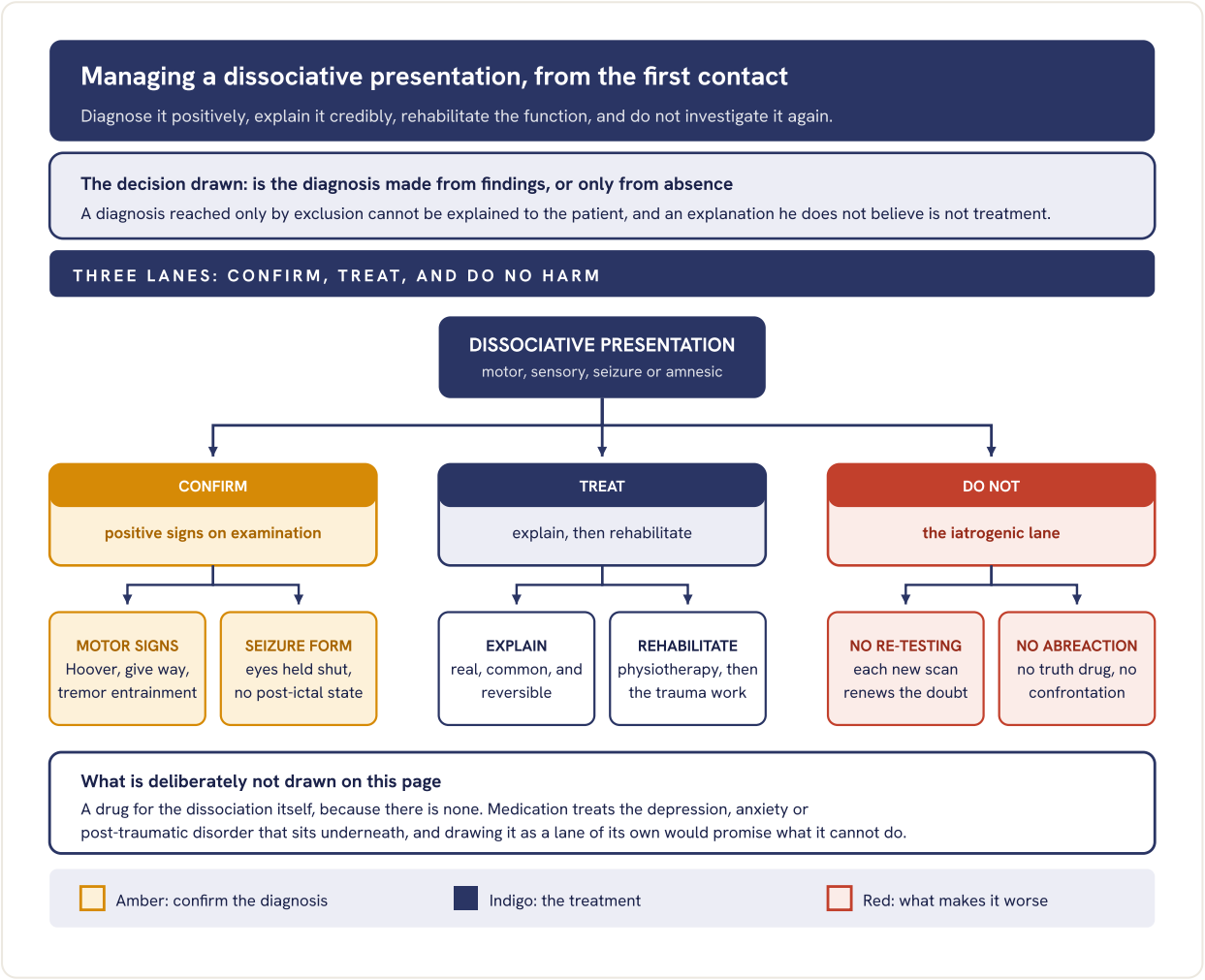


Fig 036.1 Confirmation drawn as a lane of its own, because the explanation given to the patient can only be as convincing as the findings it rests on.

- **How the explanation is given.** Name the diagnosis, show him the positive sign on his own body, say the symptom is genuine and the pathway is reversible, and give the reason the nervous system can produce it. Then hand over to rehabilitation with the same message.

PROGNOSIS, HONESTLY STATED

Short duration, acute onset, a clear precipitant, early diagnosis and the absence of ongoing gain all predict recovery, which in acute presentations is often complete. Long duration before diagnosis, chronic pain, unresolved litigation and prominent comorbidity predict a poorer outcome.

WHAT EARNS THE MARKS

- **A positive sign named,** the first amber leaf, rather than the phrase diagnosis of exclusion.
- **The explanation written as a treatment step,** with the words that are used.
- **Physiotherapy before insight work** in the motor presentations.
- **The red lane,** repeated investigation and abreaction named as harms, not as options.

SEE ALSO Slot II - 43 features and signs of conversion disorder Slot II - 41 somatoform disorder and chronic pain

SPRINT Day 15, Dissociative, conversion and anxiolytic pharmacology

II - 37

Mention the rating scales of depression and describe one of them. [10]

10 TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 The axis	Who rates it, and what the scale is for
4 The scales	Clinician rated, self rated, special populations
3 One described	Structure, scoring, bands, and its use
1 Its limitations	The criticism an examiner is listening for

STATE THE AXIS BEFORE THE LIST

Sort by who rates it and why it is being used. Severity scales measure change and do not make a diagnosis; screening scales find cases and do not grade them. A candidate who states this before listing anything has already earned the first two marks.

GROUP	SCALES	FORMAT	CHIEFLY USED FOR
Clinician rated	Hamilton depression rating scale; Montgomery and Asberg scale	Semi-structured interview	Severity and change in trials
Self rated	Beck depression inventory; Zung self rating scale	Questionnaire	Severity as the patient sees it
Screening	Patient health questionnaire, nine item	Nine items, criterion mapped	Case finding in general settings
Perinatal	Edinburgh postnatal depression scale	Ten items, no somatic items	Screening after childbirth
Elderly	Geriatric depression scale; Cornell scale in dementia	Yes or no; informant based	Avoiding somatic confounding
Medically ill	Hospital anxiety and depression scale	Fourteen items, two subscales	Use where illness mimics somatic items

The amber cells mark the two purposes that are most often confused: measuring change, and finding a case.

- **The Hamilton scale, described.** A clinician administered severity scale from 1960, most often used in its seventeen item form. Items are scored zero to four or zero to two and summed, with conventional bands running from normal through mild, moderate and severe. It takes about twenty minutes and remains the commonest primary outcome measure in antidepressant trials.
- **Its limitations.** It is weighted towards somatic and sleep items, so it inflates in the physically ill and can improve on sedation alone. It is multidimensional rather than a single construct, and it measures severity, never diagnosis.

WHAT EARNS THE MARKS

- **The axis stated first,** clinician against self rated, severity against screening.
- **Special population scales included,** perinatal, elderly and medically ill, which most answers omit.
- **One scale described with its structure and scoring,** as the question asks, not merely named again.
- **The somatic weighting criticism,** which is the line that separates a top answer.

SEE ALSO Slot II - 39 pathological grief Slot II - 38 post-traumatic stress disorder **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 38

Write clinical features, pharmacological management and prognosis of post-traumatic stress disorder. [4+3+3]

4 + 3 + 3 | TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

DNB, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

4 Clinical features	The four clusters, the time rule, the subtypes
3 Pharmacological	First line, the nightmare drug, and what is avoided
3 Prognosis	Recovery rates, and what predicts the chronic course

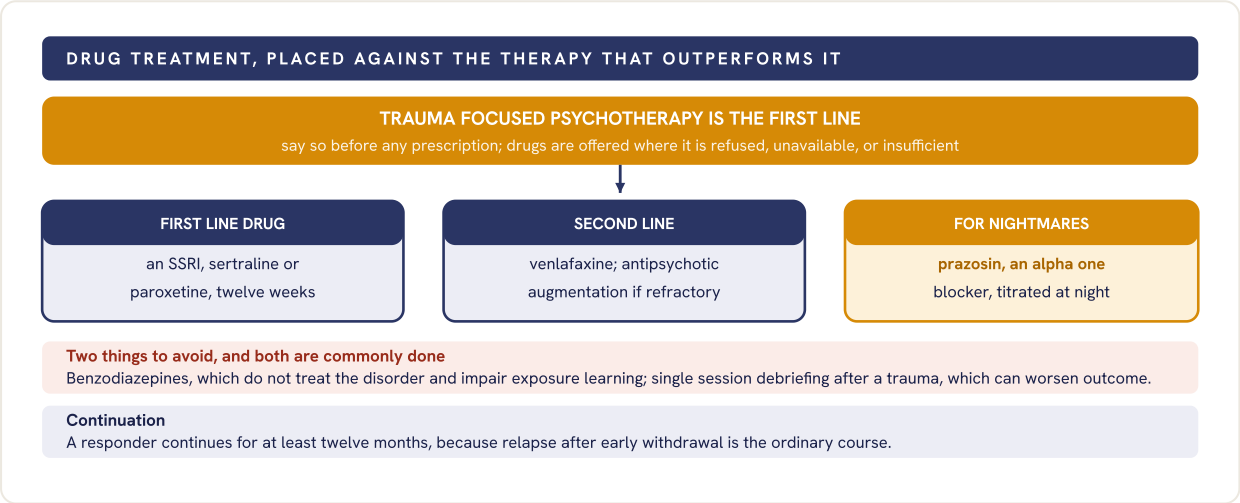


Fig 038.1 The drug lanes drawn beneath the therapy that outranks them, with the two avoidable harms carried as a red strip.

- **Clinical features.** After exposure to death, serious injury or sexual violence: intrusion as memories, nightmares and flashbacks; avoidance of reminders inside and outside; negative alterations in thinking and mood, including amnesia for part of the event and distorted self blame; and hyperarousal with startle, irritability and poor sleep. Longer than one month.
- **The variants to name.** A dissociative subtype with depersonalisation or derealisation, and delayed expression where the picture completes only months later. The newer international classification adds complex post-traumatic disorder, with affect dysregulation, a negative self concept and relationship difficulty.
- **Prognosis.** About half recover within a few months and roughly a third run a chronic course. Interpersonal and intentional trauma, childhood adversity, peritraumatic dissociation, poor social support, ongoing threat and comorbid depression or substance use all predict chronicity.

WHAT EARNS THE MARKS

- **Four symptom clusters named as clusters,** with the one month rule.
- **Psychotherapy placed above drugs,** the amber band, even though only drugs were asked for.
- **Prazosin for nightmares,** the specific answer most candidates never give.
- **Prognosis given with predictors,** not the single word variable.

SEE ALSO Slot II - 39 pathological grief Slot II - 40 coping among survivors of sexual assault

II - 39

Describe the clinical features, risk factors and management of pathological grief. [10]

10 TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

DNB, NTRUHS, RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 **Where the line falls**	Grief against disorder, by time, intensity and culture
3 **Clinical features**	Yearning, identity disruption, avoidance, impairment
2 **Risk factors**	The death, the relationship, and the person
3 **Management**	The grief specific therapy, and what drugs do and do not do

WHERE THE LINE FALLS

Not the presence of grief but its persistence, its intensity and its cost. Both current classifications require intense longing or preoccupation with the person who died, lasting well beyond the expected period for the culture, with impairment. Six months is the earlier threshold, twelve months the later and more conservative one.

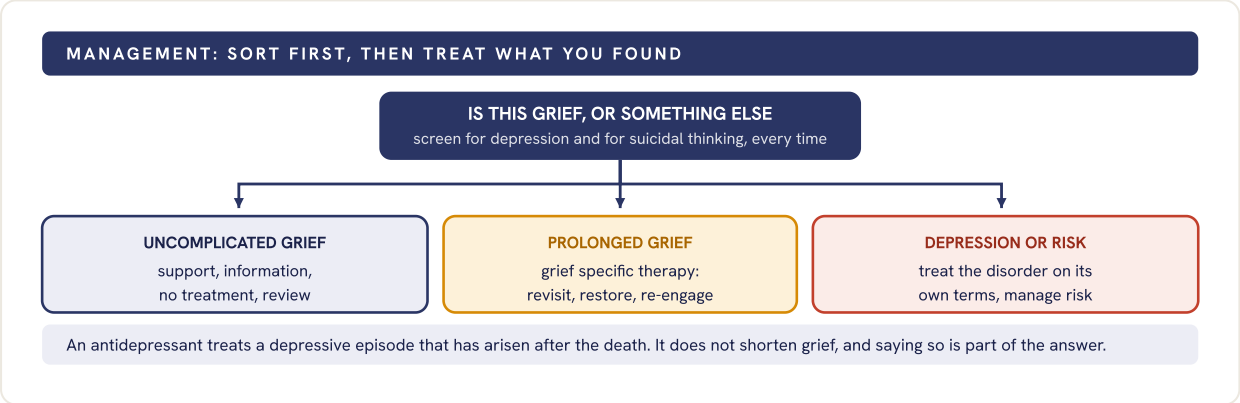


Fig 039.1 One sorting question with three destinations, drawn because the commonest error is treating all three the same way.

- **Clinical features and risk factors.** Intense yearning, preoccupation, disbelief, emotional pain, identity disruption, numbness, avoidance of reminders and a sense that life is meaningless. Risk rises with sudden, violent or self inflicted death, the loss of a spouse or a child, dependent relationships, prior depression, poor support and unresolved conflict with the person who died.
- **Culture is part of the criterion.** Both systems set the threshold against what is expected in the person's own culture and religion. Structured mourning practice with a fixed period is not pathology, and reading it as pathology is a real diagnostic error in Indian practice.

WHAT EARNS THE MARKS

- **The time threshold given as a number,** with both classifications acknowledged.
- **Identity disruption named,** which is the feature separating this from a depressive episode.
- **Grief specific therapy named as the treatment,** the amber box, rather than counselling in general.
- **Culture written into the threshold,** not added as a closing remark.

SEE ALSO Slot II - 38 post-traumatic stress disorder Slot II - 37 rating scales of depression

II - 40

Define rape. What is marital rape? Write in brief about coping skills and quality of life among rape survivors. [2+5+3]

2 + 5 + 3 | TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	The statutory definition, and the seven circumstances
2	Marital rape	The exception, its limits, and the remedies that remain
3	Coping	The phases, and which coping styles predict outcome
3	Quality of life	What is impaired, and what the clinician can change

THE DEFINITION, AND THE EXCEPTION

Indian criminal law defines rape as specified sexual acts by a man with a woman against her will or without valid consent, including consent obtained by fear or fraud, consent she cannot understand, and any act with a girl under eighteen. It was widened in 2013 beyond penetrative intercourse and carried into the criminal code that replaced the older statute in 2024. An exception excludes a husband where the wife is not below eighteen, so marital rape is a distinct offence only where she is a minor. The exception has been challenged on constitutional grounds; check its current status.

PHASE AFTER ASSAULT	WHAT IS SEEN	WHAT THE CLINICIAN DOES
Acute, days to weeks	Shock, disorganisation, numbness or agitation	**Safety, medical care, a believing response**
Outward adjustment	Apparent return to routine, suppression, avoidance	Keep contact open; do not read calm as recovery
Reorganisation	Intrusions, fear, sexual and relational difficulty	**Trauma focused therapy when it is wanted**
If it does not resolve	Post-traumatic disorder, depression, self harm, substance use	Treat the disorder that has formed

A common sequence, not a schedule: any phase may be absent, and returning to an earlier one is not deterioration.

- **Coping, and what predicts outcome.** Avoidance coping, self blame directed at her character rather than at a choice, and social withdrawal all predict worse outcome. Disclosure met with belief, practical support, problem focused coping and re-established routine predict better outcome. A disbelieving or blaming response from family, police or hospital is itself a risk factor.
- **Quality of life, and the clinician's part.** Sleep, sexual function, work, study, mobility and trust are all affected, often for years. What a clinician can change: prompt medical care with emergency contraception and infection prophylaxis, an examination conducted with consent and dignity, no discredited virginity testing, careful documentation for the record, and referral to a support service.

WHAT EARNS THE MARKS

- **The statutory definition with the consent circumstances,** and the age rule.
- **The exception stated accurately,** including that it has been challenged, without asserting an outcome.
- **Coping described by style rather than by stage alone,** since style is what predicts outcome.
- **Secondary victimisation named,** which is the part of the answer a clinician can actually act on.

SEE ALSO Slot II - 38 post-traumatic stress disorder Slot II - 39 pathological grief

II - 41

Discuss the concept of somatoform disorder. Describe the management of a case of a middle aged woman presenting with chronic pain syndrome. [6+4]

6 + 4 | TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The concept	What the older category grouped, and on what basis
3 The shift	From unexplained symptoms to a positive criterion
4 The case	Engage, one doctor, function as the goal

THE CONCEPT, AND HOW IT CHANGED

The older grouping collected physical symptoms suggesting disease that were not fully explained by it, not intentionally produced, and causing distress: somatisation, hypochondriacal, autonomic and persistent pain disorders. The current systems drop the unexplained criterion and define the disorder positively, by disproportionate thoughts, high health anxiety and excessive time given to symptoms, whether or not a physical disease is also present.

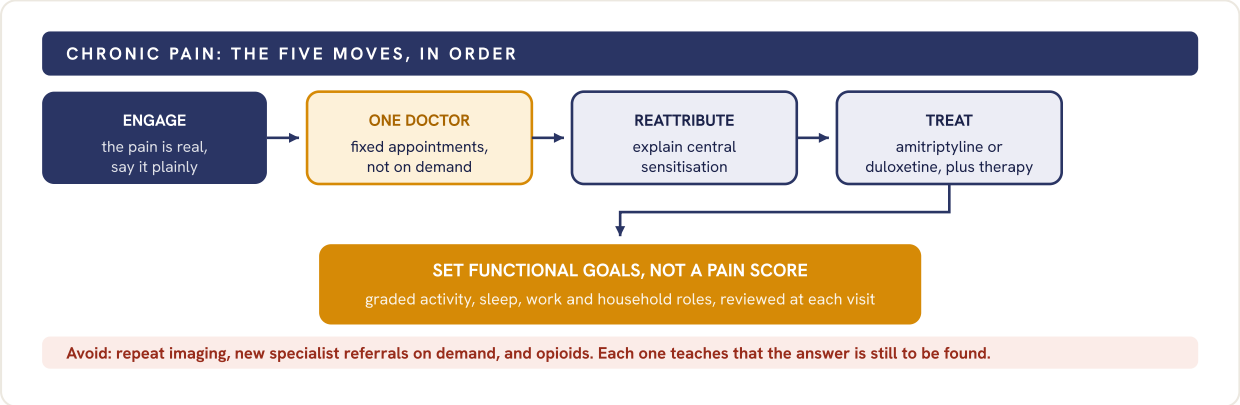


Fig 041.1 Five moves in sequence, ending in the goal that replaces cure, with the three iatrogenic traps carried along the foot.

- **The two errors to avoid in her case.** Investigating again to reassure her, which reassures nobody for long; and telling her the pain is psychological, which she hears as an accusation and which is also not what the current criterion says.

WHAT EARNS THE MARKS

- **The change in the criterion stated,** from medically unexplained to a positive psychological feature.
- **A single coordinating doctor with scheduled visits,** the amber box, which is the intervention with the best evidence here.
- **Function named as the goal instead of pain relief.**
- **The three iatrogenic traps,** repeat imaging, referral on demand, and opioids.

SEE ALSO Slot II - 42 abnormal illness behaviour Slot II - 36 management of dissociative disorders

II - 42

Abnormal illness behaviour. [10]

10 TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	The definition, and the two ideas it was built on
2	The axes	Affirming or denying, conscious or not
4	The four cells	Each with the conditions that sit in it
2	Why it is useful	What it changes in assessment and management

THE DEFINITION

The persistence of a maladaptive way of experiencing, perceiving, evaluating and responding to one's own health, despite an adequate and accurate explanation having been given. It extends the older ideas of the sick role, with its rights and its obligations, and of illness behaviour, which is how anyone responds to bodily change.

AXIS	NOT CONSCIOUSLY MOTIVATED	CONSCIOUSLY MOTIVATED
Illness affirming	Somatic symptom and illness anxiety disorders, conversion presentations	Malingering for an external gain; factitious disorder for the sick role itself
Illness denying	Unawareness of deficit after brain injury; apparent unconcern about a real disability	Deliberate concealment, to keep a job, a licence or a marriage
What decides the cell	Absence of any intended gain on careful history	An identifiable external gain, or evidence of deliberate production
What it changes	Treat, engage, reattribute	Do not confront alone; document, protect, and manage the risk

The amber row is the one that carries the marks, because separating malingering from a somatic symptom disorder is the judgement the concept was created to discipline.

- **Why the concept is useful.** It describes behaviour rather than assigning blame, it holds denial and affirmation in one frame, and it forces the question of motivation to be asked explicitly instead of assumed. A structured questionnaire exists for research use.
- **The honest limit.** Conscious motivation cannot be measured, only inferred, and inference here carries real cost to the patient. Malingering is not a psychiatric diagnosis, and it should be recorded only on evidence that would stand outside the consulting room.

WHAT EARNS THE MARKS

- **The definition quoted, with despite an adequate explanation** in it, which is the operative clause.
- **The sick role named as its ancestor,** with the obligations as well as the rights.
- **Both axes drawn, giving four cells,** not a list of somatoform conditions.
- **The limit on inferring motivation,** which is the ethically serious part of the answer.

SEE ALSO Slot II - 41 somatoform disorder and chronic pain Slot II - 43 features and signs of conversion disorder

II - 43

What are the main clinical features of dissociative (conversion) disorders? Write distinctive physical examination findings in conversion disorder. [3+7]

3 + 7 | TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Clinical features	Loss of integration, the time relation, the varieties
5 The positive signs	By system, each with how it is elicited
2 How signs are used	What they prove, and what they do not

THE CLINICAL FEATURES

A partial or complete loss of the normal integration between memory, awareness of identity, sensation and control of movement. Onset is usually close in time to a stressful event or an insoluble difficulty, symptoms follow the patient's own idea of illness rather than anatomy, and no physical disorder accounts for them.

SIGN	HOW IT IS ELICITED	WHAT IT DEMONSTRATES
Hoover sign	Feel the weak leg's heel while he flexes the good hip against resistance	**Hip extension returns when attention is elsewhere**
Give way weakness	Test power steadily against resistance	Sudden ratchety collapse rather than a fixed weakness
Tremor entrainment	Ask him to tap a rhythm with the other hand	**The tremor takes up that rhythm, or stops**
Tubular visual field	Confrontation at two distances	The field does not widen with distance as it must
Seizure form	Observe the event, and the minutes after it	Eyes held shut, long duration, no post-ictal state
Preserved substrate	Reflexes, tone, plantars, and wasting	All normal in a limb said to be paralysed for weeks

Each sign shows the same thing: the function is present when it is not being attended to. That single sentence is worth writing before the list.

- **What the signs do not prove.** They support a positive diagnosis, they do not exclude coexisting disease, and none of them says anything about motivation. La belle indifference is unreliable and should not be offered as a sign.
- **How to use them.** Demonstrate one to the patient. A sign he can see for himself makes the explanation credible, which is the first step of treatment rather than a separate exercise in diagnosis.

WHAT EARNS THE MARKS

- **Loss of integration named as the core feature,** with the temporal relation to stress.
- **The common principle stated before the list:** function returns when attention moves.
- **At least four named signs with how each is elicited,** as the middle column does.
- **The limit,** that a positive sign does not exclude disease and says nothing about intent.

SEE ALSO Slot II - 36 management of dissociative disorders Slot II - 42 abnormal illness behaviour **SPRINT** Day 15, Dissociative, conversion and anxiolytic pharmacology

AREA 06 OF 14 · P2-A02

Schizophrenia: treatment and antipsychotics

8	8.3%	II-44	II-51
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 · HIGH 1 · RECURRENT 1 · SINGLE 5

REVISION ORDER

Treatment resistance first, it is the anchor. Then the side-effect group as one block: metabolic, tardive dyskinesia and neuroleptic malignant syndrome share most of their material. The haloperidol slot next. Cognitive remediation and the stigma slot last, and neither is long.

WHAT IS IN THIS AREA

Treatment-resistant schizophrenia, first-episode psychosis in an adolescent, the first-generation antipsychotics with haloperidol in detail, the metabolic side effects and their prevention, tardive dyskinesia, neuroleptic malignant syndrome, cognitive remediation in rehabilitation, and a stigma and discrimination slot that asks for a local policy.

II - 44

Treatment resistant schizophrenia. [10]

10 SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB, RGUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

3	The definition	Four conditions, all of them, before the label is used
2	Pseudo-resistance	What is excluded first, and how each is checked
4	Clozapine	Titration, target, plasma level, and the monitoring
1	If clozapine fails	Augmentation, with the one option that has evidence

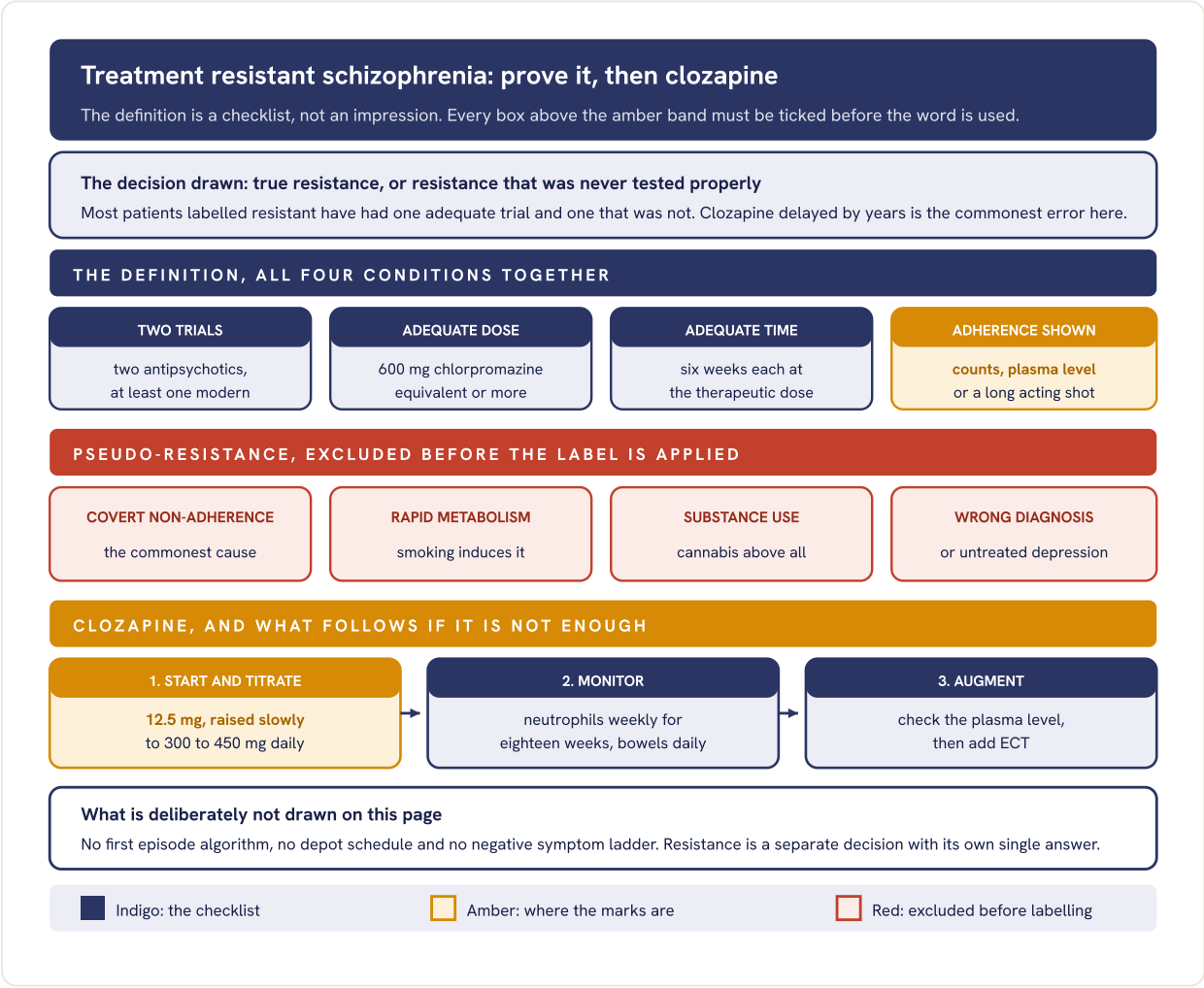


Fig 044.1 The four conditions that define resistance, the four causes of apparent resistance that are excluded first, and the clozapine sequence that follows once the label is earned.

- **Clozapine.** The only agent with established efficacy here, and roughly a third to a half respond. Non-response at a normal dose is checked against a plasma level, with about 350 nanograms per millilitre as the threshold worth reaching before it is called a failure.
- **Its dangers, in order of what actually kills.** Constipation progressing to ileus, then myocarditis in the first weeks, then agranulocytosis in about one per cent. Seizures rise with dose. Sedation, sialorrhoea and weight gain drive most discontinuation.

PITFALL

Adding a second antipsychotic instead of starting clozapine. Combination has weak evidence and certain added harm, and every month spent on it is a month of untreated illness in the period when function is still recoverable.

WHAT EARNS THE MARKS

- **All four parts of the definition, including documented adherence.** Most answers give two trials and stop.
- **Pseudo-resistance excluded before clozapine is named.**
- **Clozapine written with its titration, target range and monitoring schedule,** not just as a word.
- **Electroconvulsive therapy named as the augmentation with the best evidence.**

SEE ALSO Slot II - 29 negative symptoms Slot II - 50 metabolic effects Slot II - 52 treatment resistant depression

II - 45

Describe the pharmacological management of first episode psychosis in an adolescent. [10]

10 SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Before the first dose**	Organic and substance screen, baselines, consent and assent
4	**The first choice**	Which agent, what starting dose, what to avoid
2	**If it does not work**	Switch once, then clozapine after two adequate trials
2	**Duration and monitoring**	How long to continue, and what is tracked meanwhile

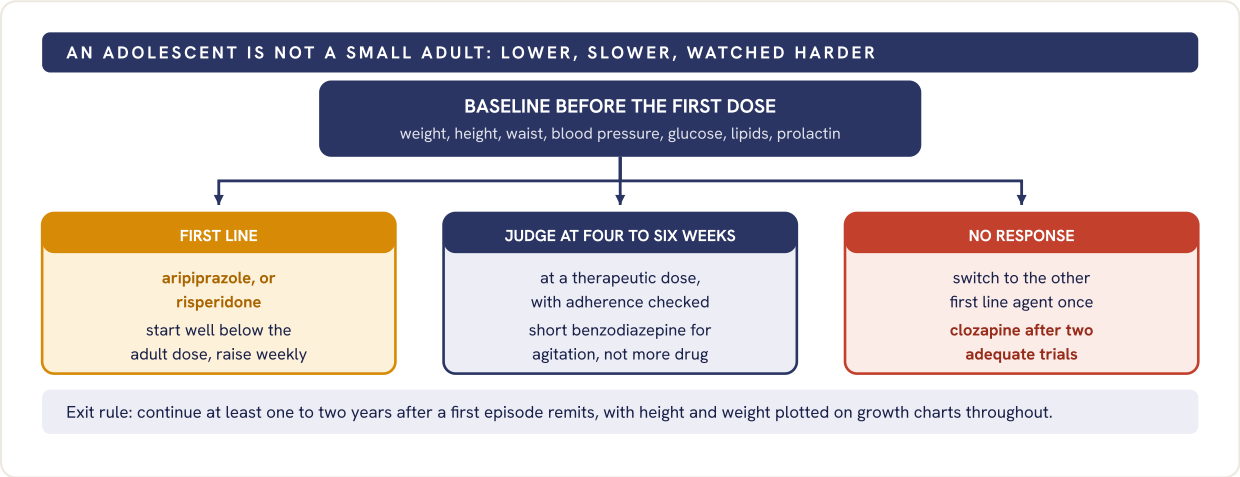


Fig 045.1 First episode pharmacology in an adolescent: the baseline that precedes the first dose, the two first line agents, and the point at which clozapine becomes the answer.

COLOUR CODE assessment and review · the drug choice · failure pathway

- **Before prescribing.** Confirm the diagnosis, exclude substance-induced and organic psychosis, record the metabolic baseline. A minor cannot consent alone: the guardian is the nominated representative, and assent is still sought.
- **Why these two agents.** Adolescents gain weight and raise prolactin more readily than adults. Aripiprazole is metabolically the least costly, risperidone raises prolactin most, olanzapine is avoided first line.
- **Pharmacology is half the plan.** Family psychoeducation and school liaison run in parallel from week one, and the answer should say so.

PITFALL

Raising the dose at two weeks for apparent non-response. Response in a drug-naive first episode builds over four to six weeks; early escalation buys side effects and a false diagnosis of resistance.

WHAT EARNS THE MARKS

- **The metabolic baseline written before the first dose.**
- **A named first line agent with a reason,** and olanzapine explicitly declined at this age.
- **An adequate trial defined, then the switch, then clozapine.** The sequence is the answer.

SEE ALSO Slot II - 30 early psychosis Slot II - 50 metabolic effects

II - 46

What is neuroleptic malignant syndrome? How would you confirm the diagnosis? Write the management of neuroleptic malignant syndrome. [2+4+4]

2 + 4 + 4 | SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **What it is**	Idiosyncratic reaction to dopamine blockade, and the tetrad
4 **Confirming it**	Criteria, the laboratory pattern, and what must be excluded
3 **Management**	Stop, support, then the specific agent by severity
1 **Rechallenge**	The rule that prevents a second episode

- **What it is.** An idiosyncratic, life threatening reaction to dopamine blockade. The tetrad is hyperthermia, lead pipe rigidity, altered sensorium and autonomic instability, usually within two weeks of a dose change.
- **Confirming it.** No test confirms it, so the diagnosis is criterion based: fever, rigidity and a creatine kinase commonly above a thousand units per litre, with leucocytosis, low serum iron and myoglobinuria. Imaging and cerebrospinal fluid exclude.
- **What must be excluded.** Serotonin syndrome, with clonus and hyperreflexia within hours. Malignant catatonia, where catatonic signs precede the drug. Malignant hyperthermia, anticholinergic toxicity, infection and heat stroke.

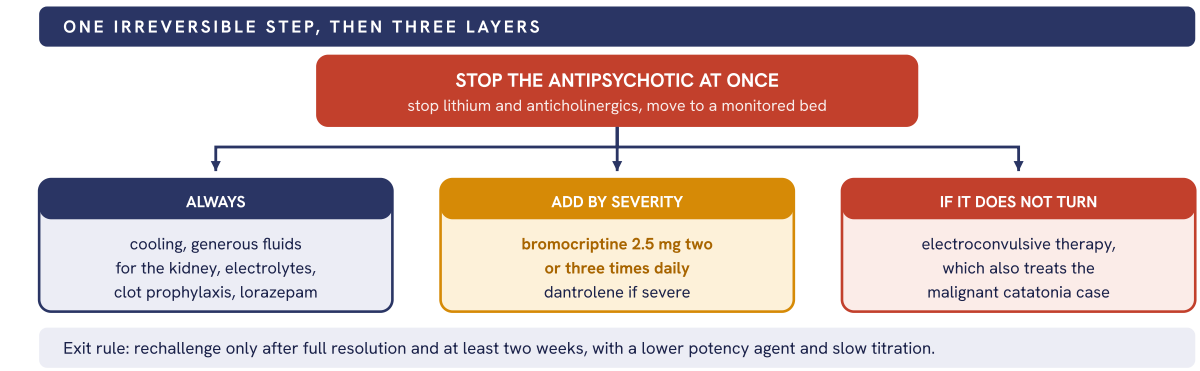


Fig 046.1 One irreversible action, then three layers: always, by severity, and if it does not turn.

PITFALL

Treating the fever as infection and continuing the antipsychotic. Rigidity with a rising creatine kinase after a dose change is this syndrome until proved otherwise.

WHAT EARNS THE MARKS

- **The tetrad named as a tetrad, with its time relation to the drug.**
- **Criterion-based diagnosis stated,** with the honest line that no test confirms it.
- **Stopping the drug written as the first line of management.**
- **The rechallenge rule.** Most answers stop at recovery and lose the last mark.

SEE ALSO Slot II - 51 tardive dyskinesia Slot II - 91 catatonia

II - 47

Classify first generation antipsychotics. Describe the mechanism of action, metabolism and side effect profile of haloperidol. [5+5]

5 + 5 | SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The axis	Chemical class first, then the potency axis that matters clinically
3	The classes	Four groups, each with its representative drugs
3	Haloperidol pharmacology	Receptor profile, occupancy window, handling by the liver
2	Its adverse effects	Grouped by the pathway that produces each of them

NAME THE AXIS BEFORE THE LIST

The classification is by chemical structure. The axis that predicts what a patient will feel is potency, and the two do not line up.

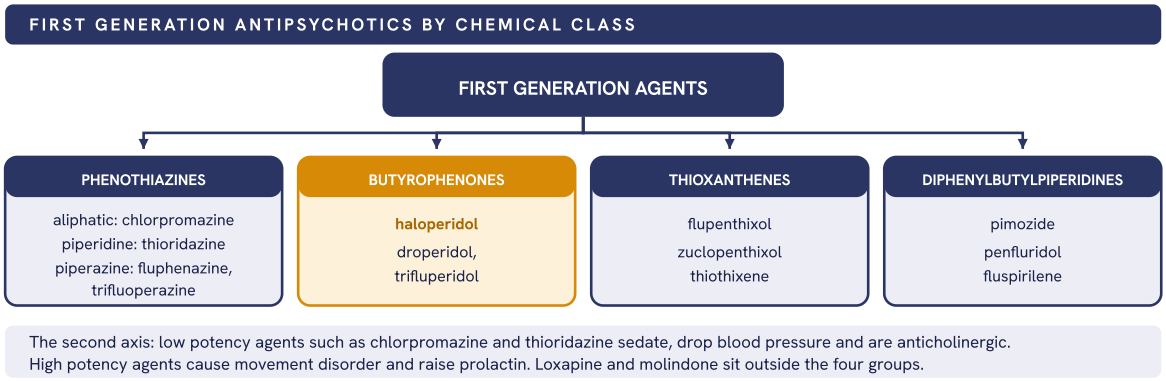


Fig 047.1 One axis, four branches, with the potency axis carried as a strip along the foot.

- **Mechanism.** A high potency D2 antagonist with little muscarinic, histamine or alpha-1 affinity. Antipsychotic effect appears near sixty five per cent striatal D2 occupancy; above eighty per cent, movement disorder is near certain.
- **Metabolism.** Well absorbed, heavily protein bound, lipophilic, with first pass loss that makes the oral dose exceed the parenteral. Cleared through CYP3A4, CYP2D6 and glucuronidation, half life roughly a day. The decanoate is given about every four weeks.
- **Adverse effects, by pathway.** Nigrostriatal blockade gives dystonia, akathisia, parkinsonism and later tardive dyskinesia; tuberoinfundibular blockade raises prolactin. Beyond them: the malignant syndrome, a lowered seizure threshold, and QT prolongation.

WHAT EARNS THE MARKS

- **The chemical axis stated, then the potency axis named as the clinically useful one.**
- **Four classes with named representatives,** not a flat list of drug names.
- **The occupancy window, with both numbers.** It explains the whole side effect profile in one line.
- **Adverse effects grouped by pathway,** which is what separates a top answer from a recalled list.

SEE ALSO Slot II - 46 neuroleptic malignant syndrome Slot II - 51 tardive dyskinesia

II - 48

What is cognitive remediation? Discuss its role in rehabilitation in schizophrenia. [2+8]

2 + 8 | SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	The consensus definition and its four active ingredients
3 What is trained	Neurocognition and social cognition, domain by domain
3 The evidence	Effect on cognition, smaller on function, and the condition
2 Place in rehabilitation	An adjunct, and what it must be paired with to pay

THE DEFINITION, AND ITS FOUR INGREDIENTS

Behavioural training that improves cognitive processes, aiming at durability and generalisation to daily function. Four ingredients define it: a trained therapist, repeated practice, explicit strategy development, and transfer procedures.

DOMAIN TRAINED	HOW IT IS TRAINED	WHAT IT SHOULD LOOK LIKE IN DAILY LIFE
Processing speed	Timed symbol and coding drills	Keeping pace with a conversation or task
Attention	Sustained detection tasks	Sitting through a shift or a class
Working memory	Span and running memory tasks	Holding an instruction long enough to act
Verbal learning	Structured list and story recall	Remembering a medication schedule
Social cognition	Emotion recognition, perspective taking	Reading intent, so fewer misfires

The third column is the point. Training that never leaves the third column has failed its own definition.

- **The evidence.** Meta-analyses show a small to moderate gain in global cognition and a smaller one in functioning, durable at follow-up. The functional gain is substantially larger when remediation runs alongside psychiatric rehabilitation than when it runs alone.
- **Why cognition is the target.** Cognitive impairment predicts work, independent living and social function better than positive symptoms do, and it persists through symptomatic remission. Antipsychotics do not treat it.
- **The honest limit.** It is an adjunct, not a stand-alone treatment. Paired with supported employment it improves job tenure; delivered as computer exercises with nothing attached, it improves test scores and little else.

WHAT EARNS THE MARKS

- **The four ingredients named,** since the two definition marks sit there.
- **Social cognition included as a target,** not only neurocognition.
- **The condition on the evidence:** functional benefit depends on pairing it with rehabilitation.
- **Cognition stated as a better predictor of function than symptoms.**

SEE ALSO Slot II - 29 negative symptoms Slot II - 31 psychosocial management **SPRINT**

Day 9, Schizophrenia: management, antipsychotics and adverse effects

II - 49

What do you understand by stigma and discrimination? Describe different types of discrimination of recent time in India. How can a policy be framed to improve stigma associated with mental illness in your locality? [3+4+3]

3 + 4 + 3 | SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The two constructs	Stigma as a process, discrimination as the behaviour it produces
4 Types in India now	Work, insurance, civil law, health care, the family
2 Framing a local policy	Target a behaviour, use contact, use platforms that exist
1 Measuring it	An attitude score and a service outcome, before and after

THE TWO WORDS, SEPARATED

Stigma is a process with five parts, needing a power difference to operate: labelling, stereotyping, separation into us and them, status loss, and discrimination. Discrimination is the last part made visible. Stigma is public, internalised, structural, or attached to the family.

WHERE	HOW IT APPEARS NOW	WHAT THE LAW SAYS
Employment	Not hired after disclosure, moved out after an episode	Prohibited; reservation in the public sector
Insurance	Cover refused or priced apart from physical illness	Parity required under section 21(4)
Civil law	Capacity assumed absent; illness cited in matrimonial cases	Capacity presumed, assessed decision by decision
Health care	Diagnostic overshadowing, poorer physical care	Chaining prohibited; least restrictive setting
Family	Concealment, restricted marriage prospects	Not reachable by law; contact works here

The last row matters most in Indian practice and is the only one a district level policy can move directly.

- **Framing the policy.** Name one audience and one behaviour, not awareness in general. Contact based intervention, direct or filmed, with a person who has lived experience, outperforms education alone.
- **Delivery.** Use platforms that already exist, the district programme, schools, primary care and local government, and add a complaint route so the protections above are enforceable.
- **Measurement.** One attitude scale before and after, plus one service outcome such as help-seeking delay. Single awareness days shift knowledge and leave behaviour untouched.

WHAT EARNS THE MARKS

- **Stigma given as a process with named parts,** and discrimination as its behavioural end.
- **Indian examples with the statutory position beside each.** The stem asks for recent India, not a general account.
- **Contact named as the active ingredient,** and a measurement plan attached. A policy with no outcome is an intention.

SEE ALSO Slot II - 33 chronic care in India Slot II - 31 psychosocial management

II - 50

Discuss metabolic side effects of the antipsychotics and strategies used to prevent such side effects. [3+7]

3 + 7 | SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 What the problem is	Five components, the excess risk, the mortality it drives
2 Why it happens	Receptor mechanisms, plus illness and circumstance
3 Prevention	Agent choice, monitoring with a threshold, lifestyle from day one
2 Once it has happened	Switch, metformin, and treating each component

STATE THE SYNDROME, THEN THE STAKE

Central obesity, raised triglycerides, low high density lipoprotein, raised blood pressure and raised fasting glucose. Around a third of patients meet the criteria, roughly twice the background rate, and this is the main reason life expectancy in schizophrenia is fifteen to twenty years shorter.

AGENT	METABOLIC RISK	WHAT FOLLOWS FROM IT
Clozapine, olanzapine	**Highest**	Used where efficacy justifies it, with the tightest monitoring
Quetiapine, risperidone, paliperidone	Intermediate	The common first choices, so the schedule must be real
Aripiprazole, lurasidone, ziprasidone	**Low**	Preferred at first episode and in young patients
Amisulpride, haloperidol	Low for weight	Cost is paid in prolactin and movement instead

Most weight is gained in the first months and is the hardest to reverse, so the first prescription matters more than any later switch.

- **Mechanism.** Histamine H1 and serotonin 5-HT2C blockade drive appetite, muscarinic M3 blockade blunts insulin release, sedation cuts activity. Diet, smoking and low income add to it independently.
- **Monitoring, with the numbers.** Weight, body mass index and waist at baseline, monthly for three months, then quarterly. Blood pressure, fasting glucose or glycated haemoglobin and lipids at baseline, at three months, then yearly. A gain above seven per cent of baseline weight is the trigger to act.
- **Prevention.** Choose a lower risk agent where efficacy allows, start diet and activity alongside the drug rather than after the weight arrives, and name who owns the physical health review.
- **Once it has happened.** Switch where the illness permits, add metformin started at 500 mg and titrated up, which is the best evidenced option, and treat each component to ordinary targets.

WHAT EARNS THE MARKS

- **The five components named,** then linked to the mortality gap rather than left as a list.
- **Agents ranked by risk,** which is what makes the prevention section actionable.
- **A monitoring schedule with intervals and an action threshold.**
- **Metformin named,** and physical health care assigned to a named person rather than left to nobody.

SEE ALSO Slot II - 44 treatment resistance Slot II - 45 first episode pharmacology

II - 51

Aetiology and management of tardive dyskinesia. [10]

10 SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Phenomenology, and the exposure rule that allows the diagnosis
3 Aetiology	Receptor supersensitivity, and what has been added to it
2 Who gets it	Risk factors, with the strongest one named first
3 Management	Remove, switch, then the specific drug class

- **What it is.** Involuntary repetitive choreoathetoid movements, classically buccolingual and masticatory, arising after months of dopamine antagonist exposure and persisting after the drug is reduced. Three months of exposure is conventionally required, one month above sixty.
- **Aetiology.** Classically postsynaptic D2 supersensitivity from chronic blockade. Added to it: maladaptive synaptic plasticity, loss of striatal inhibitory interneurons, oxidative injury, and genetic variation in the D2 and D3 receptors.
- **Risk factors.** Older age is the strongest, then cumulative dose and duration, first generation and high potency agents, early acute movement effects, diabetes and mood disorder. Incidence is roughly five per cent a year with older agents, lower but not zero with newer ones.

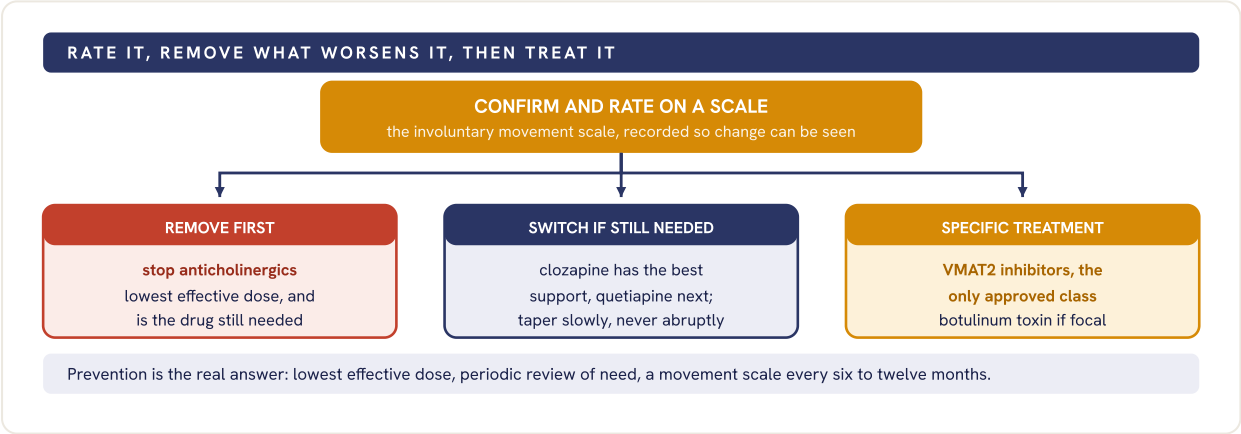


Fig 051.1 Management in order: rate the movements, remove what worsens them, switch if an antipsychotic is still required, then treat.

PITFALL

Adding trihexyphenidyl. It relieves acute dystonia and parkinsonism and makes tardive dyskinesia worse. Valbenazine and deutetrabenazine are the approved agents; in Indian practice tetrabenazine is the accessible member of the class.

WHAT EARNS THE MARKS

- **The exposure rule stated,** so the diagnosis is defined rather than described.
- **Supersensitivity given as the classical account with newer mechanisms added,** not instead of them.
- **Anticholinergic withdrawal written before any new drug,** and the VMAT2 class named.

SEE ALSO Slot II - 47 first generation antipsychotics Slot II - 46 malignant syndrome

AREA 07 OF 14 · P2-A05

Mood disorders: pharmacotherapy

7	7.5%	II-52	II-58
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 RECURRENT 1 SINGLE 5

REVISION ORDER

The two treatment-resistance slots together; one defines it and the other asks for five strategies, and they are the same answer twice. The two ECT slots next, likewise. Lithium and the MAOI slot are single-drug answers. The suicide neurobiology slot last, and it is the one that needs reading of its own.

WHAT IS IN THIS AREA

Treatment-resistant depression in two slots, the monoamine oxidase inhibitors, the long-term adverse effects of lithium, and a suicide slot that asks for the neurobiology and the immunometabolic depression link. Two of the seven slots are electroconvulsive therapy questions: mechanisms, preparation and monitoring in one, and the history, pre-treatment imaging and side effects in the other. The area is named for pharmacotherapy and the boards put the physical treatments in it.

II - 52

Treatment-resistant depression. [10]

10 MOOD DISORDERS: PHARMACOTHERAPY

DNB, KNRUHS, KUHS, MUHS, RGUHS - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2	The definition	Two adequate trials, and what adequate actually means
2	Pseudo-resistance	The four causes excluded before the label is used
4	The drug ladder	Optimise, switch, augment, combine, each given time
2	Beyond drugs	Added therapy, ECT, and the rapid-acting agents

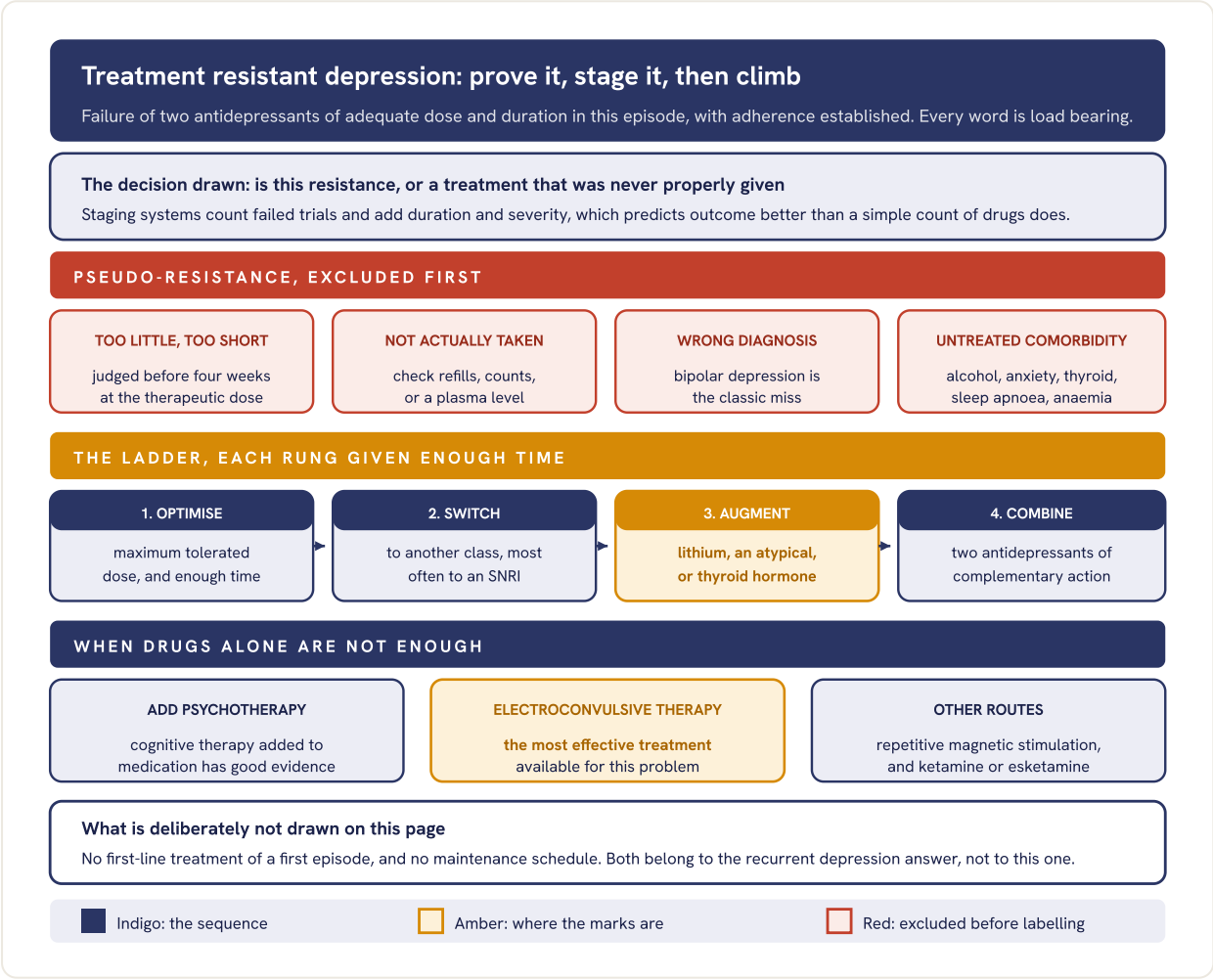


Fig 052.1 The four causes of apparent resistance excluded first, then the drug ladder, then what is done when the ladder runs out.

- **Augmentation, with the detail.** Lithium is the classical agent and carries the anti-suicide evidence. Atypical antipsychotics have the largest randomised evidence base. Thyroid hormone is the third established option, and each augmentation is judged over weeks rather than days.
- **The rapid-acting agents.** Ketamine and esketamine act within hours, including on suicidal thinking, which no oral antidepressant does. The benefit is short lived without repeated dosing, so they buy time rather than replace the ladder.

PITFALL

Counting trials the patient never really had. A drug stopped at two weeks, or at half the therapeutic dose, or never collected from the pharmacy, is not a failed trial, and an answer that does not say so has skipped the first two marks.

WHAT EARNS THE MARKS

- **The definition unpacked,** with adequate dose, adequate duration and confirmed adherence each spelled out.
- **Pseudo-resistance excluded before the ladder starts.**
- **The ladder in order, with augmentation agents named.**
- **Electroconvulsive therapy identified as the most effective option,** not held back as a last resort.

SEE ALSO Slot II - 57 true and pseudo resistance Slot II - 62 recurrent depressive disorder

II - 53

Classify monoamine oxidase inhibitors. What are the common side effects? What instructions would you give to your patient while prescribing one? [3+4+3]

3 + 4 + 3 | MOOD DISORDERS: PHARMACOTHERAPY | DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	The axis and the classes	Selectivity and reversibility, then the drugs in each box
2	Everyday side effects	What actually limits the dose in practice
2	The two emergencies	Hypertensive crisis and serotonin syndrome
3	What the patient is told	Food, drugs, warning signs, the card, the washout

CLASS	DRUGS	WHY THE CLASS MATTERS
Non-selective, irreversible	Phenelzine, isocarboxazid, tranylcypromine	**Full dietary restriction; the tyramine risk lives here**
Selective A, reversible	Moclobemide	**Displaced by tyramine, so the diet is far less restrictive**
Selective B, irreversible	Selegiline, rasagiline	Selective at low dose only; selectivity is lost as the dose rises

The classification is by selectivity and reversibility, and both axes exist only because they change what the patient may eat.

- **Everyday side effects.** Postural hypotension is the usual dose-limiting problem. Then insomnia, weight gain, sexual dysfunction and peripheral oedema. Hydrazines can be hepatotoxic; tranylcypromine is stimulant in character and can be misused.
- **The two emergencies.** Hypertensive crisis from tyramine in food or from a sympathomimetic, and serotonin syndrome from any serotonergic drug added on top. Both are avoidable and both are what the instructions exist to prevent.
- **What the patient is told.** Avoid aged cheese, cured and fermented meat, soy sauce, yeast extract, broad bean pods and draught beer; fresh food is safe. Take nothing new without asking, especially cough remedies, decongestants, pethidine, tramadol and other antidepressants. A severe throbbing headache with palpitations means hospital now. Carry a card, tell any dentist or anaesthetist, and allow a two week washout before a serotonergic drug is started.

WHAT EARNS THE MARKS

- **Both axes of the classification named,** selectivity and reversibility, before the drug list.
- **Moclobemide's lighter dietary restriction explained, not just stated.**
- **The instructions written as instructions,** in words a patient could repeat back, since that is what the last three marks reward.

SEE ALSO Slot II - 52 treatment resistant depression Slot II - 46 malignant syndrome

II - 54

What are the mechanisms of action of ECT? How would you prepare a patient for modified ECT? Write about monitoring of a patient undergoing modified ECT. [3+3+4]

3 + 3 + 4 | MOOD DISORDERS: PHARMACOTHERAPY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 **Mechanisms**	Four hypotheses, and the honest line that none is settled
3 **Preparation**	Consent, workup, fasting, and the drugs to withhold
2 **Monitoring during**	Seizure adequacy, and the physiological parameters
2 **Monitoring across the course**	Response, cognition, threshold, and when to stop

MECHANISMS, AS FOUR HYPOTHESES

Neurotransmitter: enhanced serotonergic and dopaminergic transmission with altered receptor sensitivity. Anticonvulsant: seizure threshold rises across a course, and that rise tracks response. Neuroendocrine: hypothalamic release and normalisation of the stress axis. Neuroplastic: increased neurotrophic factor and hippocampal volume. None is established alone, and saying so is part of the answer.

STAGE	WHAT IS DONE	WHAT IS EASY TO FORGET
Consent	Informed consent, or the nominated representative where capacity is absent	**Unmodified ECT is prohibited; minors need Board permission**
Workup	Physical and cardiac review, haemogram, sugar, electrolytes, renal and liver function, ECG	Imaging only where clinically indicated, never routinely
The night before	Fasting six to eight hours, bladder emptied, dentures and ornaments removed	**Withhold benzodiazepines and anticonvulsants; review lithium**
In the suite	Anticholinergic, induction agent, muscle relaxant, preoxygenation, bite block	A cuffed limb, so the motor seizure can be timed

- **Monitoring during the treatment.** Continuous pulse oximetry, electrocardiogram and blood pressure, with electroencephalographic and motor seizure duration recorded. An adequate generalised seizure is expected, and a missed or very brief seizure is restimulated at a higher charge in the same session.
- **Monitoring across the course.** Symptom response on a rating scale before every second treatment, orientation and memory checked before each session, seizure threshold and charge tracked, and the course ended when improvement plateaus rather than at a fixed number.

WHAT EARNS THE MARKS

- **Four named mechanisms with the honest closing line,** rather than one confident theory.
- **Consent handled under the statute,** including the prohibition on unmodified treatment.
- **Seizure adequacy described as something measured,** with what is done when it is inadequate.
- **Cognitive monitoring given its own place,** because it is what decides whether the course continues.

SEE ALSO Slot II - 56 history and side effects of ECT Slot II - 52 treatment resistant depression

II - 55

Describe the long-term adverse effects of lithium therapy. [10]

10 | MOOD DISORDERS: PHARMACOTHERAPY | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Frame the answer	Long term effects, not toxicity, and why the distinction matters
4	System by system	Kidney, thyroid, parathyroid, then the rest
2	What makes it worse	Drugs, dehydration, and episodes of toxicity
2	Monitoring	What is checked, how often, and when it is checked sooner

SEPARATE TWO QUESTIONS BEFORE STARTING

Long-term adverse effects accumulate at therapeutic concentrations over years. Toxicity is an acute event at high concentrations. An answer that describes tremor, ataxia and confusion has answered the wrong question.

SYSTEM	WHAT HAPPENS	WHAT IS DONE ABOUT IT
Kidney	Nephrogenic diabetes insipidus with polyuria and thirst; slow interstitial nephropathy over decades	**Track the estimated filtration rate; amiloride for the polyuria**
Thyroid	Hypothyroidism and goitre, commoner in women	Thyroxine added; lithium is not stopped for this
Parathyroid	Raised parathyroid hormone with hypercalcaemia	**Check calcium; it is the effect most often missed**
Metabolic and skin	Weight gain, acne, psoriasis worsened, hair thinning	Weight tracked; dermatology for the skin, not withdrawal
Heart and nerve	Benign T-wave change, sinus node dysfunction in the elderly, fine tremor	Propranolol for tremor; electrocardiogram in older patients

- **Monitoring.** A twelve hour post-dose level every three to six months, renal function and thyroid function every six months, calcium once a year, and weight at each review. All of it sooner after a dose change, in the elderly, and in anyone with impaired renal function.
- **What drives the risk up.** Non-steroidal anti-inflammatory drugs, thiazides, drugs acting on the renin-angiotensin system, dehydration in hot weather, and every past episode of toxicity. Cerebellar damage after severe toxicity can be permanent.
- **The line that should end the answer.** None of these is a reason to stop a drug that prevents relapse and reduces suicide. They are reasons to monitor, and to make the decision with the patient.

WHAT EARNS THE MARKS

- **Long-term effects separated from acute toxicity in the first line.**
- **Parathyroid included,** which is the omission that separates a good answer from an average one.
- **A monitoring schedule with intervals,** not a general instruction to check levels.
- **The closing judgement,** that adverse effects are managed rather than used as a reason to stop.

SEE ALSO Slot II - 63 bipolar depression Slot II - 59 course and outcome

II - 56

Briefly describe the history of development of electroconvulsive therapy in psychiatry. What are the indications of brain imaging before modified ECT? What are the side effects of modified ECT? [5+2+3]

5 + 2 + 3 | MOOD DISORDERS: PHARMACOTHERAPY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

5	The history	A wrong idea, a right treatment, then a century of refinement
2	Imaging before ECT	Never routine; the findings that make it necessary
2	Side effects	Immediate, cognitive, cardiovascular, anaesthetic
1	The safety statement	Mortality, put in a comparison a reader can use

STEP	WHAT CHANGED	WHY IT MATTERED
The premise	Meduna induced seizures on the belief that epilepsy and schizophrenia were antagonistic	**The premise was wrong, the treatment worked anyway**
Chemical to electrical	Camphor, then pentylenetetrazol, then Cerletti and Bini in 1938	Electricity made the seizure predictable and timed
Modification	Curare, then succinylcholine with general anaesthesia	Fractures and dislocations effectively disappeared
Refinement	Unilateral placement; sine wave replaced by brief and ultrabrief pulse	**Same efficacy at a much lower cognitive cost**
Regulation	Anaesthesia and muscle relaxant made a legal requirement in India	Unmodified treatment is prohibited outright

- **Imaging is not routine.** It is indicated by clinical findings: focal neurological signs, papilloedema or other evidence of raised intracranial pressure, a new or atypical headache, recent head injury or stroke, unexplained seizures, known intracranial disease, and late onset illness with unexplained cognitive decline.
- **Side effects.** Immediate: headache, jaw and muscle ache, nausea, post-ictal confusion. Cognitive: anterograde amnesia that recovers over weeks, and retrograde loss of autobiographical memory that can persist, worst with bilateral placement, sine wave and high charge. Cardiovascular: a brief bradycardia then tachycardia and hypertension. Rarely prolonged seizure, dental injury or a switch into mania.
- **The safety statement.** Mortality is of the order of a few deaths per hundred thousand treatments, comparable to minor surgery under general anaesthesia, and most of that risk is anaesthetic rather than the seizure.

WHAT EARNS THE MARKS

- **The history told as a sequence with what each step fixed,** not as a list of names and years.
- **Imaging stated as not routine,** then justified by named clinical findings.
- **Cognitive effects split into anterograde and retrograde,** with what makes each worse.
- **A mortality figure with a comparison,** which is what makes the safety claim usable.

SEE ALSO Slot II - 54 mechanisms and monitoring Slot II - 91 catatonia

II - 57

Differentiate between true and pseudo resistance in psychopharmacotherapy. How do we define treatment resistant depression? Enumerate five strategies for managing treatment resistant depression. [3+2+5]

3 + 2 + 5 | MOOD DISORDERS: PHARMACOTHERAPY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The grid	Domain by domain, with what settles each one marked
2 The definition	Two adequate trials, spelled out word by word
5 Five strategies	Exactly five, numbered, in the order they are used

DOMAIN	PSEUDO-RESISTANCE	TRUE RESISTANCE	WHAT SETTLES IT
Dose	Never reached the therapeutic range	Maximum tolerated dose reached	The prescription record, not the recollection
Duration	Judged before four weeks at that dose	Four to six weeks at that dose	Date the dose was reached, not the date started
Adherence	Partial or covert non-adherence	Adherence established	Pharmacy refills, counts, a plasma level
Diagnosis	Bipolar depression, dysthymia, substance use	The diagnosis holds on review	A life chart and collateral history
Comorbidity	Untreated anxiety, alcohol, thyroid, sleep apnoea	Comorbidity identified and treated	Screening bloods and a sleep history

The definition applies only after every left-hand cell has been excluded, which is the point of putting the grid before the definition.

- **The definition.** Failure to respond to two or more antidepressants, each of adequate dose and adequate duration, in the current episode, with adherence established. Staging systems refine it by adding episode duration and symptom severity, which predict outcome better than a count of failed drugs alone.
- **Five strategies.** One, optimise to the maximum tolerated dose for long enough. Two, switch to another class. Three, augment with lithium, an atypical antipsychotic or thyroid hormone. Four, combine two antidepressants of complementary action. Five, neurostimulation, where electroconvulsive therapy is the most effective option available.
- **Two things that are not on that list of five but belong in the plan.** Cognitive therapy added to the medication, and treating the comorbidity that is holding the depression up.

WHAT EARNS THE MARKS

- **A grid, not two paragraphs,** with the settling test named in each row.
- **The definition given word by word,** since adequate dose, adequate duration and adherence each carry weight.
- **Exactly five strategies, numbered.** The stem asks for five, and six is as wrong as four.

SEE ALSO Slot II - 52 treatment resistant depression Slot II - 44 treatment resistant schizophrenia

II - 58

What are the evidence based risk and protective factors of suicide? Describe the neurobiology of suicide. Discuss the relationship between immunometabolic depression and antidepressants. [4+3+3]

4 + 3 + 3 | MOOD DISORDERS: PHARMACOTHERAPY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	Risk and protection	Distal and proximal risk, protection, and the Indian pattern
3	Neurobiology	The stress-diathesis model, and the findings under each half
3	Immunometabolic depression	What the construct is, and what it predicts about treatment

LAYER	RAISES RISK	PROTECTS
History	**A previous attempt, the strongest single predictor**	Engagement with treatment that is continuing
Illness	Mood disorder, substance use, psychosis, personality disorder	Treatment of the disorder itself
State	Hopelessness, agitation, insomnia, recent discharge	Stated reasons for living, responsibility to children
Situation	Access to means, isolation, debt, chronic pain, a humiliating loss	**Restricting access to means, which changes population rates**

In India the leading methods are hanging and pesticide poisoning, rates are highest between fifteen and twenty nine, and the law now treats an attempt as evidence of severe stress rather than as a crime.

- **Neurobiology.** A stress-diathesis model: the diathesis is partly independent of the psychiatric diagnosis. Reduced serotonergic function is the most replicated finding, with low cerebrospinal 5-hydroxyindoleacetic acid in violent attempters and altered receptor binding in ventral prefrontal cortex, the region that restrains action on impulse. Add stress axis dysregulation, reduced neurotrophic signalling, and inflammatory changes through the kynurenine pathway.
- **Immunometabolic depression.** A proposed dimension in which energy-related atypical symptoms, hypersomnia, increased appetite, weight gain and leaden fatigue, cluster with obesity, insulin resistance, dyslipidaemia and low grade inflammation on markers such as C-reactive protein.
- **Why it matters for antidepressants.** This profile predicts poorer response to conventional antidepressants, and several of them, notably mirtazapine, paroxetine and the tricyclics, worsen the metabolic picture further. The relationship runs both ways, so agent choice and metabolic monitoring belong together in these patients.

WHAT EARNS THE MARKS

- **Risk layered into history, illness, state and situation,** with protection answered as its own column.
- **Means restriction named,** since it is the intervention with population-level evidence.
- **The stress-diathesis frame stated before the findings,** so the neurobiology reads as a model rather than a list.
- **The bidirectional relationship in the last part,** which is what the word discuss is asking for.

SEE ALSO Slot II - 52 treatment resistant depression Slot II - 59 course and outcome

AREA 08 OF 14 · P2-A04

Mood disorders: syndromes and nosology

7	7.5%	II-59	II-65
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 6

REVISION ORDER

The three bipolar slots first and in that order, because they build on one another: course and outcome, then the depressive episode, then the spectrum. Recurrent depressive disorder next. Genetics, the seasonal slot and the postpartum slot are independent of all of it.

WHAT IS IN THIS AREA

The course and outcome of bipolar affective disorder, bipolar depression, the bipolar spectrum with bipolar II in detail, the genetics of bipolar disorder with genetic counselling, recurrent depressive disorder, the seasonal affective disorder slot that opens on climate and mental health, and the postpartum slot.

II - 59

Course and outcome of bipolar affective disorder. [10]

10 MOOD DISORDERS: SYNDROMES AND NOSOLOGY

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Onset	Age, the depressive first episode, and the diagnostic delay
3	The pattern over time	Kindling, shortening cycles, predominant polarity
3	Between episodes	Subsyndromal symptoms, cognition, functional recovery
2	Outcome and mortality	Suicide, physical mortality, what predicts a worse course

THE KINDLING MODEL, STATED FIRST

Early episodes follow identifiable stressors; later ones become autonomous, and the interval between them shortens. Whatever its final status as a mechanism, it is the frame that makes early and continuous treatment the logical conclusion of the answer.

FEATURE	WHAT FOLLOW-UP SHOWS	WHAT FOLLOWS FROM IT
Onset	Late teens to mid twenties, usually a depressive episode	Years of delay before the diagnosis is corrected
Episodes	Frequency rises and cycle length shortens untreated	Prophylaxis is judged over years, not months
Between episodes	Subsyndromal symptoms in over half; depression dominates	**Residual symptoms are the strongest relapse predictor**
Rapid cycling	Develops in fifteen to twenty per cent	Review thyroid and any antidepressant exposure

Depressive weeks far outnumber manic weeks across follow-up, which is why an answer organised around mania describes the wrong illness.

- **Functional recovery lags symptomatic recovery.** Fewer than half return to premorbid work and social function even when the episode has resolved, and cognitive impairment in attention, verbal memory and executive function persists into euthymia.
- **Mortality.** Lifetime suicide is of the order of one in twenty, many times the population rate and highest early in the illness and in mixed states. Cardiovascular death accounts for most of the shortened life expectancy.
- **What predicts a worse course.** Early onset, psychotic and mixed features, rapid cycling, comorbid substance use, poor adherence and a higher count of prior episodes.

WHAT EARNS THE MARKS

- **Kindling named, then used** to justify early and continuous treatment.
- **Depression identified as the dominant burden,** which most answers invert.
- **Functional and cognitive outcome separated from symptomatic outcome.**
- **Both mortality routes given,** suicide and cardiovascular disease.

SEE ALSO Slot II - 63 bipolar depression Slot II - 65 bipolar II disorder **SPRINT**

Day 11, Mood disorders: classification, depressive and bipolar syndromes

II - 60

Describe the genetics of bipolar disorder and enumerate principles of genetic counselling. [10]

10 MOOD DISORDERS: SYNDROMES AND NOSOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The size of the effect	Heritability, and which design that number comes from
4	The four designs	Family, twin, adoption, molecular, each with its finding
2	What molecular work found	Polygenic, shared across diagnoses, not yet predictive
2	Counselling	Empirical risk, non-directive stance, guilt, what is modifiable

NAME THE AXIS BEFORE THE LIST

The classification here is by study design, because each design answers a different question: family studies show aggregation, twin studies separate genes from shared environment, adoption studies break the two apart, and molecular work asks which genes.

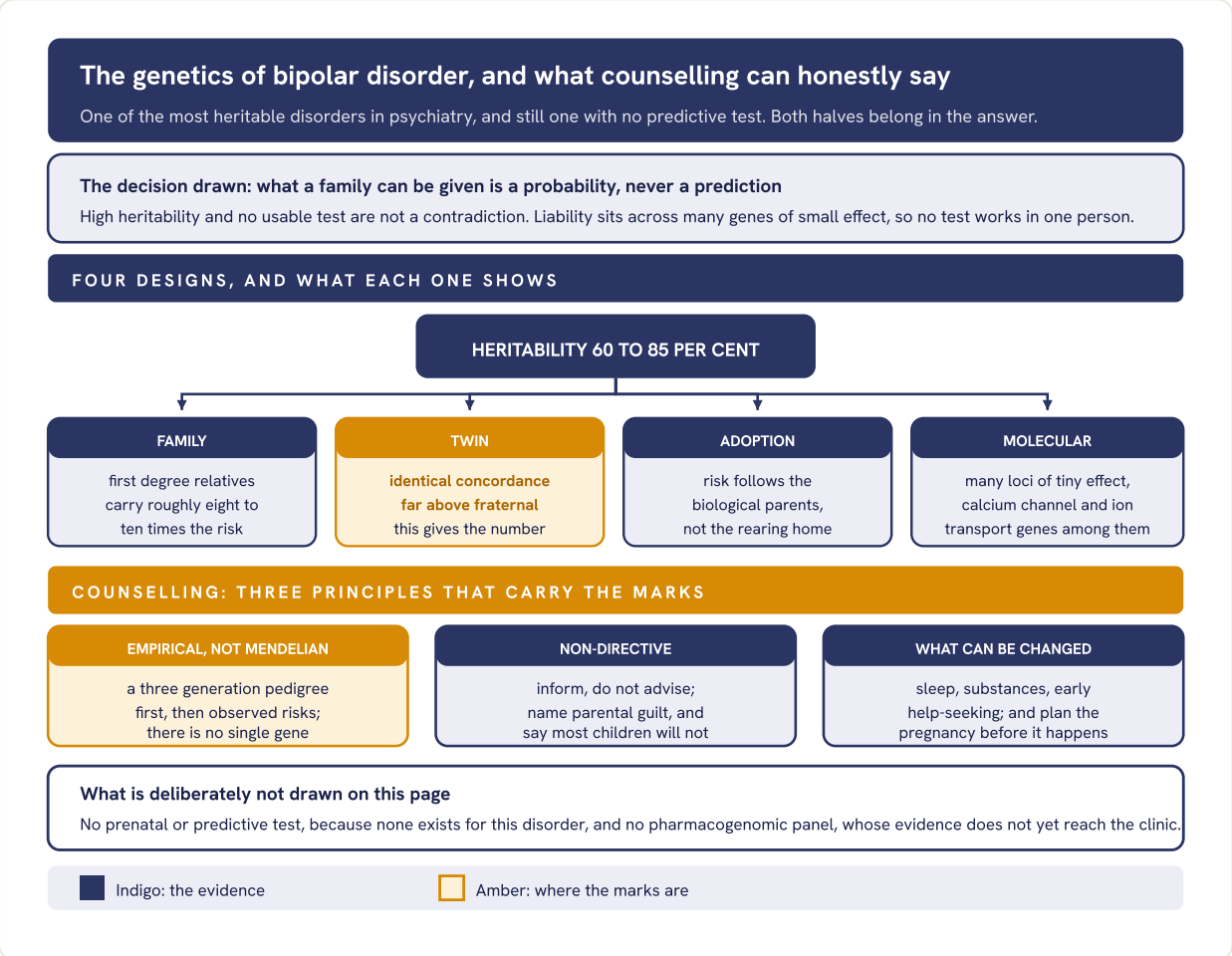


Fig 060.1 The four study designs as one tree, with the counselling principles carried below them because each principle follows from what the design above it can and cannot show.

- **What molecular work actually found.** Liability is polygenic: dozens of loci, each of very small effect, with substantial genetic overlap with schizophrenia and with depression. Polygenic risk scores explain a small fraction of variance and are not clinical instruments.
- **The counselling sentence that matters most.** High heritability is a statement about a population, not about one family, and it never entitles anyone to predict a particular child.

WHAT EARNS THE MARKS

- **The four designs distinguished by what each can show,** not merged into one paragraph on heredity.
- **Polygenicity stated,** with the honest line that no predictive test exists.
- **Counselling given as principles, not as a risk figure,** with guilt and non-directiveness both named.

SEE ALSO Slot II - 59 course and outcome Slot II - 34 endophenotypes

II - 61

Describe the effect of climate on mental health problems. Write the management of a case with seasonal affective disorder. How does light therapy work? [3+4+3]

3 + 4 + 3 | MOOD DISORDERS: SYNDROMES AND NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Climate and mental health**	Heat, disaster, drought, and the drug-specific hazards
2	**The seasonal disorder**	The specifier, the atypical picture, the latitude effect
3	**Management**	Light first, then drugs, then what prevents next winter
2	**How light works**	Retina to hypothalamus, phase advance, melatonin

- **Climate and mental health.** Rising temperature tracks with violence and with suicide rates; disasters bring post-traumatic stress and depression; drought and crop failure drive rural distress and suicide in India.
- **The drug-specific hazards.** Heat with dehydration pushes lithium toward toxicity, and anticholinergic and antipsychotic drugs impair sweating, so heatstroke is likelier in the people we treat.
- **The seasonal specifier.** A pattern, not a separate illness: episodes tied to a season across at least two consecutive years, seasonal clearly outnumbering non-seasonal. The winter type is atypical, with hypersomnia, carbohydrate craving and weight gain, and it rises with latitude.

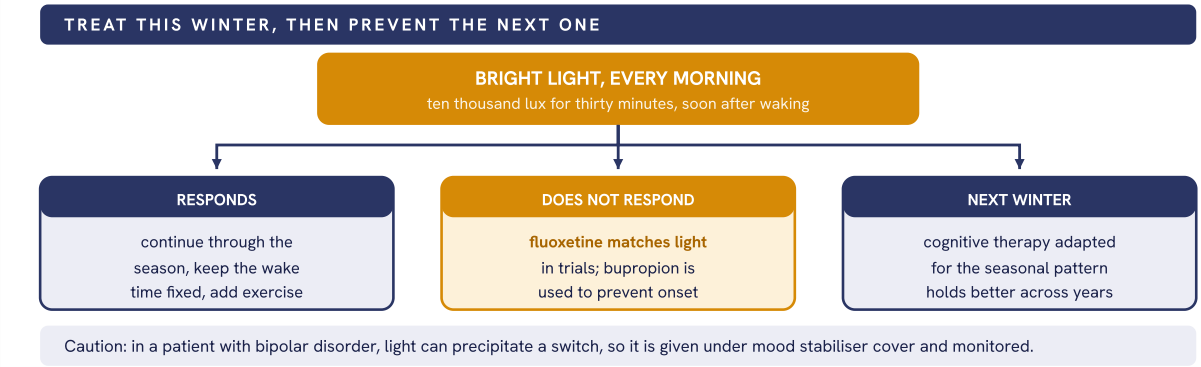


Fig 061.1 Management of the seasonal pattern, with prevention of the following winter treated as part of the same plan.

HOW LIGHT THERAPY WORKS

Light reaches melanopsin-containing retinal ganglion cells, which project through the retinohypothalamic tract to the suprachiasmatic nucleus. Morning light advances a delayed rhythm and suppresses melatonin. Side effects: headache, eye strain, switch.

WHAT EARNS THE MARKS

- **Climate answered as more than heat,** with the lithium and thermoregulation hazards named.
- **The seasonal specifier given with its two-year rule,** and the atypical symptom picture.
- **Light described with a dose, a timing and a mechanism,** which is where the last three marks are.

SEE ALSO Slot II - 62 recurrent depressive disorder Slot II - 63 bipolar depression

II - 62

Discuss the management of recurrent depressive disorder. [10]

10 MOOD DISORDERS: SYNDROMES AND NOSOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **What it commits you to**	Two episodes, no mania, so prophylaxis is the question
3 **Acute phase**	Drug choice, adequate dose and duration, measured response
2 **If it does not respond**	Check, optimise, switch, augment, then ECT
3 **Continuation and after**	How long, at what dose, who needs indefinite treatment

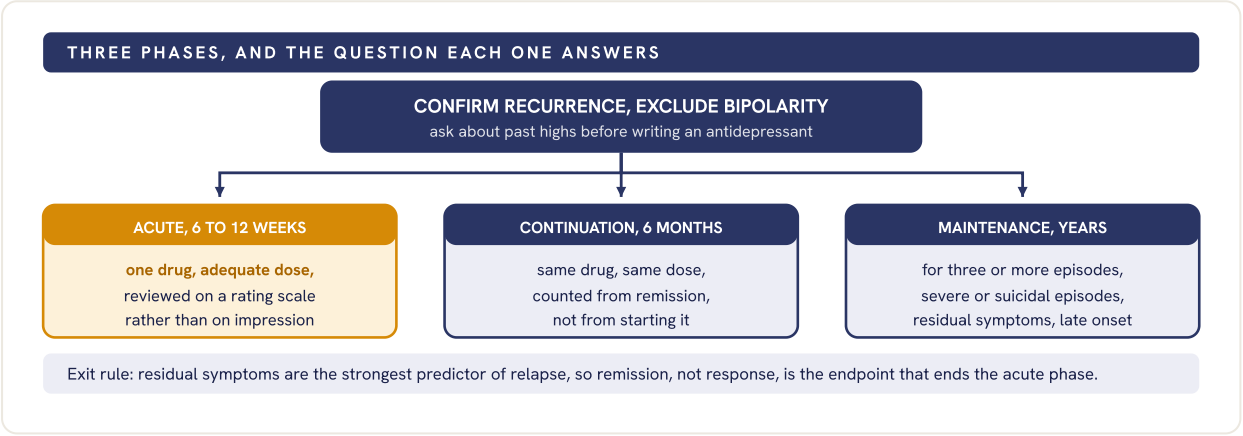


Fig 062.1 The three treatment phases, with the criteria that decide who moves from continuation into indefinite maintenance.

- **If it does not respond.** In order: check adherence and the dose actually taken, review the diagnosis, optimise to the maximum tolerated dose, then switch class or augment with lithium, an atypical or thyroid hormone. ECT for severe, psychotic or suicidal illness.
- **Psychological treatment.** Cognitive behavioural therapy or interpersonal therapy in the acute phase, and mindfulness-based cognitive therapy specifically for relapse prevention in those with three or more episodes.
- **The rest of the plan.** Treat alcohol use, sleep and thyroid disease, address the maintaining stressor, and write a relapse plan the patient keeps.

PITFALL

Stopping the antidepressant when the patient feels better. Continuation is counted from remission and at the dose that produced it; halving it is the commonest way an otherwise correct plan produces a relapse.

WHAT EARNS THE MARKS

- **Three phases named, each with its duration.** This is the structure the question is testing.
- **The maintenance criteria listed,** rather than a vague statement that some patients need long treatment.
- **Remission, not response, given as the endpoint,** with residual symptoms tied to relapse risk.

SEE ALSO Slot II - 52 treatment resistant depression Slot II - 63 bipolar depression

II - 63

Discuss management of a patient with bipolar affective disorder presenting in a depressive episode. [10]

10 MOOD DISORDERS: SYNDROMES AND NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Assess first	Mixed features, rapid cycling, suicide risk, current drugs
4 First line treatment	The agents with evidence, and what each is chosen for
2 The antidepressant	When it may be used, and when it must not be
2 After recovery	Continue what worked, and the anti-suicide argument

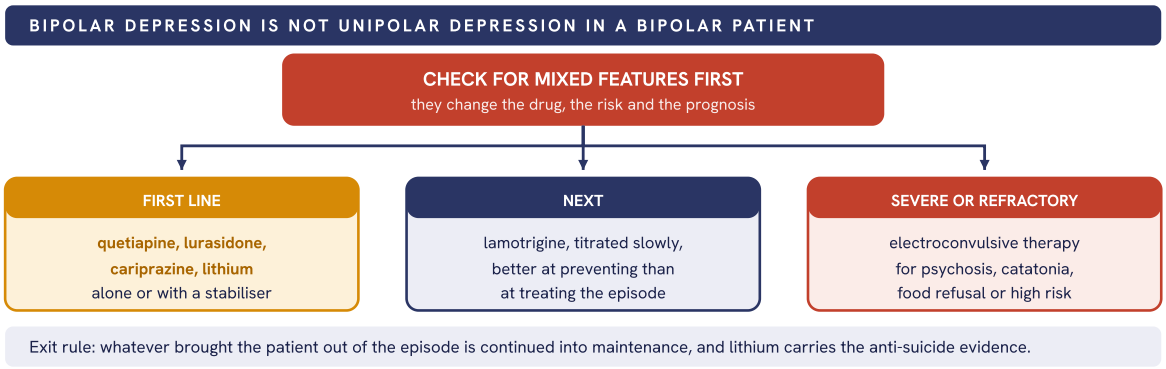


Fig 063.1 The treatment sequence for a depressive episode in bipolar disorder, entered through the mixed-features check because that answer changes everything below it.

- **The antidepressant question.** Monotherapy is not acceptable: efficacy is weak and the risks are a switch and accelerated cycling. If used at all it goes on top of an antimanic agent, and is avoided entirely in mixed features and rapid cycling.
- **Assessment that changes the plan.** Past highs, mixed features now, rapid cycling in the last year, any antidepressant already started, thyroid and renal function before lithium, and suicide risk recorded.
- **Psychological work.** Cognitive behavioural, family-focused and interpersonal and social rhythm therapy all have evidence, and routine and sleep stabilisation starts immediately.

PITFALL

Treating it as unipolar depression because that is what the patient presents with. An antidepressant alone here is the commonest prescribing error in this scenario.

WHAT EARNS THE MARKS

- **The mixed-features check placed before any prescription.**
- **Named first line agents,** rather than "a mood stabiliser" left unspecified.
- **The antidepressant answered explicitly,** including when it must not be used.
- **Continuation into maintenance,** with lithium's anti-suicide evidence stated.

SEE ALSO Slot II - 59 course and outcome Slot II - 65 bipolar II disorder

II - 64

What are the common psychiatric problems seen during the post-partum period? Write in detail about any one of these. What are the prognostic implications of post-partum psychiatric disorders for future mental health? [2+5+3]

2 + 5 + 3 | MOOD DISORDERS: SYNDROMES AND NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The three, separated	Blues, depression, psychosis, by timing and by frequency
3	Psychosis: the picture	Rapid onset, perplexity, and the two risks that define it
2	Psychosis: management	Admit, exclude the medical causes, treat, supervise
3	Prognostic implications	Recurrence, the bipolar link, and the effect on the child

CONDITION	HOW COMMON	ONSET	WHAT IT NEEDS
Postpartum blues	Most mothers	Days three to five	Explanation and support; it resolves
Postpartum depression	About one in ten	Weeks to months	Screening, therapy, an antidepressant if moderate
Postpartum psychosis	One or two per thousand	First two weeks	Emergency admission and supervision of the infant
Anxiety and obsessional states	Common, under-recognised	Any point	Distinguish intrusive thoughts from intent

Onset timing is the single most useful discriminator, which is what the amber cells mark.

POSTPARTUM PSYCHOSIS, IN DETAIL

Onset within days, with perplexity and a fluctuating, almost delirious quality, labile mood and delusions that often concern the baby. An emergency, because of suicide and harm to the infant. Infection, eclampsia and thyroiditis are excluded.

- **Management.** Admit, ideally with the baby where a mother and baby facility exists, and never leave the infant unsupervised until risk has been assessed. An antipsychotic with lithium is the usual combination, and electroconvulsive therapy works quickly and well when the illness is severe.
- **Prognosis for the mother.** Postpartum psychosis is usually the first appearance of a bipolar diathesis. Recurrence after a further delivery runs at roughly one in three to one in two, and more than half go on to have episodes unrelated to childbirth.
- **The child and the next pregnancy.** Untreated maternal depression affects infant cognitive, behavioural and attachment outcomes. Prophylaxis started immediately after the next delivery reduces recurrence.

WHAT EARNS THE MARKS

- **All three separated by timing and frequency** before any one is described.
- **Psychosis named as an emergency,** with both risks and the medical exclusions.
- **The bipolar link stated,** with the child and the next pregnancy included, since the stem asks about future mental health.

SEE ALSO Slot II - 59 course and outcome Slot II - 63 bipolar depression

II - 65

What is meant by bipolar spectrum? Discuss the clinical features, comparative nosology, course, outcome and management of bipolar II disorder. [10]

10 | ASKED AT 15 MARKS | MOOD DISORDERS: SYNDROMES AND NOSOLOGY

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The spectrum idea	What it claims, and the objection it has to answer
4	The grid	Domain by domain against bipolar I, discriminators marked
2	Course and outcome	Depression dominates, and what that costs
2	Management	What is treated, and the antidepressant caution

DOMAIN	BIPOLAR I	BIPOLAR II	WHAT SETTLES IT
The high	Mania, one week or admission	Hypomania, at least four days	Has there ever been a manic episode
Impairment in the high	Marked, work and relationships break	An unmistakable change, function largely held	Was function actually lost
Psychosis in the high	May be present	Never, by definition	Psychosis in a high makes it bipolar I
Depression	Present, but shares the burden	Dominates the course by a wide margin	Depressive weeks outnumber hypomanic ones heavily
How it is found	Usually diagnosed in the manic episode	Usually missed for years	Collateral history for past hypomania

Not a milder illness. It carries comparable suicide risk, more chronic depression and more comorbidity than bipolar I.

- **The spectrum idea.** That bipolarity is dimensional rather than categorical, running from full mania through hypomania and antidepressant-associated highs to hyperthymic temperament. It explains cases that fall between the categories.
- **The objection.** Widening the boundary risks recoding temperament and irritability as illness, and prescribing mood stabilisers to people the trials never studied. A good answer states both the case and the objection.
- **Management.** Quetiapine has the strongest evidence for the depressive phase, with lithium, lamotrigine and lurasidone as alternatives. Antidepressant monotherapy carries switch and cycling risk here too. Add psychoeducation, routine and sleep stabilisation, and continue what worked into maintenance.

WHAT EARNS THE MARKS

- **The grid, with the discriminators identified as discriminators.** A list of ten differences without ranking is a mid-band answer.
- **The spectrum argued from both sides,** because the stem asks for comparative nosology.
- **The explicit statement that it is not the milder illness,** which is the line most answers get wrong.

SEE ALSO Slot II - 59 course and outcome Slot II - 63 bipolar depression

AREA 09 OF 14 · P2-A13

Psychotherapies

7	6.9%	II-66	II-72
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 6

REVISION ORDER

The two cognitive slots together, they are one answer at two lengths. Brief psychodynamic and couple therapy next. The yoga and mobile therapy slots share a critical-appraisal shape and prepare as one. Mutism last, and separately.

WHAT IS IN THIS AREA

Cognitive therapy and the components of cognitive behaviour therapy in two slots, brief psychodynamic therapies, couple therapy with its types and contraindications, the evidence base for yoga in psychiatric practice with the research methodology it raises, and a critique of mobile-based psychotherapies. The seventh slot asks about mutism, its causes and the organic against psychological differential. That is a clinical question the boards set inside this area, and it is answered on its page as it was set.

II - 66

Write a note on cognitive therapy. [10]

10 PSYCHOTHERAPIES KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 The model	Three levels of cognition, and the maintenance cycle
3 Structure	Formulation, agenda, homework, time-limited course
3 Techniques	Cognitive and behavioural, named with what each is for
2 Evidence and limits	Where it works, and where it is not first choice

THE MODEL, IN ONE PARAGRAPH

Beck's proposition is that it is the appraisal of an event, not the event, that produces the emotion, and that appraisal runs at three levels: automatic thoughts at the surface, intermediate beliefs as rules and assumptions, and core beliefs about self, others and the world. Therapy makes the appraisals explicit, tests them against evidence, and changes the behaviour that keeps them untested.

TECHNIQUE	WHAT IT DOES	WHAT IT IS FOR
Thought record	Situation, thought, emotion, evidence, alternative	**Makes automatic thoughts visible**
Socratic questioning	Guided discovery rather than persuasion	The patient reaches the conclusion
Behavioural experiment	A prediction is written down, then tested in life	**The strongest belief-change tool**
Activity scheduling	Graded activity with mastery and pleasure ratings	Depression, early sessions
Downward arrow	Repeated "what would that mean" questioning	Reaches the core belief

Amber marks the two techniques an examiner expects by name and by mechanism. The rest support them.

- **Evidence and limits.** Strong support in depression, anxiety disorders, obsessive-compulsive disorder and post-traumatic stress disorder, and as an adjunct in psychosis and bipolar disorder. It asks for a degree of reflective capacity, and in acute severe illness it follows stabilisation rather than replacing it.

WHAT EARNS THE MARKS

- **The three levels of cognition,** named in order and distinguished from one another.
- **Structure described as structure:** shared formulation, set agenda, homework between sessions, a fixed number of sessions.
- **Techniques tied to their purpose,** which is what the grid does and a bare list does not.
- **A stated limit.** Naming where it is not first choice reads as command of the method.

SEE ALSO Slot II - 71 components of cognitive behaviour therapy Slot II - 70 brief psychodynamic therapies SPRINT Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 67

Give an account of the evidence based integration of yoga into psychiatric practice. What methodological issues arise in research in this field? [5+5]

5 + 5 | PSYCHOTHERAPIES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What is tested	Yoga is a package, so name its components first
3	The evidence	Condition by condition, with the study type
2	Mechanism	What is measured to change biologically
3	Methods	Why these trials are hard to read

- **What is being tested.** Not one intervention but a package: postures, regulated breathing, meditation and an ethical frame. Trials differ in which components they deliver, so two studies of "yoga" may share almost nothing.

CONDITION	WHAT THE EVIDENCE SHOWS	STUDY BASE	STATUS
Depression	Adjunctive benefit; some support as monotherapy in milder illness	Randomised trials, meta-analysed	**Adjunct, reasonable**
Schizophrenia	Add-on gains in negative symptoms and social cognition; little on positive symptoms	Indian randomised add-on trials	**Adjunct, promising**
Anxiety and stress	Reduction in symptom scores, weaker than for depression	Smaller trials, mixed controls	Supportive only
Insomnia	Modest gains in sleep quality	Small trials	Preliminary
Mechanism studies	Raised brain-derived neurotrophic factor, lowered cortisol, higher vagal tone	Biomarker substudies	Hypothesis generating

STATUS: WHY THE TRIALS ARE HARD TO READ

Participants and instructors cannot be blinded, so expectancy is unavoidable. Waitlist controls inflate the effect, and few studies use an active comparator matched for attention and contact time. The intervention is not standardised in content, dose or teacher training. Samples are small, follow up is short, and attrition is high. Allegiance and publication bias run in one direction in this literature. Outcome measures are not always validated in the population studied. The honest position is that yoga is a reasonable adjunct with a real but modest and imprecisely measured effect, not a replacement for established treatment.

WHAT EARNS THE MARKS

- **Naming the components first,** because the heterogeneity argument depends on it.
- **Each condition tied to its study type,** not a general claim that yoga helps.
- **Blinding and the choice of control named as the two central problems.**
- **The status line:** adjunct rather than replacement, stated in one sentence.

SEE ALSO Slot II - 69 mobile based psychotherapies **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 68

What is couple therapy? What are the different types of couple therapy? Write the contraindications of couple therapy. [2+6+2]

2 + 6 + 2 | PSYCHOTHERAPIES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Definition**	The relationship, not a partner, is the patient
2 **The axis**	Classify on what the model changes first
4 **The schools**	Four families, drawn, with the best evidenced named
2 **Contraindications**	Safety first, then agenda, then capacity

- **Definition.** A psychological treatment in which the couple relationship is the unit of intervention and both partners attend together, directed at the pattern between them rather than at one partner's symptoms.

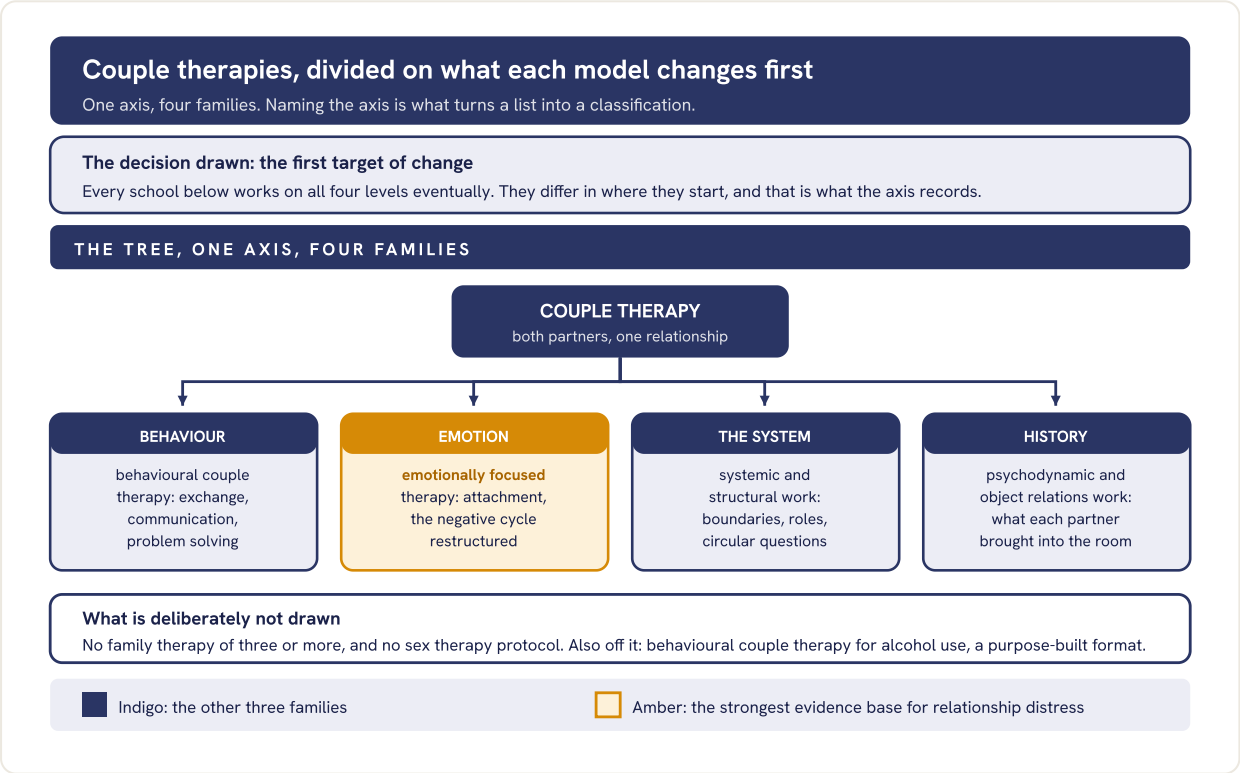


Fig 068.1 One axis, the first target of change, dividing the field into four families, with the emotionally focused branch marked because it carries the largest trial base.

CONTRAINDICATIONS

Ongoing intimate partner violence or coercive control, where joint sessions can raise risk and individual safety work comes first. One partner having privately decided to separate and using the sessions to soften the exit. A secret affair the therapist is asked to hold. Acute psychosis, intoxication or untreated severe illness in either partner. A partner attending under pressure rather than by choice.

WHAT EARNS THE MARKS

- **The axis stated before the list,** since a classification without an axis is only a list.
- **Every branch of the tree reproduced,** with one named model in each.
- **Violence named first among contraindications,** which is the safety mark.

SEE ALSO Slot II - 70 brief psychodynamic therapies **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 69

Write a critique of mobile based psychotherapies in mental disorders. [10]

10 PSYCHOTHERAPIES DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What they are	Guided, unguided, blended, and conversational agents
4	Claim against evidence	Take each promise and test it
2	Risks	Safety, privacy, and who is left out
2	Where it fits	A step in stepped care, not a replacement

- **What they are.** Structured psychological interventions delivered by smartphone: unguided self-help apps, guided programmes with brief clinician contact, blended care alongside face-to-face sessions, and conversational agents.

THE CLAIM	WHAT THE EVIDENCE ACTUALLY SHOWS	THE CRITIQUE
It works	Small to moderate effects in depression and anxiety, larger when guided	**Effects shrink sharply against active controls**
It scales	Reach is real where a therapist workforce does not exist	Language, literacy, shared devices and connectivity exclude many
It is used	Adherence in trials is supported by research contact	**Real-world engagement falls away within weeks**
It is safe	Trials exclude acute risk and severe illness	Most apps have no pathway when risk is disclosed
It is regulated	A minority of store apps carry any trial evidence	Claims are largely unpoliced, and data sharing is common

Amber marks the two lines that carry the critique: the comparator problem and the gap between trial adherence and real use.

PITFALL

Writing a critique that is only a list of benefits with a caveat at the end. A critique takes each claim in turn and says what supports it and what does not. The balanced position is that guided digital therapy has a defensible place at the low-intensity step of stepped care, with a named clinician attached, and no place as a substitute in severe illness or where risk is active.

WHAT EARNS THE MARKS

- **Guided separated from unguided** in the first two lines, since the effect sizes differ.
- **The comparator argument,** that waitlist controls flatter these trials.
- **The engagement gap between trial and real use,** stated as the central practical objection.
- **A placed conclusion,** naming the step of care where it belongs rather than approving or dismissing it.

SEE ALSO Slot II - 67 yoga in psychiatric practice SPRINT

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 70

Discuss brief psychodynamic therapies. [10]

10 PSYCHOTHERAPIES KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	What makes them brief	Focus, time limit, therapist activity, worked ending
4	The schools	Each with its signature move and its length
2	Selection	Who does well, and who should not be offered it
1	Evidence	Where it stands against cognitive therapy

WHAT MAKES A DYNAMIC THERAPY BRIEF

Four features, present in all of them: a single agreed focus rather than the whole personality; a time limit set at the outset and named to the patient; an active therapist who works within Malan's triangle of conflict and triangle of person; and termination treated as therapeutic material from the first session rather than as an administrative ending.

SCHOOL	THE SIGNATURE MOVE	TYPICAL LENGTH
Malan	Brief focal therapy; the two triangles made explicit	**About twenty to thirty sessions**
Sifneos	Anxiety-provoking therapy on an oedipal focus, with a high selection bar	Twelve to twenty sessions
Davanloo	Sustained active challenge to defences early in the work	Up to about forty sessions
Mann	A central issue about separation and time; the limit is itself the treatment	**Exactly twelve sessions**
Luborsky	Supportive-expressive work on the core conflictual relationship theme	Sixteen to twenty sessions

Amber marks the two most quotable pairings. Attaching a length to a name is the cheapest mark in this question.

- **Selection.** Psychological mindedness, capacity to form an alliance quickly, a circumscribed focus, motivation, tolerance of anxiety, and at least one good relationship in the history.
- **Not offered where** there is psychosis, active dependence, poor impulse control, acute risk, or personality pathology that needs a longer frame. Interpersonal psychotherapy grew from this tradition but is not itself psychodynamic.

WHAT EARNS THE MARKS

- **The four shared features named before any school,** which is the structure of the answer.
- **Malan's two triangles by name,** since they are the shared vocabulary of the field.
- **Schools with lengths attached,** read straight off the grid.
- **Selection criteria and exclusions given separately,** not merged into one paragraph.

SEE ALSO Slot II - 66 cognitive therapy Slot II - 68 couple therapy **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 71

What do you understand by the term cognitive behaviour therapy? Discuss in detail its main components. [3+7]

3 + 7 | PSYCHOTHERAPIES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The term	A family of treatments, not one therapy
4 The components	Formulation, behavioural, cognitive, relapse work
2 Session architecture	What every hour looks like, in order
1 The third wave	What was added, and what it changed

THE TERM

Not a single therapy but a family of structured, time-limited, problem-focused treatments sharing one proposition: thought, feeling, body and behaviour maintain one another, so changing any of them changes the rest. What makes a treatment a member of the family is the architecture, a shared formulation, a session structure and homework, rather than any particular technique.

COMPONENT	WHAT IS DONE	WHY IT IS IN THE PACKAGE
Formulation	A maintenance cycle drawn with the patient, plus the longitudinal account	**Everything else is chosen from it**
Psychoeducation	The model explained and the patient socialised to it	Consent and collaboration
Behavioural work	Activation, graded exposure with response prevention, experiments, skills	**Carries much of the effect**
Cognitive work	Thought records, evidence testing, downward arrow to core beliefs	Changes the appraisal
Homework	Agreed tasks between sessions, reviewed at the next	**Dose relates to outcome**
Relapse prevention	A written blueprint: warning signs, what worked, the plan	Protects the gain

Amber marks the three components that carry the marks: the formulation everything hangs on, the behavioural work, and the homework.

- **Every session, in order.** Mood check, agenda set together, homework reviewed, one or two agenda items worked, summary and feedback taken, new homework agreed. Naming this sequence is worth as much as naming a technique.

WHAT EARNS THE MARKS

- **The word family in the definition,** since the question asks what the term means.
- **Behavioural components given real weight,** not treated as an afterthought to the cognitive ones.
- **The session sequence written out,** which few answers do and every supervisor recognises.
- **The third wave named:** acceptance and commitment therapy, mindfulness-based cognitive therapy, dialectical behaviour therapy and metacognitive therapy.

SEE ALSO Slot II - 66 cognitive therapy, the model in depth SPRINT

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 72

What is mutism? What are its causes? How will you differentiate organic from psychological mutism? Discuss the management of mutism. [1+2+4+3]

1 + 2 + 4 + 3 | PSYCHOTHERAPIES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1	Definition	And the four things it is not
2	Causes	Neurological, psychiatric, drug related
4	The differentiation	Domain by domain, with what settles each
3	Management	Support, the challenge test, then the cause

- **Definition and causes.** Absence or marked reduction of speech in a person who has language, with hearing and the speech apparatus intact. It is not aphasia, dysarthria, aphonia or alogia. Neurological causes include akinetic mutism, frontal and supplementary motor lesions, posterior fossa surgery in children, seizures and delirium. Psychiatric causes include catatonia, depressive stupor, dissociative mutism, selective mutism, and autism with intellectual disability.

DOMAIN	ORGANIC	PSYCHOLOGICAL	WHAT SETTLES IT
Onset	With a neurological event or illness	With a psychosocial precipitant	History from an informant
Consciousness	Often clouded or fluctuating	Clear	**Level of awareness**
Focal signs	Gaze palsy, pyramidal or cerebellar signs	Absent	**Neurological examination**
Catatonic signs	Usually absent	Posturing, negativism, waxy flexibility, echophenomena	Bedside catatonia rating
Lorazepam challenge	No change	Catatonic mutism often eases within minutes	**The single most useful test**
Investigations	Imaging or electroencephalogram abnormal	Normal	Imaging and electroencephalogram

Amber cells are the discriminators. The remaining rows are supporting evidence, and saying which is which is itself a mark.

- **Management.** Support first: airway, hydration, nutrition, pressure areas and thromboprophylaxis in stupor. Then the lorazepam challenge and, if it responds, a lorazepam course for catatonia. Electroconvulsive therapy where catatonia does not respond or where there are malignant features, with antipsychotics withheld in that situation. Otherwise treat the cause found: the lesion, the mood disorder, or behavioural and family work with a serotonergic agent in selective mutism.

WHAT EARNS THE MARKS

- **Mutism separated from aphasia, dysarthria and aphonia** in the opening line.
- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **The lorazepam challenge named as the decisive bedside test,** and not buried in management.
- **Withholding antipsychotics in malignant catatonia,** which is the safety line in this answer.

SEE ALSO Slot II - 66 cognitive therapy SPRINT Day 19, Psychotherapies, clinical assessment, MSE and nosology

AREA 10 OF 14 · P2-A10

Personality disorders

7	6.6%	II-73	II-79
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 SINGLE 5

REVISION ORDER

Borderline first and hardest: it carries the highest-scoring question in this book, and the caregiver slot reuses it. Cluster B next, which the borderline answer already half writes. The classification-change slot after that. Organic personality disorder, the avoidant slot and the law slot last.

WHAT IS IN THIS AREA

Borderline personality disorder in two slots, one on clinical features and management and one on caregiver burden, cluster B as a group, the anxious-avoidant presentation, organic personality disorder, the recent changes to the classification of personality and substance use disorders, and the slot on personality disorder at the interface with the law.

II - 73

Discuss clinical features and management of borderline personality disorder. [4+6]

4 + 6 PERSONALITY DISORDERS

DNB, KNRUHS, KUHS, NTRUHS, RGUHS, TN-MGRMU - 9 APPEARANCES, 9 OF 78 SESSIONS

SKELETON

4 **Clinical features**	Instability across four domains, with the criteria
3 **The frame**	What holds treatment before any therapy begins
3 **Therapy, drugs, crisis**	What works, what is targeted, what harms

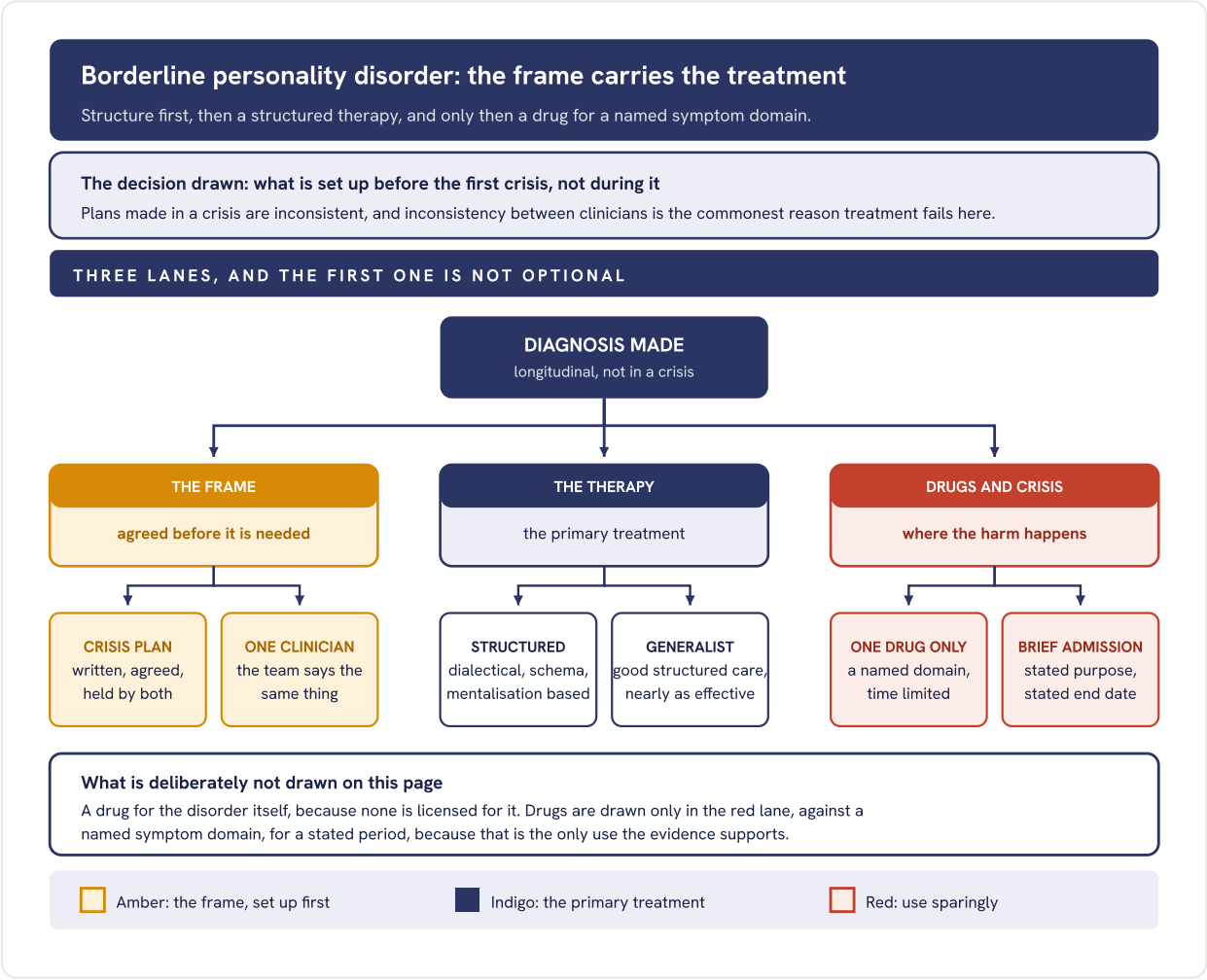


Fig 073.1 The frame drawn as a lane of its own and placed first, because it is what makes the other two safe rather than a preliminary to them.

- **Clinical features.** Instability across four domains: affect, with marked reactivity lasting hours; identity, with chronic emptiness; relationships, alternating idealisation and devaluation with frantic efforts to avoid abandonment; and behaviour, with impulsivity, intense anger, recurrent self harm and transient stress related paranoid or dissociative states.
- **Prognosis, which belongs in the answer.** Long follow up shows most patients no longer meet criteria after some years, with self harm and impulsivity remitting earliest. Social and occupational recovery lags well behind symptom remission, and saying both halves is what makes the statement useful.

WHAT EARNS THE MARKS

- **Features organised into domains,** not recited as nine criteria in order.
- **The frame written before any therapy,** the amber lane, with the crisis plan agreed in advance.
- **Psychotherapy named as the primary treatment,** with drugs confined to a named domain.
- **Prognosis given honestly,** symptom remission ahead of functional recovery.

SEE ALSO Slot II - 75 caregiver burden in borderline disorder Slot II - 76 cluster B personality disorders

II - 74

Discuss the recent changes in diagnostic classification systems related to personality disorders and substance use disorder. [5+5]

5 + 5 | PERSONALITY DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What changed and why	Categories failed on comorbidity and on reliability
3 Personality disorders	Severity and traits in one system, an alternative in the other
3 Substance use	One graded disorder, and a simplified dependence
2 Where it stands	What is actually coded in Indian practice today

AREA	THE CHANGE	WHY IT WAS MADE	STATUS
Personality, international system	**The ten types abolished; severity graded, with five trait domains**	Types overlapped heavily and were unreliable in practice	In force since 2022
Borderline pattern	Kept as a qualifier despite the abolition of types	Clinical and research continuity, and treatment selection	In force, pragmatic
Personality, American system	The ten types retained; a dimensional alternative placed alongside	Evidence supported change, the field was not ready	**Alternative model, for study**
Substance use, American system	Abuse and dependence merged into one disorder, graded by criteria met	The abuse category was unreliable and not a milder stage	In routine use
Substance use, international system	Harmful use kept; dependence simplified to three features	Simplicity and global usability, including non-specialist settings	In force since 2022
Behavioural addictions	Gambling, and then gaming, placed with the substance disorders	Shared course, comorbidity and neurobiology	Gambling routine, gaming newer

Craving was added as a criterion and legal problems removed; a graded severity replaced the two-category split in both fields at once.

STATUS: WHERE THIS SITS IN INDIAN PRACTICE

Indian records and certificates are coded on the international system, so the severity and trait model is the one that will be written down. The American categories remain the language of most teaching and of the trial literature, and the dimensional alternative there is still positioned for further study. Expect to read both, and to code one.

WHAT EARNS THE MARKS

- **The reason for the change, not only the change:** comorbidity, poor reliability, and arbitrary thresholds.
- **The borderline qualifier explained as a deliberate exception,** which shows the system is understood.
- **Merged substance disorder with graded severity,** and craving in, legal problems out.
- **The status strip,** separating what is coded here from what is taught.

SEE ALSO Slot II - 76 cluster B personality disorders Slot II - 90 dependence, abuse and harmful use

II - 75

What is caregiver burden? How to assess and manage caregiver burden of a patient with borderline personality disorder? [2+3+5]

2 + 3 + 5 | PERSONALITY DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	Objective and subjective burden, and expressed emotion
3 Assessment	Who to interview, what to ask, which instruments
3 What is specific here	Why this disorder burdens a family differently
2 Management	Educate, train, involve, relieve, and treat

THE DEFINITION

The physical, psychological, social and financial cost carried by the family of a person with illness. Objective burden is the measurable disruption to income, work, routine and health. Subjective burden is what it feels like: distress, shame, resentment and guilt. The two correlate poorly, so both are asked about.

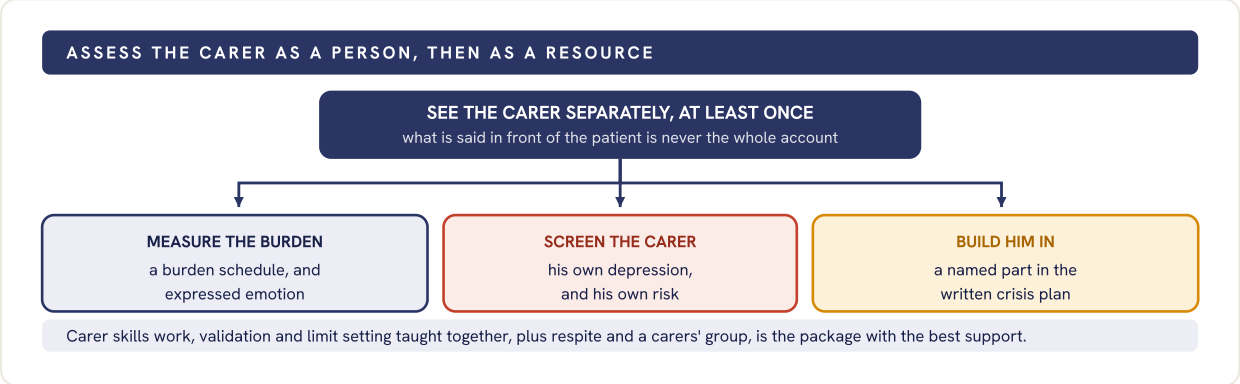


Fig 075.1 Three destinations from one interview, with the carer's own morbidity in red because it is the part most often left unasked.

- **What is specific to this disorder.** Repeated self harm and crisis calls, real fear that he will die, feeling blamed by services, and the pull to rescue that erodes the family's own limits. High expressed emotion is common and worsens the course, and it is usually exhaustion rather than hostility.
- **The guilt about limits.** Families are told to set limits and then feel responsible when distress follows. Say plainly that a limit held consistently is part of treatment, and give them the crisis plan that makes holding it safe.

WHAT EARNS THE MARKS

- **Objective and subjective burden distinguished,** with expressed emotion named alongside.
- **The carer interviewed separately,** the first box, and screened for his own illness.
- **A burden instrument named,** including one developed for Indian families.
- **The carer written into the crisis plan,** which converts assessment into management.

SEE ALSO Slot II - 73 borderline personality disorder Slot II - 77 organic personality disorders **SPRINT** Day 39, Community psychiatry and public health

II - 76

Cluster B personality disorders. [10]

10 PERSONALITY DISORDERS RGUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	The cluster	What the grouping is, and what holds it together
4	The four disorders	Each by its core motive, not by its symptom list
2	Overlap	Why they co-occur, and how they are separated
2	Where it is going	The dimensional reading of the same patients

THE CLUSTER

The dramatic, emotional and erratic group: antisocial, borderline, histrionic and narcissistic personality disorders. They share emotional dysregulation, impulsivity and difficulty sustaining relationships, and they co-occur so often that the cluster is better read as a region than as four separate boxes.

DISORDER	CORE MOTIVE	HOW IT SHOWS	WHAT SETTLES IT
Antisocial	Getting what he wants, others not counted	Deceit, aggression, recklessness, no remorse	**Conduct disorder before fifteen**
Borderline	Not being abandoned	Affective storms, self harm, idealise then devalue	**Self directed harm, abandonment fear**
Histrionic	Being attended to	Theatrical, suggestible, shifting shallow emotion	Distress at not being the centre
Narcissistic	Being admired	Grandiosity, entitlement, envy, poor empathy	**Rage on a blow to self esteem**

The motive column is what separates them under pressure. All four can look impulsive and angry in a crisis; only the reason differs.

- **Overlap and comorbidity.** Criteria sets overlap by design, and most patients meeting one set meet part of another. Substance use, depression, eating disorder and post-traumatic disorder are common, and childhood adversity is over-represented across the whole cluster.
- **Where classification is going.** The current international system does not use clusters at all. The same patients are described by severity plus trait domains, with dissociality and disinhibition carrying most of what this cluster used to name, and a borderline qualifier retained.
- **The caution worth writing.** These labels attract judgement. Antisocial personality disorder is not the same as psychopathy, most people carrying these diagnoses never offend, and all four are treatable.

WHAT EARNS THE MARKS

- **Each disorder given by its motive,** as the second column does, rather than four recited criteria lists.
- **The three amber discriminators,** which is what separates them in an examination vignette.
- **Overlap acknowledged,** since a cluster that never co-occurs would not be a cluster.
- **The dimensional reading,** which shows the classification is current.

SEE ALSO Slot II - 73 borderline personality disorder Slot II - 74 recent changes in classification SPRINT
Day 16, Personality disorders: framework, types and treatment

II - 77

Clinical features and management of organic personality disorders. [10]

10 PERSONALITY DISORDERS KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	A change after brain insult, in a previously different person
3	Clinical features	The six domains, and the syndromes by site
2	Causes and workup	What must be found before it is called a personality
3	Management	Environment first, behaviour second, drugs by target

THE DEFINITION, AND THE WORD THAT CARRIES IT

A persistent change in the habitual pattern of behaviour, following brain disease, damage or dysfunction, in someone whose premorbid personality was different. Change is the operative word: without an informant who knew him before, this diagnosis cannot honestly be made.

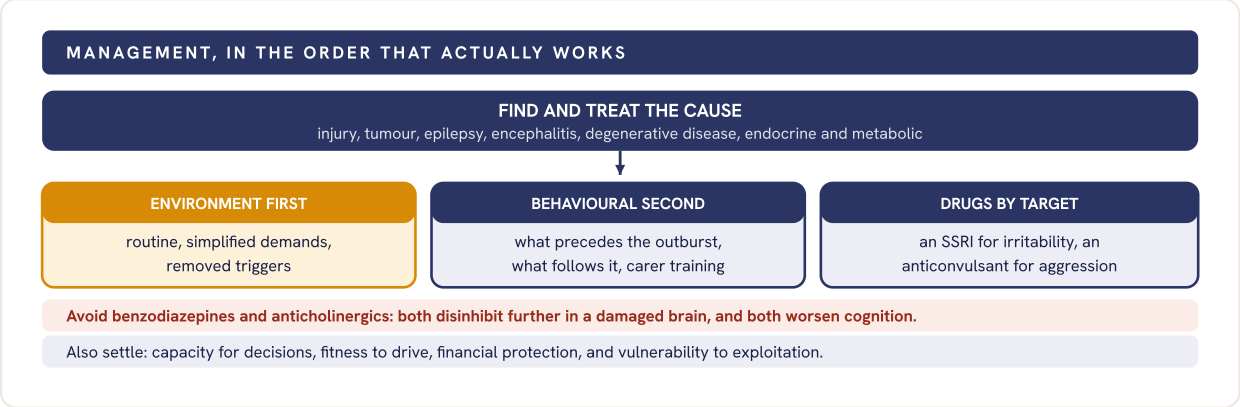


Fig 077.1 Cause, then environment, then behaviour, then drugs, with the two classes that make it worse carried as a red strip.

- **Clinical features.** Reduced persistence with goal directed activity; emotional change with lability, shallow euphoria or apathy; disinhibited expression of impulses; suspiciousness or preoccupation with a single theme; altered rate and flow of speech with circumstantiality and stickiness; and altered sexual behaviour.
- **By site.** Orbitofrontal damage disinhibits, dorsolateral damage produces apathy with executive failure, and medial frontal damage gives loss of drive. Long standing temporal lobe epilepsy is associated with a described pattern of viscosity, hyperreligiosity and circumstantial writing.

WHAT EARNS THE MARKS

- **Change from a known premorbid personality,** with the informant stated as necessary.
- **The features given as domains,** and at least one syndrome tied to its site.
- **Environment and behaviour before drugs,** the order drawn on the figure.
- **The two drug classes to avoid,** and the capacity and driving questions.

SEE ALSO Slot II - 75 caregiver burden Slot II - 76 cluster B personality disorders

II - 78

Personality disorders at the interface of psychiatry and the law: the legal use and the clinical clarification. [5+5]

5 + 5 | PERSONALITY DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Where the question arises	Criminal, civil and mental health law, separately
3	The legal use	What each forum wants the diagnosis to decide
3	The clarification	What the diagnosis can and cannot support
2	The clinician's role	Inform the court; do not answer its question

THE ONE DISTINCTION THE WHOLE ANSWER RESTS ON

A diagnosis is a clinical statement about a pattern of functioning. Responsibility, capacity and dangerousness are legal findings. The diagnosis is evidence towards them and is never equivalent to any of them, and most errors at this interface come from treating the two as the same statement.

FORUM	WHAT IT WANTS DECIDED	WHAT A PERSONALITY DISORDER ACTUALLY SUPPORTS
Criminal responsibility	Did he know the nature of the act, or that it was wrong	**Almost never a defence: reasoning and knowledge are preserved**
Fitness for trial	Can he understand and instruct counsel	Rarely impaired by personality alone
Mental health legislation	Is there a mental illness, and has capacity been lost	**Admission turns on capacity, not on the diagnosis**
Sentencing and risk	How dangerous is he, and for how long	A risk factor within a structured assessment, never the whole judgement
Civil matters	Testamentary capacity, contracts, custody, service fitness	Decided function by function, at a stated time

The statutory definition of mental illness turns on gross impairment of judgement, behaviour or the ability to meet ordinary demands of life, which is a functional test rather than a list of diagnoses.

- **The clinical clarification.** Diagnose longitudinally, never from one interview in custody. Separate the enduring pattern from state effects of intoxication, withdrawal, depression and detention itself. Antisocial personality disorder is not psychopathy, most people carrying these diagnoses never offend, and the disorders are treatable, which is itself relevant to disposal.
- **The ethical hazard.** A diagnosis can be used to extend detention on grounds of risk. The clinician's obligation is to state what the evidence supports and its uncertainty, in the court's language, and to leave the legal finding to the court.

WHAT EARNS THE MARKS

- **Diagnosis separated from the legal finding** in the first two lines.
- **The insanity test quoted in substance,** with the reason personality disorder rarely meets it.
- **Capacity, not diagnosis, named as the trigger** under mental health legislation.
- **The role boundary,** informing the court rather than answering it.

SEE ALSO Slot II - 76 cluster B personality disorders Slot II - 73 borderline personality disorder

II - 79

Write a short note on anxious-avoidant personality disorder. [10]

10 | PERSONALITY DISORDERS | WBUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	Wants closeness, avoids it, for one specific reason
3 Clinical features	The criteria, and how the life is narrowed
3 Differential	Sorted by what is feared and whether closeness is wanted
2 Course and treatment	What helps, and the order it is offered in

THE DEFINITION

A pervasive pattern of social inhibition, feelings of inadequacy and hypersensitivity to criticism, from early adulthood and across situations. He wants relationships and avoids them, restricting work and social life to stay safe from rejection, and will not become involved unless he is sure of being liked.

CONDITION	WHAT IS FEARED	WHAT SETTLES IT
Anxious avoidant personality	Being judged inadequate, then rejected	**Wants closeness, believes he is inferior**
Social anxiety disorder	Scrutiny in specific situations	Later onset, situation bound, less pervasive self view
Schizoid personality	Nothing; closeness is not wanted	**No desire for relationships at all**
Dependent personality	Being left to manage alone	Clings once attached, rather than avoiding attachment
Autism spectrum	Not fear; social communication itself differs	Lifelong, with restricted interests and sensory features

The two amber rows carry the note: wanting closeness separates this from schizoid, and a pervasive belief of inferiority separates it from situational social anxiety.

- **How the life narrows.** Jobs below ability are chosen for their low contact, promotions are declined, friendships are few and slow to form, and criticism is anticipated before it occurs. Depression, social anxiety and alcohol use are the usual comorbidities.
- **Treatment.** Cognitive behavioural work with graded social exposure and skills practice, and schema focused therapy where the inferiority belief is entrenched. A serotonergic drug treats comorbid social anxiety or depression and makes the exposure possible; it does not treat the personality pattern.
- **In the current international system.** Coded as personality disorder with prominent detachment and negative affectivity, graded by severity, rather than as a named type.

WHAT EARNS THE MARKS

- **Wanting contact while avoiding it,** stated in the definition, which is the whole of the disorder.
- **The schizoid comparison,** the second amber cell, which is the commonest examination discriminator.
- **Pervasive and early, against situational,** when separating it from social anxiety disorder.
- **Drugs placed as an enabler of therapy,** not as treatment of the personality.

SEE ALSO Slot II - 76 cluster B personality disorders Slot II - 74 recent changes in classification

AREA 11 OF 14 · P2-A14

Clinical assessment and cross-cutting nosology

6	5.5%	II-80	II-85
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 2 SINGLE 4

REVISION ORDER

EEG first, it is the one most often set. The nosology slot next, and give it time: it is three questions in one. Culture-bound syndromes after that. Functional MRI, functional impairment and the compliance slot are all short.

WHAT IS IN THIS AREA

EEG and functional MRI in psychiatry, the history of nosology with the Indian concepts and the conceptual differences between DSM-5 and ICD-11, the culture-bound syndromes with two Indian ones in detail, functional impairment and the tools that measure it, and treatment compliance set against adherence.

II - 80

Discuss briefly the role of functional magnetic resonance imaging in psychiatry. [10]

10 CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it measures	An indirect signal, and the two designs
4	Disorder findings	The replicated ones, disorder by disorder
2	Clinical uses	Where it touches a patient today
2	Limits	Why it is not a diagnostic test

WHAT IT ACTUALLY MEASURES

The blood oxygen level dependent signal, a haemodynamic proxy for neuronal activity rather than the activity itself. Good spatial resolution, poor temporal resolution. Two designs: task-based, comparing conditions, and resting state, mapping intrinsic networks such as the default mode, salience and central executive networks.

DISORDER	THE FINDING MOST OFTEN REPLICATED	WHAT IT IS TAKEN TO MEAN
Schizophrenia	Inefficient prefrontal recruitment on working memory; salience network disruption	**Dysconnectivity rather than one lesion**
Depression	Amygdala hyperreactivity to negative material with weak prefrontal regulation	A control-and-reactivity imbalance
Obsessive-compulsive	Hyperactivity in the cortico-striato-thalamo-cortical loop	**A circuit target for neurosurgery**
Post-traumatic stress	Amygdala overactivity, reduced ventromedial prefrontal control	Failure of extinction learning
Addiction	Ventral striatal cue reactivity with prefrontal control deficits	Reward learning gone wrong

- **Where it reaches a patient.** Presurgical language and memory mapping; individualised targeting of transcranial magnetic stimulation using functional connectivity; real-time neurofeedback in trials; and target identification for deep brain stimulation.
- **The limits.** Findings are group averages with overlapping distributions, so they do not diagnose an individual. Individual measures are of limited test-retest reliability, motion corrupts data, and cost and access confine it largely to research.

WHAT EARNS THE MARKS

- **Saying the signal is indirect** in the first line, which frames everything after it.
- **Task-based and resting-state separated,** with a named network.
- **Findings tied to what they are taken to mean,** which is what the grid does.
- **The clear statement that it is not a diagnostic test,** which most answers avoid saying outright.

SEE ALSO Slot II - 81 electroencephalography **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 81

Write a note on electroencephalography in psychiatry. [10]

10 CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What it records	Cortical postsynaptic potentials, and the rhythms
4 Patterns that decide	The findings that change the diagnosis
2 Other uses	Drugs, electroconvulsive therapy, sleep, research
2 Limits	What a normal and an abnormal record each fail to prove

WHAT IT RECORDS

Summated postsynaptic potentials of cortical pyramidal neurons, with excellent temporal and poor spatial resolution. Rhythms by frequency: delta below four hertz, theta four to eight, alpha eight to thirteen, beta thirteen to thirty, gamma above that. In psychiatry it is used mainly to answer one question: is this presentation driven by something other than a primary psychiatric disorder.

PATTERN	POINTS TO	WHY IT MATTERS HERE
Diffuse slowing	Delirium, and it tracks with severity	**Separates delirium from a functional psychosis**
Triphasic waves	Metabolic, classically hepatic encephalopathy	A treatable cause found
Periodic sharp wave complexes	Creutzfeldt-Jakob disease	A rapidly progressive dementia explained
Extreme delta brush	Anti-NMDA receptor encephalitis	**Immunotherapy, not antipsychotics**
Ictal or continuous discharges	Non-convulsive status epilepticus	**A psychiatric presentation that is a seizure**
Normal during an event	Dissociative non-epileptic seizures	Video recording is the standard

Amber marks the three patterns that change management the same day they are read.

- **Other uses.** Drug effects: benzodiazepines raise beta, clozapine causes dose-related slowing and spikes, lithium slows the record. Seizure adequacy and post-ictal suppression during electroconvulsive therapy. Polysomnography for sleep disorders. In research, event-related potentials such as the reduced P300 and mismatch negativity in schizophrenia.
- **The limits.** No electroencephalographic pattern establishes a primary psychiatric diagnosis. A normal record does not exclude epilepsy, and minor abnormalities occur in well people. Sleep deprivation and repeat recording raise the yield.

WHAT EARNS THE MARKS

- **The purpose stated up front:** exclusion of a non-psychiatric cause.
- **Named patterns tied to named conditions,** which is what the grid delivers.
- **Non-convulsive status epilepticus mentioned by name,** the highest-value line in the answer.
- **Both limits given,** that a normal record proves little and an abnormal one is not specific.

SEE ALSO Slot II - 80 functional imaging SPRINT Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 82

Describe the history of nosology in psychiatry. What are the Indian concepts in it? Discuss the conceptual differences between DSM-5 and ICD-11. [3+2+5]

3 + 2 + 5 | CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The lineage	Five moments, each with what it settled
2	The Indian strand	Unmada, and what survives in current practice
5	The two systems	Purpose, structure, personality, new categories

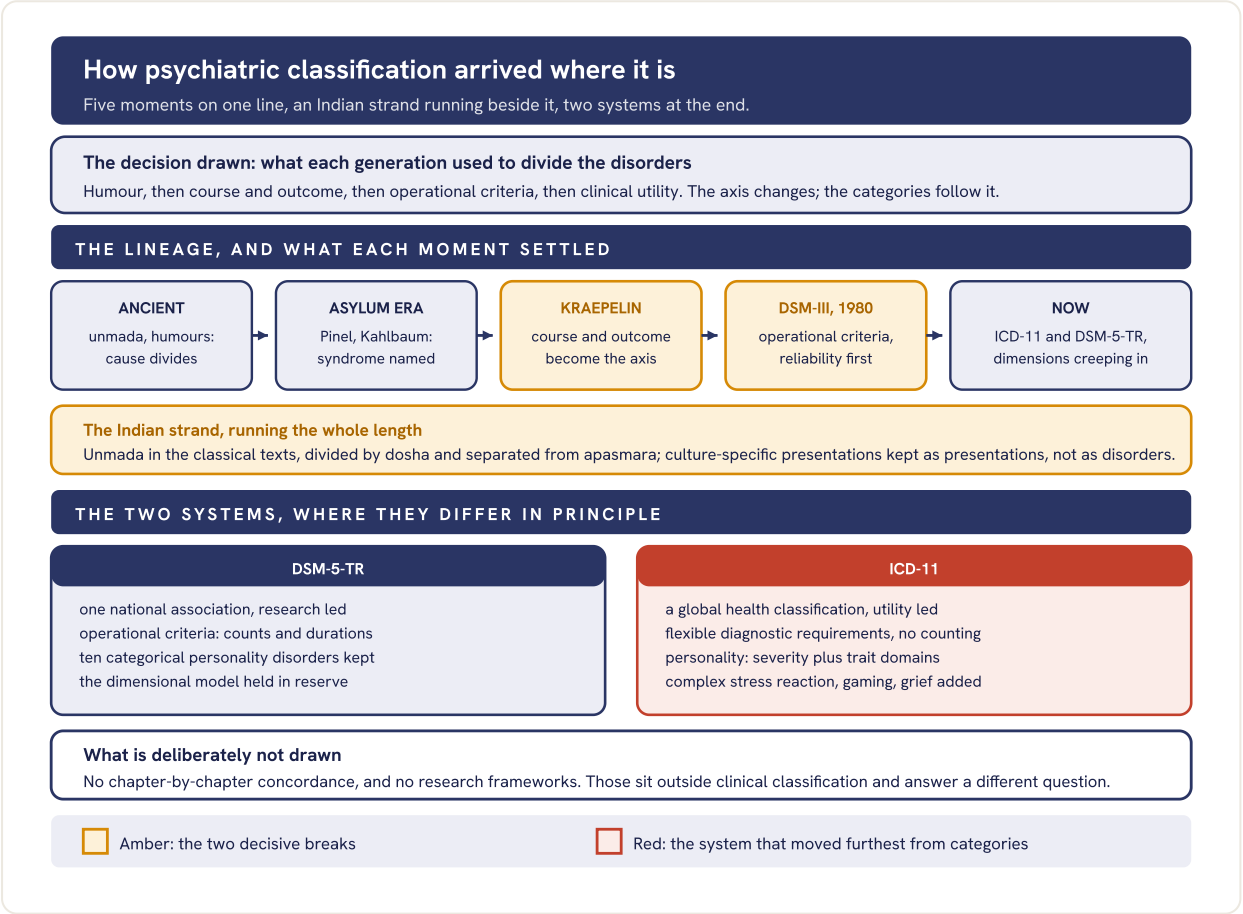


Fig 082.1 One axis, what each generation used to divide the disorders, carrying the field from cause to course to criteria to clinical utility, with the Indian strand shown as continuous rather than as an appendix.

- **The deepest difference.** Purpose. A manual written for research reliability counts symptoms; a classification written for global health service use describes a prototype and trusts the clinician. Almost every structural difference follows from that.

WHAT EARNS THE MARKS

- **The axis named at each stage,** cause, then course, then criteria, then utility.
- **Unmada given with its own internal division,** not mentioned as a passing courtesy.
- **Personality disorder used as the worked example** of the conceptual gap between the systems.

SEE ALSO Slot II - 83 culture bound syndromes **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 83

What are culture bound syndromes? Name those prevalent in India. Describe any two Indian examples in detail. [2+2+6]

2 + 2 + 6 | CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

DNB, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	And the three terms that replaced it
2 The Indian list	Named, with where each is seen
6 Two in detail	Picture, belief, differential, management

THE DEFINITION, AND WHY THE TERM CHANGED

Locality-specific patterns of distress and behaviour, recurrent within a culture, which may or may not map onto a diagnostic category. DSM-5 retired the single term and replaced it with three: the cultural syndrome, the cultural idiom of distress and the cultural explanation of cause. ICD-11 treats them as culturally specific presentations of recognised disorders. The Cultural Formulation Interview is the tool for eliciting them.

PRESENTATION	THE CORE COMPLAINT	WHERE REPORTED
Dhat syndrome	Fatigue and weakness attributed to loss of semen in urine	**Across India, chiefly young men**
Possession states	Identity taken over by a spirit or deity, time limited	**Nationwide, often at shrines**
Koro-like episodes	Fear of genital retraction, sometimes in local epidemics	Eastern and north-eastern India
Suchi-bai	Overwhelming preoccupation with ritual pollution and washing	Eastern India, chiefly widows
Ascetic syndrome	Withdrawal into religious austerity with social renunciation	Young men, described in India

- **Dhat syndrome.** A young man, often unmarried and from a rural background, reports whitish discharge in urine believed to be semen, with weakness, poor concentration, anxiety and sexual dysfunction. The belief rests on the classical idea of semen as the most refined bodily essence. Differential: depressive and anxiety disorders, urinary infection, sexual dysfunction. Management: examine, then explain the physiology, correct the myth, treat the comorbid disorder, and stop the cycle of tonics and repeat tests.
- **Possession states.** Altered identity and consciousness with a changed voice, most often in young women with little social power, usually brief and socially sanctioned. Separate sanctioned trance from dissociative trance disorder, from psychosis and from temporal lobe seizures. Engage the family and the faith healer rather than confronting them, and treat what is treatable.

WHAT EARNS THE MARKS

- **The three replacement terms named,** which shows the current position rather than the older one.
- **A list with locations attached,** not just names.
- **Each detailed example carried through four beats:** picture, belief, differential, management.
- **Collaboration rather than confrontation with traditional healers,** which is the practice mark.

SEE ALSO Slot II - 82 nosology and the Indian strand **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 84

Define functional impairment. What are its manifestations? What scales and assessment tools are used? [2+2+6]

2 + 2 + 6 | CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	The three-level framework, named properly
2 Manifestations	Domain by domain, not a list of symptoms
4 The instruments	Generic, disorder specific, and the Indian statutory one
2 Choosing one	What the answer depends on

THE DEFINITION

Reduction in the ability to carry out activities and fulfil life roles expected for a person's age, sex and social context, arising from a health condition. The international classification of functioning separates three levels: impairment of body function or structure, activity limitation, and participation restriction, each modified by environmental and personal context. Impairment is not the same as disability, and neither equals symptom severity.

INSTRUMENT	WHAT IT MEASURES	WHERE IT IS USED
WHO Disability Assessment Schedule	Six domains: cognition, mobility, self-care, getting along, life activities, participation	**The generic standard, all diagnoses**
Indian Disability Evaluation and Assessment Scale	Self-care, interpersonal activity, communication, work, plus duration of illness	**Statutory certification in India**
Sheehan Disability Scale	Three items: work, social life, family life	Brief, common in trials
Personal and Social Performance Scale	Four areas, clinician rated	Schizophrenia
Functioning Assessment Short Test	Six domains including autonomy and cognition	Bipolar disorder
Global Assessment of Functioning	A single number for symptoms and function together	Withdrawn for conflating the two

Amber marks the two that must appear: the generic instrument the classification recommends, and the one Indian certification actually runs on.

- **Manifestations by domain.** Self-care and household tasks; work or study, through both absence and reduced output while present; family and social roles; leisure and community participation; independent living and finances; and the burden that falls on the carer.

WHAT EARNS THE MARKS

- **The three-level framework named,** since it is what separates impairment from disability.
- **Manifestations given as domains,** which is what a functional assessment actually asks about.
- **The Indian statutory instrument named** and linked to certification under disability law.
- **A reason for withdrawing the global scale,** which shows why function is rated separately from symptoms.

SEE ALSO Slot II - 85 treatment compliance SPRINT Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 85

What is treatment compliance and why does it matter? How does it differ from adherence? Discuss the methods used to improve compliance in psychiatry. [1+1+2+6]

1 + 1 + 2 + 6 | CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The three terms	What each assumes about who decides
2	Why it matters	Relapse, readmission, false treatment resistance
5	Improving it	Patient, treatment, service, each with real measures
1	Measuring it	How you would know whether any of it worked

TERM	WHAT IT DESCRIBES	WHO SETS THE PLAN	WHAT SETTLES THE DIFFERENCE
Compliance	Behaviour matching the prescriber's instruction	The clinician	**The patient is passive in the word itself**
Adherence	Behaviour matching a recommendation the patient agreed to	Both, by agreement	**Autonomy is preserved; the preferred term**
Concordance	The quality of the consultation, not the behaviour after it	A negotiation of beliefs	It describes the meeting, not the pills
Persistence	How long treatment is continued at all	Neither, it is a duration	A different measure, often confused

Amber cells carry the distinction the question is asking for. The fourth row is included because persistence is routinely reported as if it were adherence.

- **Why it matters.** Partial or complete non-adherence is the commonest identifiable cause of relapse in schizophrenia and bipolar disorder, and it drives readmission, suicide risk, and the mislabelling of an untried treatment as treatment resistant.
- **Improving it, at three levels.** Patient: psychoeducation with the family, motivational interviewing, working with the person's own explanatory model. Treatment: simplify to once daily, actively treat side effects, offer a long-acting injectable, monitor plasma levels where available. Service: continuity with one clinician, shared decision making, reminders, assertive follow up, and involving the family who in practice supervise medication at home.

PITFALL

Treating non-adherence as a fault in the patient. It is usually a modifiable problem in the treatment or the service, and side effects are the commonest single reason. Ask about them before assuming anything else.

WHAT EARNS THE MARKS

- **The terms distinguished on who sets the plan,** which is the real difference and not a matter of style.
- **Measures grouped at three levels,** rather than offered as one undifferentiated list.
- **The long-acting injectable named,** with family supervision noted as the Indian reality.
- **A method of measurement,** self-report, pill count, refill records or plasma level, which most answers never supply.

SEE ALSO Slot II - 84 functional impairment **SPRINT** Day 19, Psychotherapies, clinical assessment, MSE and nosology

AREA 12 OF 14 · P2-A06

Substance use: general concepts and alcohol

5	4.5%	II-86	II-90
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 2 SINGLE 3

REVISION ORDER

Delirium tremens first, it is short and it is set often. The neuropsychiatric slot next, it is the longest here. Early onset dependence and relapse prevention after that. The definitional slot last: it rewards precision rather than length.

WHAT IS IN THIS AREA

The neuropsychiatric complications of chronic alcohol use with their treatment and a locality reduction plan, delirium tremens, early onset alcohol dependence syndrome, non-pharmacological relapse prevention, and the definitional slot that separates use, abuse, harmful use and dependence and asks for the role of dopamine.

II - 86

Discuss in detail various neuropsychiatric disorders associated with chronic alcohol use/dependence. Write, in brief, about their treatment. How can you plan to reduce alcohol use in your area? [3+4+3]

3 + 4 + 3 | SUBSTANCE USE: GENERAL CONCEPTS AND ALCOHOL

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The syndromes	Deficiency, toxic and withdrawal groups, each named
4	Treatment	Thiamine before glucose, then the group specific step
3	The area plan	Screen, brief intervention, refer, district programme

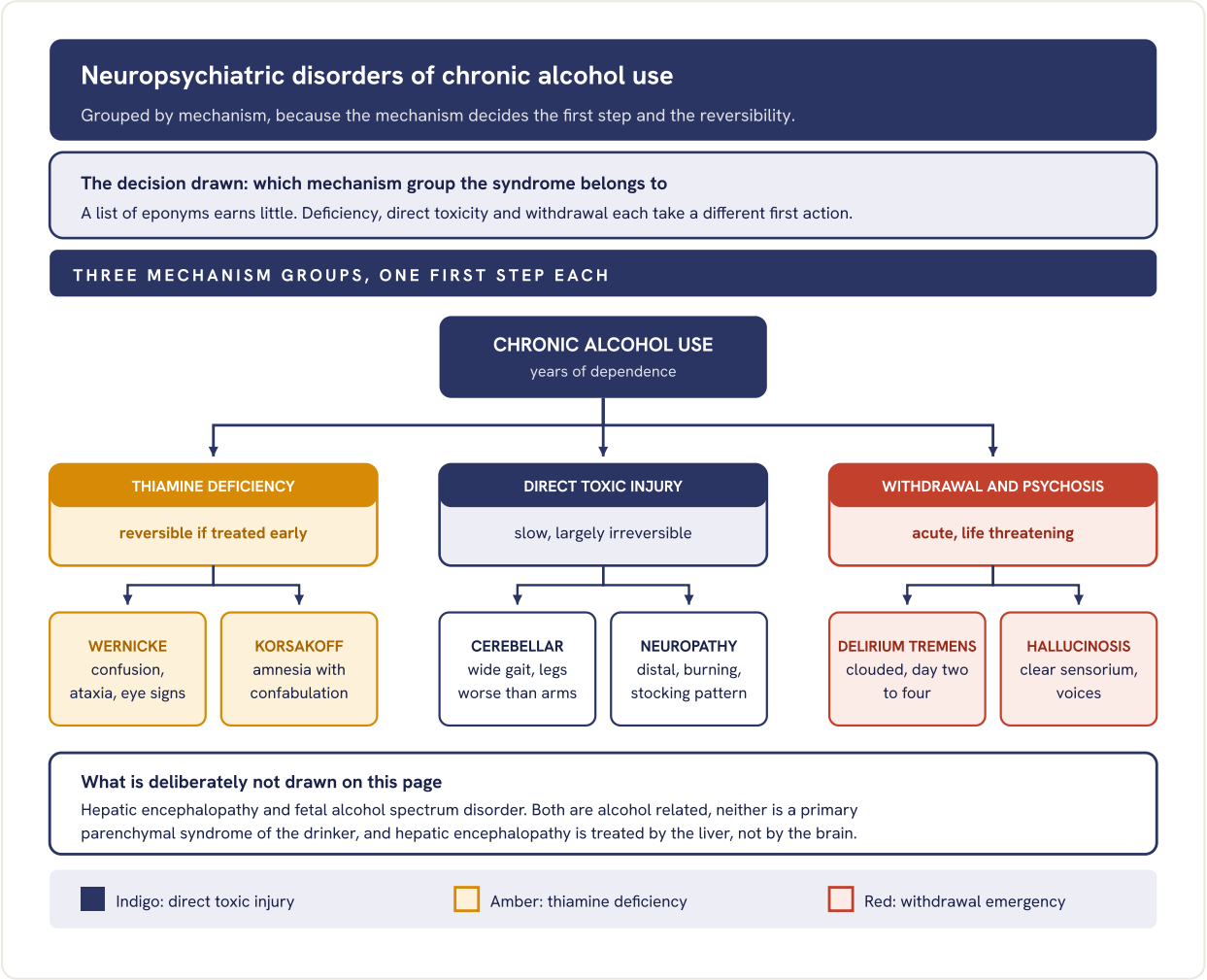


Fig 086.1 The syndromes sorted by mechanism, with the two reversible amber leaves kept together because they share one treatment.

- **Treatment.** Parenteral thiamine before any glucose load; 500 mg intravenously thrice daily for three days in suspected Wernicke encephalopathy, then oral. Benzodiazepine cover for withdrawal. Once dry, naltrexone, acamprosate or disulfiram with supervision.
- **The area plan.** Map the burden, screen every general clinic attender with AUDIT, deliver brief intervention on the spot, refer to the district de-addiction service under the national drug demand reduction plan, and link recovery to mutual help groups.

PITFALL

Korsakoff is not dementia. Selective anterograde amnesia with confabulation and intact procedural learning follows a thiamine emergency; global decline over years is alcohol related dementia, and thiamine will not reverse it.

WHAT EARNS THE MARKS

- **The three mechanism groups on the plate, named as groups.** Grouping is what turns a list of eponyms into an answer.
- **Thiamine written before glucose,** ahead of every other treatment line.
- **Korsakoff separated from alcohol related dementia,** as the pitfall box does.
- **An area plan built on screening and referral,** with a named national programme, not on exhortation.

SEE ALSO Slot II - 87 delirium tremens Slot II - 89 relapse prevention without drugs **SPRINT** Day 13, Substance use disorders

II - 87

Delirium tremens. [10]

10 SUBSTANCE USE: GENERAL CONCEPTS AND ALCOHOL

KUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	The withdrawal delirium, and when in the week it starts
2 Where it sits	The withdrawal timeline, so the diagnosis is dated
3 Clinical picture	Clouding, hallucinations, tremor, autonomic storm
3 Management	Benzodiazepine, thiamine, fluids, treat the precipitant

THE DEFINITION

An acute, transient, fluctuating organic brain syndrome occurring on withdrawal of alcohol in a physically dependent drinker, marked by clouding of consciousness, disorientation, vivid perceptual disturbance, coarse tremor and autonomic overactivity. It is a medical emergency, not a psychiatric one.

HOURS DRY	WHAT APPEARS	CONSCIOUSNESS	WHAT SETTLES IT
6 to 12	Tremor, sweating, nausea, anxiety	Clear	Simple withdrawal
12 to 24	Alcoholic hallucinosis, usually voices	Clear	**Clear sensorium excludes delirium**
24 to 48	Generalised withdrawal seizures	Post-ictal only	Fits precede, they do not define
48 to 96	Delirium tremens	Clouded and fluctuating	**Clouding plus autonomic storm**

Dating the picture from the last drink is the cheapest mark on this question, and the one most often left out.

- **Clinical picture.** Disorientation to time and place, vivid visual hallucinations that are often small animals, tactile misperception, marked tremor, fever, tachycardia, hypertension and profuse sweating, worse at night, with fear and psychomotor overactivity.
- **Management.** Monitored medical bed. Chlordiazepoxide by symptom triggered regimen, or lorazepam 2 to 4 mg when liver function is poor. Parenteral thiamine before glucose. Correct fluids, potassium and magnesium. Add haloperidol only under benzodiazepine cover.
- **Look for the precipitant.** Infection, head injury, pancreatitis, gastrointestinal bleed or hepatic decompensation is present in a large minority, and untreated it is what kills.

WHAT EARNS THE MARKS

- **The 48 to 96 hour window, stated as a number.** The table dates it; prose usually does not.
- **Clouded consciousness named as the discriminator** from alcoholic hallucinosis in the row above it.
- **Benzodiazepine as the treatment and antipsychotic as an adjunct,** never the reverse.
- **The search for a precipitant,** written as part of management rather than as an afterthought.

SEE ALSO Slot II - 86 neuropsychiatric disorders of alcohol Slot II - 88 early onset alcohol dependence SPRINT

Day 13, Substance use disorders

II - 88

Early onset alcohol dependence syndrome. [10]

10 SUBSTANCE USE: GENERAL CONCEPTS AND ALCOHOL

RGUHS, WBUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Definition	Dependence, and what the word early adds to it
3	The typologies	Cloninger and Babor, and the axis each divides on
3	Clinical picture	Temperament, family loading, comorbidity, course
2	Why it matters	What treatment changes because onset was early

THE DEFINITION

Alcohol dependence syndrome by the usual criteria, with onset of dependent drinking before about twenty five years. Age at onset is used as a marker for a distinct subtype rather than as a simple description, because it tracks heritability, temperament and treatment response.

DOMAIN	EARLY ONSET	LATE ONSET	WHAT SETTLES IT
Typology	Cloninger type II, Babor type B	Cloninger type I, Babor type A	Age at first dependence
Temperament	High novelty seeking, low harm avoidance	High harm avoidance, high reward dependence	Novelty seeking is the pivot
Heredity	Male limited, strong paternal loading	Milieu dependent, both sexes	Paternal alcohol use and criminality
Drinking	Spontaneous seeking, inability to abstain	Binges with loss of control, then guilt	Abstain versus control failure
Comorbidity	Antisocial traits, other drugs, violence	Anxiety and depression	Externalising versus internalising
Course	Faster progression, poorer outcome	Slower, better outcome	Prognosis, not severity alone

Amber cells are the discriminators. Age alone is descriptive; temperament and family loading are what make the subtype a claim.

- **Why it matters.** Early onset predicts poorer response to insight oriented work and a better response to structured coping skills training. Ondansetron has trial support in early onset drinkers specifically, while naltrexone suits the later onset, family history positive pattern.
- **The honest limit.** These are dimensional tendencies, not categories. A sizeable proportion of drinkers fit neither type cleanly, and no typology is diagnostic in either classificatory system.

WHAT EARNS THE MARKS

- **Both typologies named,** Cloninger and Babor, with the axis each divides on stated before the grid.
- **Temperament and paternal loading marked as the discriminators,** not age by itself.
- **A treatment consequence,** which is what turns a classification into an answer.
- **The honest limit line.** Naming it reads as command of the literature.

SEE ALSO Slot II - 87 delirium tremens Slot II - 90 dependence, abuse and harmful use SPRINT

Day 13, Substance use disorders

II - 89

Non pharmacological methods in the relapse prevention of alcohol use disorder. [10]

10 | ASKED AT 7 MARKS | SUBSTANCE USE: GENERAL CONCEPTS AND ALCOHOL

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Why non drug**	Relapse is situational; medication does not teach coping
2	**Engage first**	Stage of change, then motivational enhancement
4	**The three lanes**	Individual, family and couple, community
2	**Evidence and aftercare**	What the trials showed, and the follow-up rule

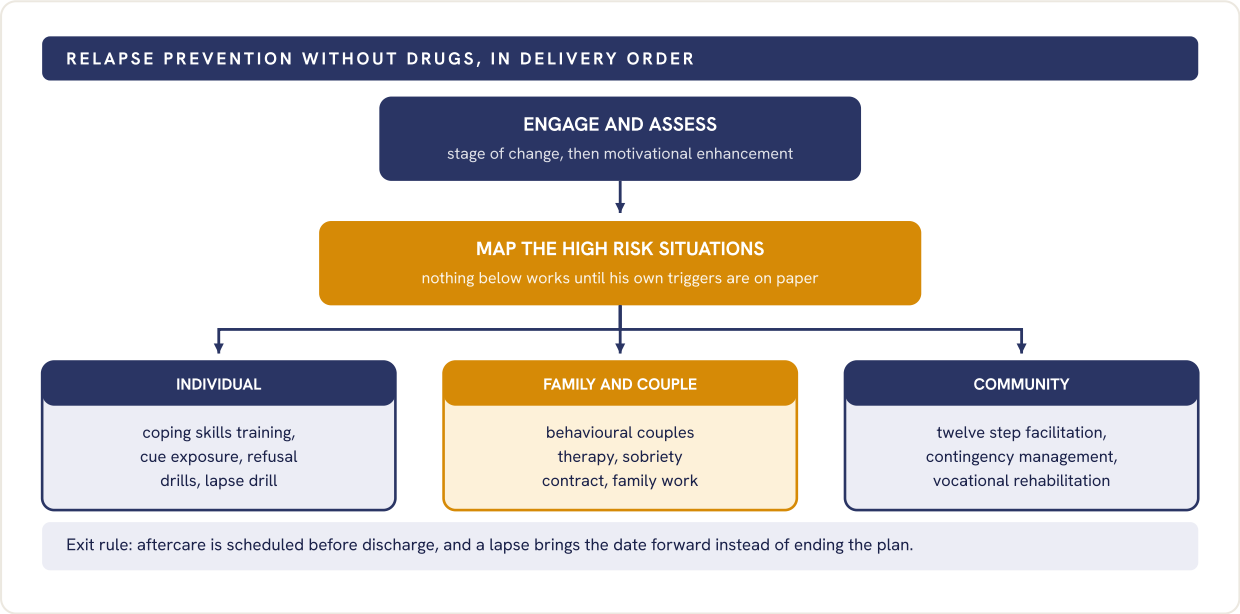


Fig 089.1 Engagement first, the trigger map as the gate, then three lanes that run together rather than in sequence.

- **The cognitive behavioural core.** Identify high risk situations, train coping responses, rehearse drink refusal, correct outcome expectancies, and plan for the seemingly irrelevant decisions that walk a man past his old bar.
- **The abstinence violation effect.** A lapse read as proof of failure becomes a relapse. Reframing it as a high risk situation that was mishandled is itself a technique.
- **Match the method to the stage.** For an ambivalent drinker motivational enhancement is the intervention, not a warm up; coping skills training delivered in precontemplation wastes both.

WHAT EARNS THE MARKS

- **The trigger map placed before the lanes,** as the amber gate on the flowchart.
- **Three lanes kept apart,** individual, family and community, rather than one list of therapy names.
- **The abstinence violation effect, named.** It is the concept the question is testing.
- **Aftercare scheduled before discharge,** the exit rule on the foot of the figure.

SEE ALSO Slot II - 86 neuropsychiatric disorders of alcohol Slot II - 13 motivational enhancement therapy **SPRINT** Day 13, Substance use disorders

II - 90

Define dependence of psychoactive substance. How is it different from use, abuse and harmful use? Discuss the role of dopamine in context of psychoactive substance use. [3+3+4]

3 + 3 + 4 | SUBSTANCE USE: GENERAL CONCEPTS AND ALCOHOL

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The definition	The dependence syndrome, quoted, with its time rule
3	The grid	Use, harmful use and dependence, domain by domain
2	The circuit	Where dopamine sits and what all these drugs share
2	What dopamine does	Wanting, not liking, and the shift to habit

THE DEFINITION

A cluster of physiological, behavioural and cognitive phenomena in which use of a substance takes on a much higher priority than behaviours that once had greater value. Three of six features, together, at some time in the previous twelve months.

DOMAIN	HARMFUL USE	DEPENDENCE	WHAT SETTLES IT
Core question	Has damage occurred	Has control been lost	Loss of control, not quantity
Physiology	Tolerance and withdrawal not required	Tolerance and withdrawal usual	Relief drinking or dosing
Salience	Use is one activity among several	Use displaces work and family	Priority over former interests
Craving	Not part of the definition	Strong desire or compulsion	The reported compulsion
Persistence	May stop once harm is shown	Continues with the harm known	Use despite known harm

Use is consumption without damage or loss of control. Abuse is the older American term and maps closely onto harmful use.

- **The circuit.** Every drug of dependence raises dopamine in the nucleus accumbens, directly as cocaine and amphetamine do, or by disinhibiting ventral tegmental neurons as opioids, alcohol and nicotine do.
- **What dopamine actually does.** It is not the pleasure signal. Phasic dopamine encodes reward prediction error and attaches incentive salience to cues, so wanting grows and outlasts liking.
- **The shift.** With repetition control migrates from ventral to dorsal striatum and use becomes habit. Striatal D2 availability falls, which is why abstinence alone does not restore free choice.

WHAT EARNS THE MARKS

- **The definition written out with the three of six rule** and the twelve month window.
- **Loss of control named as the discriminator,** as the first amber cell does, rather than a list of ten differences.
- **Dopamine described as wanting rather than pleasure,** and the ventral to dorsal shift that links it to relapse.

SEE ALSO Slot II - 88 early onset alcohol dependence Slot II - 11 neurobiology of opioid use disorder **SPRINT** Day 13, Substance use disorders

AREA 13 OF 14 · P2-A03

Other psychoses and catatonia

5	4.3%	II-91	II-95
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 RECURRENT 1 SINGLE 3

REVISION ORDER

The two delusional disorder slots together, because they are the same answer asked at two widths, and the shared-delusion slot sits directly on top of them. Catatonia next, and it is the anchor here. Schizoaffective disorder last, where the nosology is the part that earns the marks.

WHAT IS IN THIS AREA

Catatonia, schizoaffective disorder and its nosological status, delusional disorder in two slots, and shared delusional disorder. Five slots, and four of them are one family.

II - 91

What is catatonia? Briefly discuss etiology, clinical features and management of catatonia. [10]

10 OTHER PSYCHOSES AND CATATONIA

TN-MGRMU, WBUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	What it is	A syndrome across diagnoses, not a subtype of schizophrenia
3	Aetiology	Psychiatric, neurological, medical and drug related causes
3	Clinical features	Three presentations, the signs, and the rating scale
2	Management	Lorazepam, then ECT, and stopping what worsens it

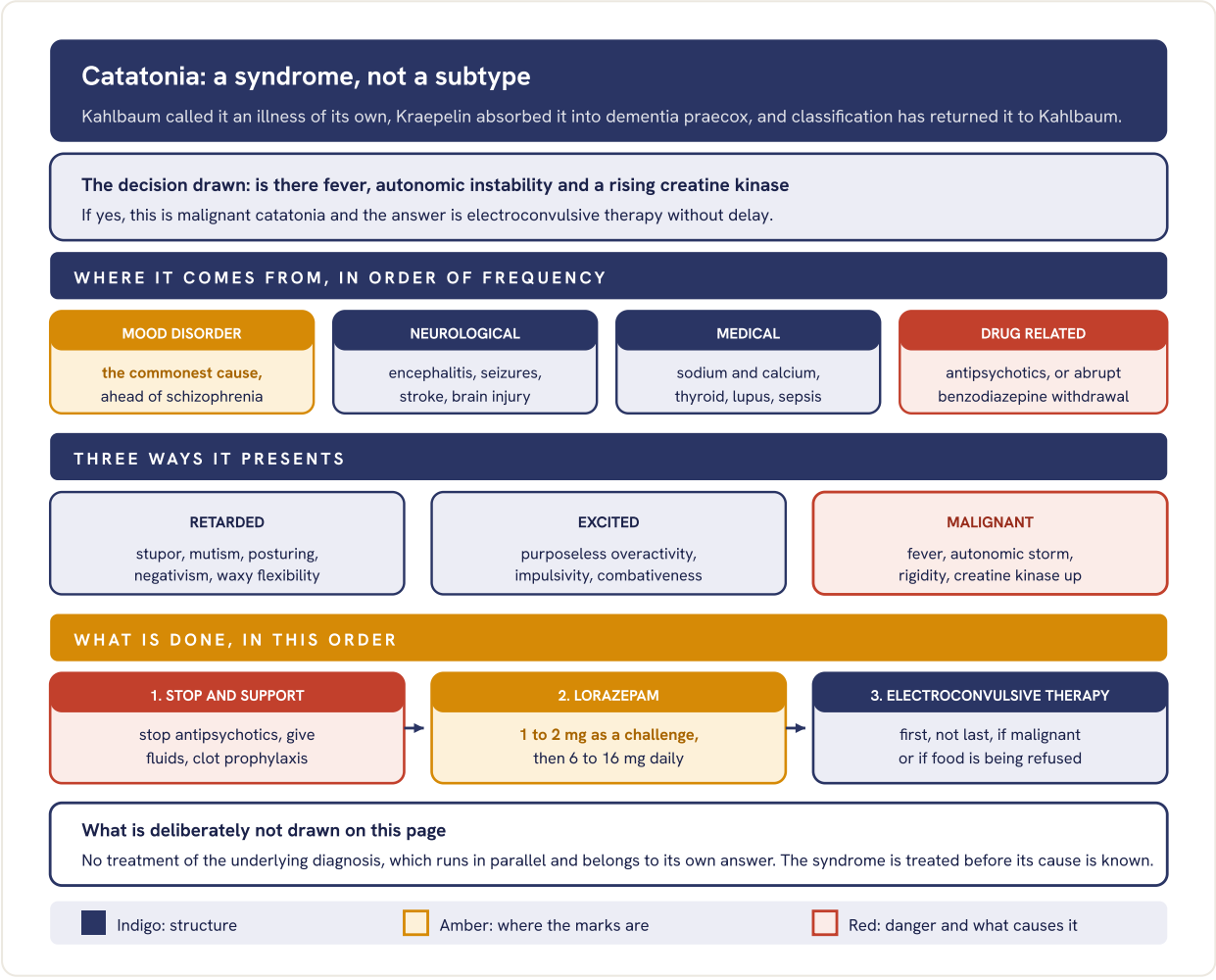


Fig 091.1 Catatonia as cause, presentation and treatment, with the malignant form separated because it changes the order.

- **Recognising it.** Fourteen signs make the bedside screen and twenty three the full scale: stupor, mutism, negativism, posturing, catalepsy, waxy flexibility, staring, grimacing, echolalia, echopraxia, stereotypy, automatic obedience, ambitendency, refusal of food.
- **The challenge test.** One to two milligrams of lorazepam given intravenously or intramuscularly, with the scale rated before and after. Improvement within ten minutes is both diagnostic and the start of treatment, and most non-malignant cases respond.

- **Investigations.** Creatine kinase, electrolytes, calcium, renal and liver function, thyroid, an electroencephalogram to exclude non-convulsive status, and an antibody screen if onset was rapid.

PITFALL

Giving an antipsychotic to a mute, immobile patient assumed to be psychotic. It can convert simple catatonia into the malignant form. The antipsychotic is stopped, not started, until the catatonia has cleared.

WHAT EARNS THE MARKS

- **Catatonia named as a transdiagnostic syndrome,** with mood disorder given as the commonest cause.
- **Causes grouped into four families,** not listed as one run of conditions.
- **The lorazepam challenge described as diagnostic and therapeutic at once.**
- **Electroconvulsive therapy placed first in the malignant form.**

SEE ALSO Slot II - 46 neuroleptic malignant syndrome Slot II - 92 schizoaffective disorder

II - 92

Discuss the nosological status, differential diagnosis, and biological and psychological treatment of schizoaffective disorder. [2+2+3+3]

2 + 2 + 3 + 3 | OTHER PSYCHOSES AND CATATONIA | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Nosological status**	Where it sits, and why the category stays contested
2	**The rules that decide it**	The two week window, and the majority of illness rule
3	**Biological treatment**	Antipsychotic backbone, then the mood agent, then ECT
3	**Psychological treatment**	Psychoeducation, relapse signature, family and work

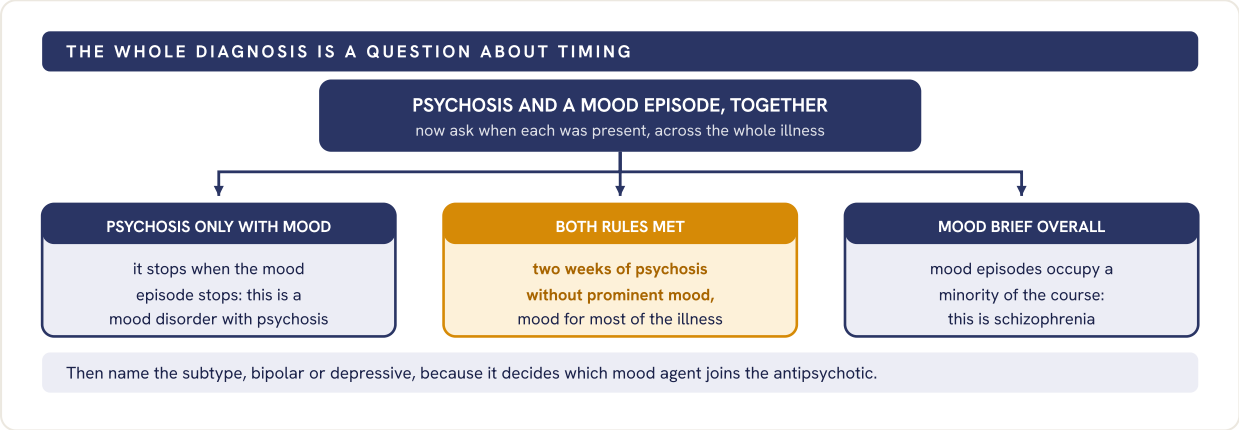


Fig 092.1 The differential drawn as a timing question, because both separating rules are rules about when.

- **Nosological status.** Described by Kasanin, it sits between the halves of the Kraepelinian dichotomy and is standing evidence against a clean split. Reliability and temporal stability are both low.
- **Where the systems differ.** One makes it a lifetime diagnosis, needing the two week window and mood for the majority of the illness. The other keeps it cross-sectional, a label for one episode.
- **Biological treatment.** An antipsychotic is the backbone, paliperidone the one agent with a specific licence. Lithium for the bipolar type, an antidepressant under cover for the depressive type, ECT when severe or refractory.
- **Psychological treatment.** Psychoeducation for a two-syndrome illness, cognitive therapy for psychosis, a written relapse signature, family work and vocational support.

PITFALL

Diagnosing it from one admission. Both rules are longitudinal, so the diagnosis needs a life chart; made cross-sectionally it is usually wrong.

WHAT EARNS THE MARKS

- **Both timing rules stated,** since together they are the whole differential.
- **The disagreement between classification systems named.**
- **Subtype used to select the mood agent,** and psychological treatment answered separately, since it carries its own three marks.

SEE ALSO Slot II - 91 catatonia Slot II - 63 bipolar depression

II - 93

Discuss diagnosis and treatment of delusional disorder. [10]

10 | OTHER PSYCHOSES AND CATATONIA | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Diagnosis**	The criteria, the duration, and what must be absent
2	**Assessment**	Corroboration, is the belief actually false, organic screen
3	**Treatment**	Engagement first, then the drug, then adapted therapy
2	**Risk and course**	Who is dangerous, how long to treat, what relapse looks like

- **Diagnosis.** One or more delusions for at least a month, with functioning outside the theme largely preserved. Hallucinations, if any, are not prominent and stay tied to the theme, and criterion A for schizophrenia is never met.
- **Assessment.** Corroborative history is not optional, because the belief is plausible and jealousy is sometimes true. Late onset demands an organic screen: dementia, stroke, substance use, untreated deafness or blindness.

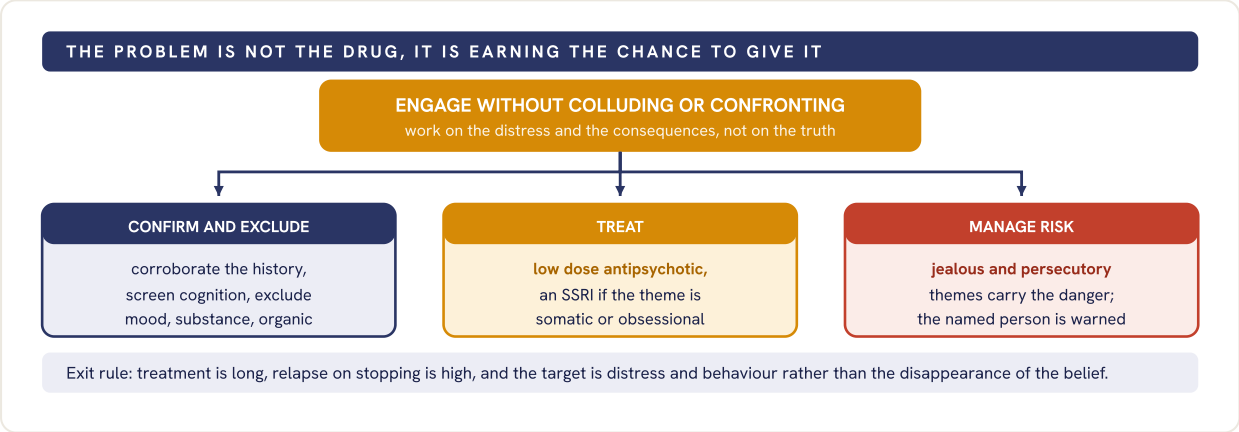


Fig 093.1 Management arranged around the real obstacle, engagement, with the three arms that follow it.

- **Drug treatment, honestly stated.** The evidence is thin, largely open studies, with roughly half improving. Second generation agents at low dose are the practical first choice; pimozide is not superior and carries cardiac risk.
- **Psychological treatment.** Cognitive therapy adapted for psychosis, starting with befriending and moving to reality testing that is offered, never imposed. Direct challenge hardens the belief.

PITFALL

Answering the truth question. Arguing that the belief is false, or agreeing it is true, both end treatment. The distress is real and worth treating either way.

WHAT EARNS THE MARKS

- **Duration and the preserved functioning criterion,** which is what separates this from schizophrenia.
- **Corroborative history and an organic screen in late onset.**
- **Engagement written as the first treatment step,** before any drug, with risk handled as its own task.

SEE ALSO Slot II - 94 the types of delusional disorder Slot II - 95 shared delusional disorder **SPRINT**

Day 10, Other psychotic disorders, delusional syndromes and catatonia

II - 94

Discuss the various types, clinical features, and management of delusional disorders. [10]

10 | ASKED AT 15 MARKS | OTHER PSYCHOSES AND CATATONIA

TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	The types	Five themes, each with the eponym it is known by
3	Clinical features	Encapsulated, systematised, personality preserved around it
3	Management by theme	Risk decides the plan more than the drug choice does
1	Where they present	Rarely to psychiatry, and never by self-referral

TYPE	THE BELIEF	EPONYM AND WHERE IT TURNS UP
Persecutory	Conspired against, followed, poisoned	Commonest; querulous variant reaches the courts
Jealous	The partner is being unfaithful	**Othello syndrome; carries the highest violence risk**
Erotomanic	A person of higher status is secretly in love	De Clerambault syndrome; presents by stalking
Somatic	Infestation, foul odour, a misshapen part	Ekbom syndrome; reaches dermatology first
Grandiose	A special talent, discovery or mission	Exclude mania and an organic cause first

Capgras and Fregoli are misidentification syndromes and usually sit inside another illness rather than here.

- **Clinical features.** The delusion is encapsulated and systematised, elaborated over years, and non-bizarre enough to be plausible. Around it, affect, speech, volition and personality stay intact, which is why work and family life can continue for a long time.
- **The give-away signs.** The specimen brought in a container in delusional infestation, the file of correspondence in the querulous type, and the partner interrogated about timings in the jealous type.

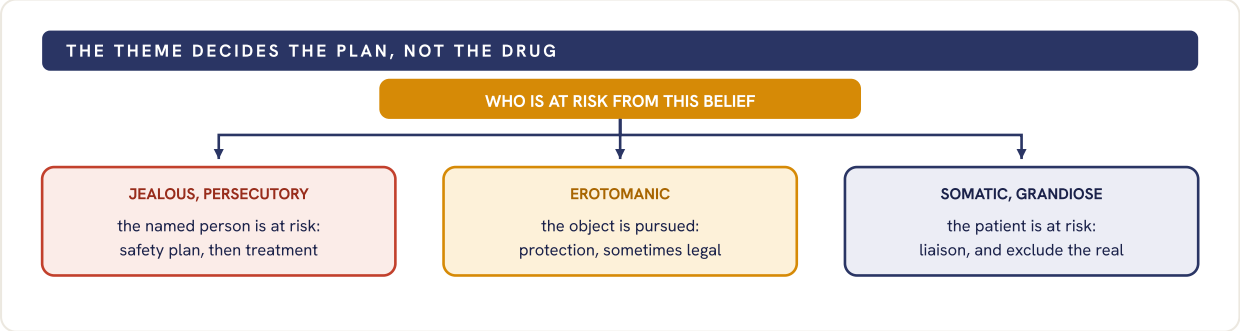


Fig 094.1 Management sorted by who the belief endangers, which is the axis that changes the plan.

WHAT EARNS THE MARKS

- **All five themes with their eponyms.** The stem asks for types first, so the grid is the answer to a third of the paper mark.
- **Encapsulation named as the defining feature,** with function preserved around it.
- **Management differentiated by theme,** rather than one antipsychotic for all five.

SEE ALSO Slot II - 93 diagnosis and treatment Slot II - 95 shared delusional disorder **SPRINT**

Day 10, Other psychotic disorders, delusional syndromes and catatonia

II - 95

Describe the clinical features and management of shared delusional disorder. [10]

10 | OTHER PSYCHOSES AND CATATONIA | KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	The pair, their roles, and where classification puts it now
3 Preconditions and forms	Closeness and isolation, and the four described forms
2 Clinical features	Shared persecutory content, an otherwise well recipient
3 Management	Separate, assess apart, treat the inducer, safeguard

- **What it is.** A delusion held by one person, the inducer, transmitted to a closely attached second person, the recipient. Described as folie a deux by Lasegue and Falret. Neither current system keeps it as a separate category: the recipient is now coded as having a delusional disorder in their own right.
- **Preconditions and forms.** A long, close, usually blood or marital tie, isolation from anyone who would contradict the belief, and a dominant partner who is genuinely ill. Four forms: imposed, where separation cures the recipient; simultaneous, both predisposed; communicated, the belief kept after separation; and induced.
- **Clinical features.** The content is usually persecutory and plausible, so it survives casual scrutiny. The recipient has few other psychotic features and intact premorbid function, so the pair reaches services late, usually through neglect.

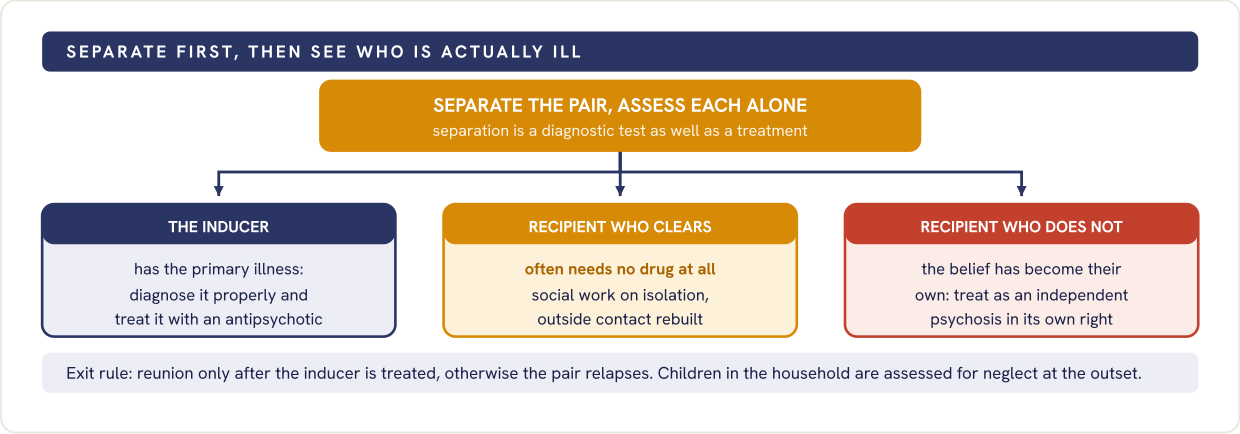


Fig 095.1 Separation used as both test and treatment, with the three outcomes it produces.

PITFALL

Admitting and medicating both. The recipient frequently needs neither, and treating them as equally ill misses that the real intervention is separation, safeguarding of any children, and proper treatment of the inducer.

WHAT EARNS THE MARKS

- **Inducer and recipient named as distinct roles,** with the current classification position said plainly.
- **The four described forms,** since they are what the outcome after separation depends on.
- **Separation written as the first management step,** and as a diagnostic test.
- **Safeguarding of children and the reunion condition,** which most answers never reach.

SEE ALSO Slot II - 93 diagnosis and treatment Slot II - 94 the types of delusional disorder

AREA 14 OF 14 · P2-A12

Suicide, self-harm and impulse control

5	4.0%	II-96	II-100
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 5

REVISION ORDER

Build one suicide answer that covers assessment, biology and prevention, and three of the five slots open. Then the policy limb, which only the last slot asks for. Aggression and behavioural addiction are independent and both are short.

WHAT IS IN THIS AREA

Suicide in three slots: the enquiry with its genetic disposition, the biological aspects with prevention and post-attempt management, and the biopsychosocial slot that asks for the Indian prevention strategy by name. Aggression and behavioural addiction carry the impulse-control limb. Every slot in this area was seen once in the record, which is the whole of what its chips claim.

II - 96

Describe the suicidal enquiry and the diagnostic category to which suicide belongs. Write about genetic disposition in suicide. [4+3+3]

4 + 3 + 3 | SUICIDE, SELF-HARM AND IMPULSE CONTROL

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	The enquiry	Graded questions, collateral, and what gets written down
3	Where it sits	Neither system carries suicide as a mental disorder
3	Genetics	Family, twin, adoption, molecular, endophenotype

THE ENQUIRY, IN THE ORDER IT IS ASKED

Ask unhurriedly and in plain words, moving from the general to the specific: distress and hopelessness, then wish to be dead, then thoughts of ending life, then plan, method, access, preparatory acts, intent and timing, then deterrents and reasons for living. Cover the current episode, the most severe past episode and the most recent one. Take collateral history. Asking directly has not been found to raise risk, and it is what allows the rest of the assessment to happen.

- **Where it sits.** Suicidal behaviour is not a diagnosis in either system. DSM-5-TR carries suicidal behaviour and non-suicidal self-injury as conditions that may be a focus of clinical attention. ICD-11 codes intentional self-harm as an event, not as a mental disorder, so the diagnosis recorded is the underlying condition.

LINE OF EVIDENCE	THE FINDING	WHAT IT SUPPORTS
Family	Aggregation in first-degree relatives beyond the transmitted psychiatric disorder	**Something specific is transmitted**
Twin	Higher concordance in monozygotic than dizygotic pairs	A genetic contribution to attempt
Adoption	Excess in biological, not adoptive, relatives	**Genes rather than shared home**
Molecular	Serotonergic and stress-axis candidates; few loci replicate at genome-wide level	Polygenic, small individual effects
Endophenotype	Impulsive aggression tracks through families	The trait is inherited, not the act

Amber marks the two rows that answer the question actually asked: what is transmitted, and whether it is genetic rather than environmental.

WHAT EARNS THE MARKS

- **A graded enquiry written as a sequence,** not the single question "are you suicidal".
- **Access to means and deterrents both asked,** since these are what the plan is built on.
- **The nosological point stated plainly,** that the recorded diagnosis is the underlying disorder.
- **Impulsive aggression named as the endophenotype,** which is the line that lifts the genetics section.

SEE ALSO Slot II - 98 prevention and post-attempt care Slot II - 100 biopsychosocial aspects **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

II - 97

Define aggression. How would you assess and manage an adult presenting with frequent aggression? [2+4+4]

2 + 4 + 4 | SUICIDE, SELF-HARM AND IMPULSE CONTROL

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Definition**	Aggression, and the impulsive against planned split
4 **Assessment**	Exclude organic, then pattern, then structured risk
3 **Acute management**	Environment, de-escalation, then medication
1 **Longer term**	Treat the syndrome, not the behaviour alone

- **Definition.** Behaviour intended to harm another who is motivated to avoid it. Two forms divide the management: impulsive aggression, with high arousal, a trigger and later remorse; and premeditated aggression, planned, goal directed and with low arousal.
- **Assessment.** Exclude delirium, intoxication and withdrawal, head injury, post-ictal states and dementia first. Then the psychiatric cause, an antecedent-behaviour-consequence analysis, a structured risk tool and collateral history.

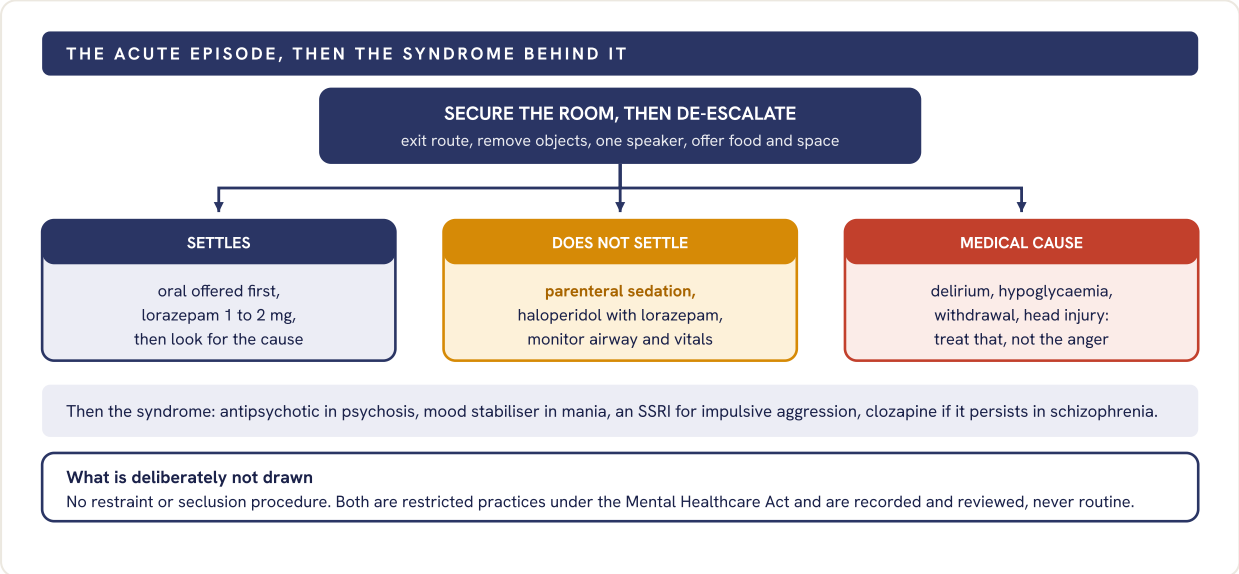


Fig 097.1 The room is made safe before anything is given, and the three lanes are separated by whether the episode settles and whether a medical cause is driving it.

PITFALL

Sedating before excluding a medical cause, which buries the delirium that produced the aggression. The second trap is running chronic aggression on as-required sedation.

WHAT EARNS THE MARKS

- **The impulsive against premeditated split,** stated in the definition because it decides the drug.
- **Organic causes excluded first,** written as a step rather than a caveat at the end.
- **De-escalation before medication,** with the environment described concretely.
- **A named risk tool and collateral history,** which separate assessment from impression.

SEE ALSO Slot II - 99 behavioural addiction **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

II - 98

Describe the biological aspects of suicidal behaviour. Outline the prevention of suicide. Discuss the treatment approach following a self-harm attempt. [3+3+4]

3 + 3 + 4 | SUICIDE, SELF-HARM AND IMPULSE CONTROL

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Biology	Serotonin, stress axis, and what is inherited
3 Prevention	Three tiers, each with its own owner
4 After an attempt	Stabilise, assess, plan, follow up

- **Biology.** Reduced central serotonergic function, with lower cerebrospinal 5-hydroxyindoleacetic acid in violent attempts; altered prefrontal serotonin binding at post-mortem; stress-axis dysregulation; and heritability running through impulsive aggression rather than the act.

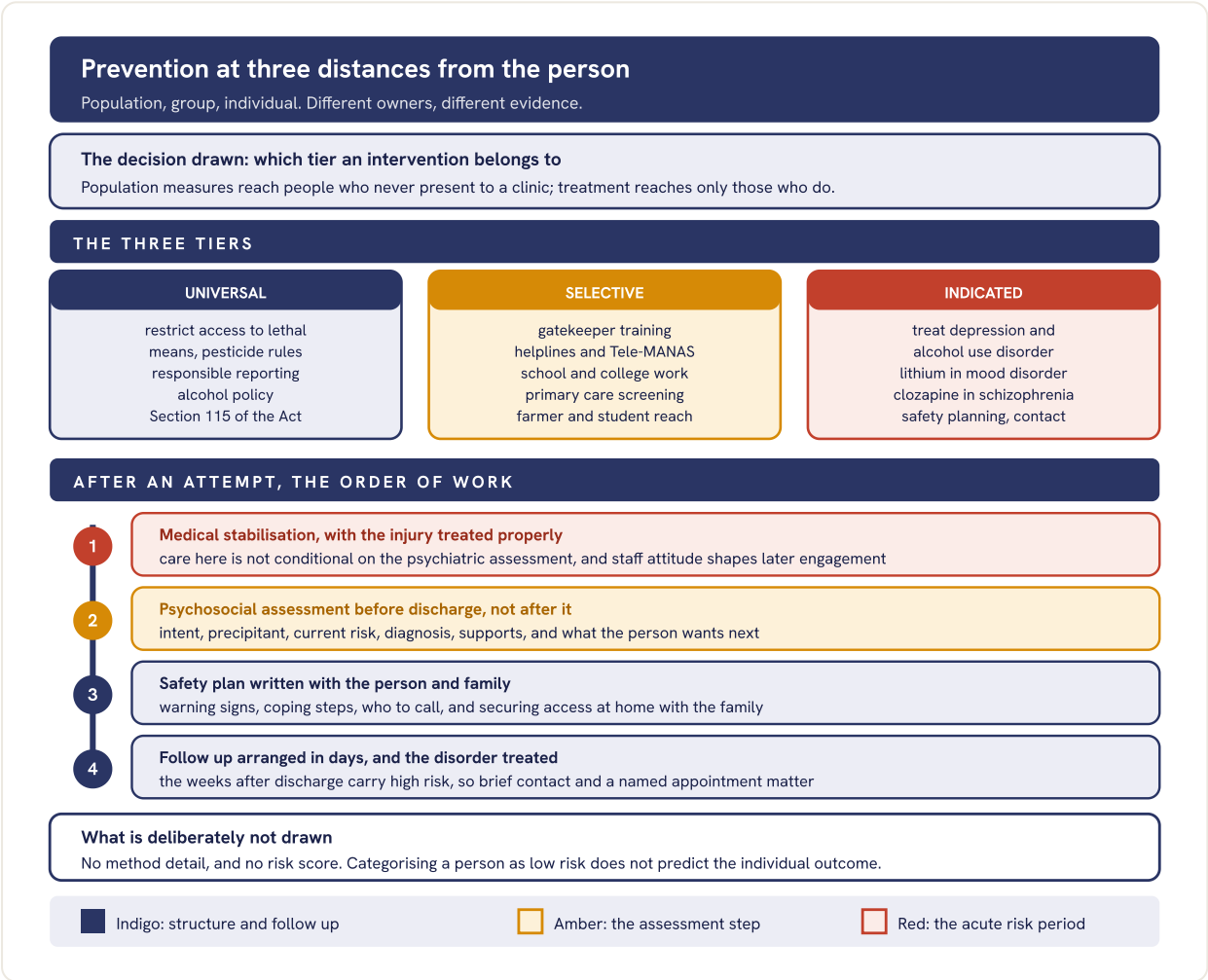


Fig 098.1 Prevention across three tiers, then the post-attempt sequence down a spine, with assessment before discharge, where it is most often lost.

WHAT EARNS THE MARKS

- **Serotonergic and stress-axis findings named,** not "biological factors".
- **Prevention split into three tiers,** with restriction of access at population level where its evidence sits.
- **Assessment placed before discharge,** the most examinable line here.
- **Follow-up named in days,** and the family involved in securing the home.

SEE ALSO Slot II - 96 suicidal enquiry Slot II - 100 the Indian strategy **SPRINT**

Day 18, Sexual, impulse-control disorders and suicide

II - 99

Discuss in detail the epidemiology, aetiology, clinical features and management of behavioural addiction. [10]

10 | ASKED AT 15 MARKS | SUICIDE, SELF-HARM AND IMPULSE CONTROL

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Where it sits	What the systems recognise, and what they still do not
2	Epidemiology	Who, when, and how soft the numbers are
3	Aetiology and features	Reward learning, design, and the clinical picture
3	Management	Screen, psychological work, then the drug question

CONDITION	STATUS IN THE SYSTEMS	REPORTED FREQUENCY	WHAT THE DIAGNOSIS RESTS ON
Gambling disorder	In ICD-11 and DSM-5-TR	Usually under one percent	**Impaired control, priority over life, harm continued, for a year**
Gaming disorder	ICD-11 only; research condition in DSM-5-TR	Estimates vary by instrument	**The same three features, applied to gaming**
Compulsive sexual behaviour	ICD-11: impulse control, not addiction	Not established	Deliberately not an addiction
Shopping, exercise, social media	In neither system	Not established	Described, not diagnosable

Amber marks the only two an examiner can hold you to. Naming what is not yet a diagnosis is itself a mark.

- **Aetiology and features.** Variable-ratio reinforcement, cue reactivity with blunted response to ordinary reward, weak prefrontal control, trait impulsivity, and product design built on near-misses. Clinically: preoccupation, escalation, chasing losses, concealment, irritability on stopping, falling attendance, debt, and comorbid depression, attention deficit and substance use.

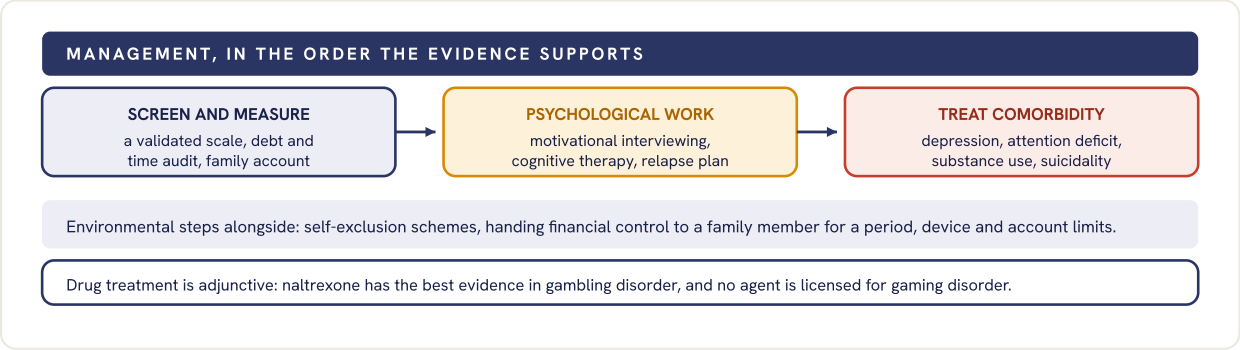


Fig 099.1 Screening, therapy and comorbidity run left to right, with environmental limits and the drug question carried as strips below.

WHAT EARNS THE MARKS

- **The three ICD-11 features named,** which is what makes a behaviour an addiction and not a habit.
- **Honesty about the prevalence figures,** which reads as command rather than evasion.
- **Psychological treatment placed first** and the drug named as adjunctive.
- **Environmental and financial measures,** which most answers omit.

SEE ALSO Slot II - 97 aggression SPRINT Day 18, Sexual, impulse-control disorders and suicide

II - 100

Discuss in detail the biopsychosocial aspects of suicide. Comment on the suicide prevention strategy adopted in India with reference to the National Suicide Prevention Policy. [10]

10 | ASKED AT 15 MARKS | SUICIDE, SELF-HARM AND IMPULSE CONTROL

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The frame	Why one axis alone explains none of it
4 The three domains	Biological, psychological, social, each with a named model
4 India	The statute, the strategy, and what is still missing

DOMAIN	WHAT IT CONTRIBUTES	THE MODEL TO NAME
Biological	Reduced serotonergic function, stress-axis dysregulation, heritable impulsive aggression	The stress-diathesis account
Psychological	Hopelessness, cognitive rigidity, entrapment with no perceived exit	Beck on hopelessness; the cry of pain
Psychiatric	Mood disorder, alcohol use, schizophrenia; most carry a disorder at the time	Psychological autopsy findings
Interpersonal	Thwarted belonging, perceived burdensomeness, acquired capability	The interpersonal theory
Social	Debt, unemployment, agrarian distress, examination pressure, alcohol, media portrayal	Durkheim's four types

Amber marks the two theories examiners expect by name. A model attached to each domain converts a list into an account.

- **The Indian picture.** National crime records show the recorded number rising over the past decade, with daily wage earners, homemakers and students among the largest recorded groups. Access to highly toxic agricultural chemicals is the most modifiable environmental factor.

STATUS: WHERE INDIAN POLICY SITS TODAY

Section 115 of the Mental Healthcare Act 2017 presumes severe stress in a person who attempts suicide, bars prosecution, and places a duty on the government to provide care. The National Suicide Prevention Strategy of 2022 targets a ten percent reduction in suicide mortality by 2030 through three time-bound goals: surveillance within three years, prevention services through district mental health services within five, and a mental well-being curriculum in educational institutions within eight. Tele-MANAS is the tele-counselling arm. Implementation is uneven and dedicated funding is not yet attached.

WHAT EARNS THE MARKS

- **A named model in each domain,** which separates an account from a list of risk factors.
- **Section 115 given by number and by effect,** including the duty to provide care and not only the removal of the offence.
- **The strategy's three time-bound goals,** stated as goals and not as achievements.
- **The closing line on uneven implementation,** which reads as command of the policy.

SEE ALSO Slot II - 96 suicidal enquiry Slot II - 98 prevention tiers SPRINT

Day 39, Community psychiatry and public health

PAPER II APPENDIX, PAGE ONE OF TWO

Ten sets, ten sittings: Paper II

The same hundred Paper II questions, dealt into ten papers of ten. Each cell gives the slot and the page it is answered on.

Read down a row to sit a paper. The deal is not random: the hundred are ranked by score and dealt one question from each rank decile into each set, so every set holds one of the ten strongest, one of the ten weakest, and one from each band between. That is why the sets are comparable to each other and why none of them is an easy paper.

SET	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10
II-S1	II-73 p 235	II-20 p 169	II-92 p 260	II-31 p 182	II-40 p 193	II-83 p 247	II-16 p 163	II-18 p 165	II-71 p 232	II-94 p 262
II-S2	II-28 p 178	II-38 p 191	II-47 p 202	II-12 p 159	II-56 p 213	II-15 p 162	II-26 p 175	II-68 p 228	II-97 p 266	II-85 p 249
II-S3	II-52 p 208	II-91 p 258	II-54 p 211	II-48 p 203	II-86 p 251	II-75 p 238	II-62 p 221	II-17 p 164	II-98 p 267	II-09 p 154
II-S4	II-01 p 145	II-03 p 148	II-96 p 265	II-11 p 158	II-33 p 184	II-14 p 161	II-34 p 185	II-08 p 153	II-72 p 233	II-90 p 256
II-S5	II-29 p 179	II-53 p 210	II-22 p 171	II-10 p 156	II-61 p 220	II-06 p 151	II-35 p 186	II-51 p 206	II-84 p 248	II-89 p 255
II-S6	II-02 p 146	II-30 p 181	II-39 p 192	II-74 p 237	II-67 p 227	II-05 p 150	II-42 p 195	II-93 p 261	II-78 p 241	II-100 p 270
II-S7	II-36 p 188	II-45 p 200	II-46 p 201	II-55 p 212	II-04 p 149	II-58 p 215	II-07 p 152	II-77 p 240	II-87 p 253	II-99 p 269
II-S8	II-44 p 198	II-21 p 170	II-80 p 244	II-66 p 226	II-13 p 160	II-41 p 194	II-43 p 196	II-27 p 176	II-70 p 231	II-65 p 224
II-S9	II-19 p 167	II-60 p 218	II-23 p 172	II-32 p 183	II-25 p 174	II-82 p 246	II-63 p 222	II-76 p 239	II-88 p 254	II-95 p 263
II-S10	II-37 p 190	II-59 p 217	II-24 p 173	II-81 p 245	II-49 p 204	II-57 p 214	II-50 p 205	II-69 p 230	II-64 p 223	II-79 p 242

No set repeats a question, a merged variant of a question, or a near-duplicate, and no set takes more than three questions from any one area.

PAPER II APPENDIX, PAGE TWO OF TWO

How to sit one of these

SET	MARKS	MEAN SCORE	AREAS	ANCHOR OR HIGH	HEAVIEST AREAS IN THE SET
II-S1	100	0.449	8	4	Other psychoses 2; Substance use: opioids 2; Personality disorders 1
II-S2	100	0.427	9	3	Substance use: opioids 2; Schizophrenia: aetiology 1; Trauma 1
II-S3	100	0.428	9	4	Mood disorders: pharmacotherapy 2; Other psychoses 1; Schizophrenia: treatment 1
II-S4	100	0.424	6	1	Eating 3; Substance use: opioids 2; Schizophrenia: aetiology 2
II-S5	100	0.423	9	2	Schizophrenia: aetiology 2; Mood disorders: pharmacotherapy 1; Anxiety 1
II-S6	100	0.415	7	2	Eating 2; Trauma 2; Personality disorders 2
II-S7	100	0.410	7	3	Schizophrenia: treatment 2; Mood disorders: pharmacotherapy 2; Eating 2
II-S8	100	0.409	7	3	Anxiety 2; Psychotherapies 2; Trauma 2
II-S9	100	0.398	7	3	Anxiety 3; Mood disorders: syndromes 2; Schizophrenia: aetiology 1
II-S10	100	0.401	8	3	Mood disorders: syndromes 2; Schizophrenia: treatment 2; Trauma 1

Mean score is the selection score of the ten questions in that set, on the same scale used across all four books. The spread between the strongest and the weakest set is small by construction.

Three hours, ten questions, one hundred marks. That is the shape of the real paper, so it is the shape of these. Eighteen minutes a question leaves you twenty minutes at the end, which is roughly what you will have and roughly what you will need.

Write the skeleton first, for all ten. Spend the first twenty minutes writing nothing but mark splits and section headings across all ten answers. The map on each page shows what that skeleton should look like, so you can mark your own against it before you have written a line of content. Most marks lost in this examination are lost to allocation, not to ignorance.

Then mark yourself against the anchors. Every map ends with three or four things an examiner ticks. Those are the marking scheme, as close as this book gets to one. Count how many you hit before you read the page, because reading it first will convince you that you knew it.

Sit them in order, or sit them backwards. Set one holds the strongest question in the book and set ten the weakest, but the decile deal keeps the mean scores close, so any set is a fair paper. In order simply means the first one you sit is the one most like the paper you will get.

What a set is not. It is not a mock paper from any board, it is not a leak, and it is not a prediction. It is ten questions a board set, arranged so that ten of them look like a paper. Sit five and you will have written a hundred answers in the shape the examination wants, which is the only preparation that has ever reliably worked.

PART THREE OF FOUR

PAPER III

Child, Forensic and Special Populations

Child and adolescent psychiatry, intellectual disability and the learning disorders, criminal and civil forensic work, ethics and consent, emergency psychiatry, and the populations a syllabus calls special.

13	100	78	8	11.3%
AREAS, IN WEIGHT ORDER	ANSWER MAPS	SITTINGS COUNTED	EXAMINING BOARDS	HEAVIEST AREA SHARE

ANCHOR 5 · HIGH 14 · RECURRENT 5 · SINGLE 76

Heaviest area in this paper: Criminal and civil forensic psychiatry. The weightage chapter overleaf shows how every area in Paper III was weighted, and what the weight could not promise.

THE WEIGHTAGE CHAPTER, PAGE ONE OF THREE

Where the marks have gone

Nothing in this book is a prediction. It is a count of 78 sittings from 8 examining boards, and a statement of where those boards have put their marks in Paper III.

Thirteen areas, weighted. The university boards are pooled by how much each is trusted, DNB is held out and given a fixed thirty percent, and the resulting weight becomes a share of one hundred and then a whole number of slots. The ledger of which boards were counted, how their papers reached us, and how far each one is trusted, is on the page opposite.

AREA, IN WEIGHT ORDER	UNIV POOL	DNB	WEIGHT	SHARE	SLOTS
01 Criminal and civil forensic psychiatry	0.239	0.301	0.257	11.34%	10
02 Intellectual disability and learning disorders	0.212	0.333	0.248	10.94%	10
03 Ethics, consent and child protection	0.201	0.343	0.243	10.72%	10
04 Childhood emotional, tic, attachment and elimination disorders	0.172	0.370	0.232	10.20%	10
05 Mental health legislation and disability law	0.181	0.302	0.217	9.58%	9
06 ADHD and disruptive behaviour disorders	0.212	0.223	0.215	9.48%	9
07 Geriatric, consultation-liaison and transcultural psychiatry	0.087	0.452	0.196	8.65%	8
08 ECT and neurostimulation	0.214	0.096	0.178	7.86%	8
09 Emergency psychiatry	0.209	0.000	0.147	6.45%	7
10 Perinatal and women's mental health	0.204	0.000	0.143	6.30%	7
11 Autism spectrum disorder	0.120	0.067	0.104	4.59%	5
12 Child assessment, pharmacotherapy and interventions	0.033	0.162	0.072	3.16%	4
13 Normal child development	0.024	0.000	0.017	0.73%	3

Univ pool is the trust-weighted verdict of the seven university boards; DNB is the national paper, held out. Weight = 0.70 times the pool plus 0.30 times DNB. Share is that weight as a fraction of Paper III. Slots are the share turned into whole questions by largest remainder, with a floor of two and a cap of twenty; Paper III reached neither.

THE WEIGHTAGE CHAPTER, PAGE TWO OF THREE

How the hundred were picked

BOARD	SITTINGS COUNTED	HOW THE PAPERS REACHED US	TRUST
RGUHS	12	Hand transcription and public mirrors of the printed papers	0.806
KUHS	9	Official university portal PDFs	0.600
TN-MGRMU	35	Public archive of the printed papers, exact sitting dates recovered	0.480
KNRUHS	3	Official university portal PDFs	0.300
MUHS	2	Two watermark-confirmed papers, read by OCR. A third-party recurrence compilation was tested against them, matched none, and was removed from the sitting count	0.267
NTRUHS	2	Mirror text of the printed papers	0.160
WBUHS	2	Public archive of the printed papers	0.180
DNB	10	Official NBE material; 32 of 40 files verified by checksum against the board's own published path	fixed 0.30

Trust is how far a board sets the university pool: its standing, its number of sittings, and the quality of its evidence. DNB sits outside the pool. A third-party compilation claiming thirteen further MUHS sittings was tested against the only confirmed MUHS papers inside its own window, matched none of them, and was removed from this ledger; its topic distribution still contributes to area mass at reduced quality.

1 Count the sittings

78 sittings from 8 boards, each weighted so that it halves roughly every six years and is reduced again when the paper reached us as a mirror, an archive or a transcription rather than an official file.

2 Weight the areas

Each board reads each area twice: breadth, how often it examines there, and mark share, how much of its marks land there. The two are averaged, the boards are pooled by trust, and DNB is held out at a fixed thirty percent.

3 Turn weight into slots

The thirteen weights become shares of one hundred, then whole slots by largest remainder, with a floor of two and a cap of twenty so no area disappears and none swallows the book.

4 Score every eligible question

Four components: weighted recurrence (45 per cent), recency (30), how many boards have asked it (15), and how well it is shaped as a ten-mark question (10). Normalised across all four papers, so one score means one thing everywhere.

5 Fill the slots

The highest scorers in each area, up to that area's slot count, near-duplicates merged first. All 100 were filled from questions actually set: 5 anchor, 14 high, 5 recurrent, 76 single-appearance.

THE WEIGHTAGE CHAPTER, PAGE THREE OF THREE

What this book cannot promise

A frequency count is a strong prior, not a prophecy. Here is exactly what that sentence costs, stated before you rely on it.

It does not predict a paper. Nothing here says what your board will set. It says where the marks have gone across 78 sittings, which is a different claim and a smaller one. A board that changes its examiner, its syllabus or its habits can leave every number in this book behind in one sitting.

Most questions were seen once. Of the 224 selectable Paper III questions in the record, 196 appeared in a single sitting. That is not a defect in the counting, it is what happens when eight boards set different papers: the same TOPIC returns constantly while the same QUESTION rarely does. So the selection is driven by AREA weight, where the signal is dense, and each map prints a band rather than a frequency claim about its own question.

The band on a map is a description, not a forecast. Anchor means this question has come back across boards and years. Single means it was set once in the record we hold. A single-appearance question is here because its AREA is heavy, and it is a fair worked example of that area, which is the only thing it is offered as.

Nothing here is invented. All 100 slots in this book are questions a board actually set. Where a paper in this series cannot fill its slots from observed questions, the shortfall is authored to syllabus and labelled as such on the map. Paper III has no such slots.

The record is uneven, and openly so. Two of the eight boards set papers in a legacy structure that does not map onto the modern four, so their sittings are counted against all of their papers rather than one. Two more contribute only two sittings each. The trust column on the first page of this chapter is where that unevenness is priced in, and it is the number to argue with if you want to argue with the weighting.

No board has endorsed this. Board names appear on every page because a count that hides its sources is not a count. None of them was asked, and none of them is responsible for anything in this book.

AREA 01 OF 13 · P3-A08

Criminal and civil forensic psychiatry

10	11.3%	III-01	III-10
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 · HIGH 2 · RECURRENT 1 · SINGLE 6

REVISION ORDER

Start with testamentary capacity and do not leave it until all three of its slots read the same way. McNaughton and the expert-witness slot next, because they share their structure. Ganser and adoption last.

WHAT IS IN THIS AREA

Capacity and the will (four slots), criminal responsibility and the psychiatrist in court, risk of violence, adoption, and Ganser syndrome as the forensic differential. Testamentary capacity alone takes three of the ten, which is the single strongest question-level signal in this paper.

III - 01

Testamentary capacity [10]

10 CRIMINAL AND CIVIL FORENSIC PSYCHIATRY

KNRUHS, KUHS, RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	The test	A sound disposing mind, and where the test comes from
4	The four limbs	Each limb, what it asks, what defeats it
2	What the statute adds	Sound mind, not a minor, and the execution rules
2	The psychiatrist	Contemporaneous, documented, and never the judge

THE DEFINITION

Testamentary capacity is the mental ability to make a valid will: a sound disposing mind at the moment of execution. The classical test is Banks v Goodfellow, 1870, which Indian courts continue to apply alongside Section 59 of the Indian Succession Act 1925.

LIMB	WHAT THE TESTATOR MUST DO	WHAT DEFEATS IT
The act	Understand that he is making a will and what it does	Dementia, delirium, intoxication
The property	Know in general terms what he owns	Not an exact valuation
The claims	Recall those who have a claim on his bounty	Amnesia, severe neglect of family
No perverting disorder	Be free of any delusion that shapes the disposition	A delusion that does not touch the will is no bar

The two amber cells are where most answers overreach: capacity is not perfect knowledge, and mental illness is disqualifying only when it operates on this disposition.

- **The statute and the moment.** Section 59 requires a person of sound mind who is not a minor; execution requires signature and two attesting witnesses. Capacity is judged at the time of execution, so a lucid interval suffices, and a valid will is not undone by later deterioration.

WHAT THE TEST DOES NOT COVER

Undue influence, coercion and fraud are separate grounds that void a will even where capacity is intact. Nor does the psychiatrist decide validity: the court does, and suspicious circumstances shift the burden to the propounder.

WHAT EARNS THE MARKS

- **All four limbs, attributed to Banks v Goodfellow.**
- **Capacity fixed to the moment of execution,** with the lucid interval named.
- **The delusion qualifier.** It must operate on the disposition.
- **Undue influence kept separate from capacity.**

SEE ALSO Slot III - 03 assessing testamentary capacity **SPRINT**

Day 25, Mental health legislation and forensic psychiatry

III - 02

Discuss about the various admission procedures used for mentally ill criminal persons. What are the criteria for Fit to stand trial? [5+5]

5 + 5 | CRIMINAL AND CIVIL FORENSIC PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Who is covered**	Undertrial, convicted, and the acquitted unsound
3 **The routes**	In order, with the actor and the authority at each
3 **Fitness criteria**	The five abilities, from Pritchard onward
2 **What follows unfitness**	Not acquittal: postponement, with review

STEP	WHAT HAPPENS	WHO ACTS	AUTHORITY
1	Unsoundness suspected before or during trial	Magistrate or Court of Session	**Inquiry is mandatory**
2	Examination and opinion on fitness	Civil surgeon or psychiatrist	On the court's reference
3	Trial postponed; bail with security, or safe custody	The court	Custody in a mental health establishment
4	Prisoner found mentally ill while in custody is transferred	Prison medical officer	**A prison mental health establishment per State**
5	Fitness restored, trial resumes; if acquitted as unsound, safe custody with review	Court, then the State Government	Periodic review and release

The procedure sits in the criminal code's chapter on accused persons of unsound mind, carried forward with renumbering into the Bharatiya Nagarik Suraksha Sanhita 2023; the custodial provisions sit in the Mental Healthcare Act 2017.

- **Fit to stand trial.** The accused must be able to understand the charge, follow the course of the proceedings, know that he may challenge a juror or a witness, comprehend the evidence, and instruct his counsel. Fitness is about the present; insanity is about the time of the act.

WHAT THE PROCEDURE DOES NOT COVER

Unfitness is not a verdict and not an acquittal: it postpones the trial, which resumes on recovery. Nor does it decide criminal responsibility, which is judged on the accused's state at the time of the offence.

WHAT EARNS THE MARKS

- **The routes given as a sequence with the deciding authority at each.**
- **All five fitness abilities.** Four out of five is a mid-band answer.
- **Present fitness distinguished from responsibility at the time of the act.**
- **The prison establishment provision,** which most answers omit entirely.

SEE ALSO Slot III - 08 the McNaughton rule **SPRINT** Day 25, Mental health legislation and forensic psychiatry

III - 03

a) What is testamentary capacity? b) How do you assess it? c) What is role of psychiatrist in it? [3+4+3]

3 + 4 + 3 | CRIMINAL AND CIVIL FORENSIC PSYCHIATRY

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3 What it is	A sound disposing mind, in four limbs
4 The assessment	Step by step, each limb put to a question
2 The role	Examiner, recorder, witness, expert
1 The boundary	Opinion on capacity, never on validity

- **What it is.** The mental ability to make a valid will at the moment of execution: to understand the act and its effect, to know the extent of the property, to recall those with a claim, and to be free of any delusion that shapes the disposition. Banks v Goodfellow, 1870.

STEP	WHAT IS DONE	WHO ACTS	CONDITION
1	Clarify who instructed the assessment and tell the testator its purpose	Psychiatrist	**Consent, and no confidentiality assumed**
2	History, medication, sensory state, and collateral from the solicitor	Psychiatrist with family	Reversible causes corrected first
3	Examine each limb by open question, in the testator's own words	Psychiatrist alone with him	Beneficiaries out of the room
4	Cognitive testing, and specific enquiry into any delusion about the heirs	Psychiatrist	Screening test is supportive only
5	Record verbatim answers, ideally contemporaneous with execution	Psychiatrist	**The record is the evidence**

Where the will is already executed and the testator has died, the same limbs are reconstructed from records, witnesses and the solicitor's attendance notes.

- **The role.** To examine and give an opinion; to record it in a form that survives cross-examination; to act as attesting witness or as expert witness, but not both in the same matter without disclosure.

WHAT THE PSYCHIATRIST DOES NOT DO

He does not pronounce the will valid, does not assess undue influence as a legal conclusion, and does not infer incapacity from a diagnosis or from an unfair disposition. An eccentric will made with capacity is a valid will.

WHAT EARNS THE MARKS

- **The four limbs, then an assessment mapped onto them.**
- **Examination alone, beneficiaries excluded,** and verbatim recording.
- **Contemporaneous documentation,** named as the evidence it becomes.
- **The limit of the role,** capacity and not validity.

SEE ALSO Slot III - 10 reporting on capacity to make a will **SPRINT**

Day 25, Mental health legislation and forensic psychiatry

III - 04

Ganser syndrome. [10]

10 CRIMINAL AND CIVIL FORENSIC PSYCHIATRY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	Named for Ganser, 1898, described in prisoners
3	The four features	Approximate answers first, then the other three
3	Differential	Four rivals, each separated on one finding
2	Course and forensic weight	Abrupt recovery, amnesia, and fitness to plead

THE DEFINITION

A dissociative disorder whose cardinal feature is the approximate answer: a reply that is plainly wrong yet demonstrates that the question was understood. Described by Ganser in 1898 in prisoners awaiting trial, and classified as a dissociative rather than a factitious condition.

CONFUSED WITH	WHAT IT LOOKS LIKE	WHAT SEPARATES IT
Malingering	Deliberate false symptoms	**Conscious, goal directed, and improves unobserved**
Factitious disorder	Symptoms produced for the sick role	Motive is care, not release
Pseudodementia	Do not know replies in depression	Mood precedes, and answers are omissions
Delirium	Clouding, disorientation, fluctuation	An organic cause, and no approximate answers

The amber cell is the distinction the examiner is looking for, and it is also the one that cannot be made with certainty at a single interview.

- **The four features.** Approximate answers, the German vorbeireden, talking past the point: asked how many legs a horse has, the patient says five. With them, clouding of consciousness; somatic conversion features; and hallucinations, usually visual and vivid.
- **Where it sits.** ICD places it among the dissociative disorders and DSM among the other specified dissociative disorders, so it is understood as unconsciously produced. That classification is itself the answer to the malingering question, and it is contested.
- **Course and forensic weight.** Abrupt onset in custody or under any severe stress, abrupt recovery with amnesia for the episode, and a real bearing on fitness to plead, which is why it is assessed over time and never on one meeting.

WHAT EARNS THE MARKS

- **The approximate answer defined precisely,** wrong but showing the question was understood.
- **All four features, in order.**
- **Separated from malingering on consciousness and motive,** not asserted.
- **The eponym and the custodial setting,** which is where the syndrome was found.

SEE ALSO Slot III - 02 fitness to stand trial SPRINT Day 15, Dissociative and conversion disorders

III - 05

Write in detail about the Forensic aspects of sexual offenses and its psychiatric manifestation. [10]

10 | ASKED AT 15 MARKS | CRIMINAL AND CIVIL FORENSIC PSYCHIATRY

TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 **The legal frame**	The penal provisions, and what 2013 changed
3 **The examination**	Steps, consent, timing, and one banned practice
3 **In the survivor**	Acute reaction, then the disorders that follow
2 **In the accused**	What is assessed, and what it does not excuse

STEP	WHAT HAPPENS	WHO ACTS	CONDITION
1	Informed consent for examination, treatment and evidence collection, taken separately	Survivor, or guardian if a child	**Refusal of any part is permitted**
2	Free first aid and treatment, which no hospital may refuse	Any hospital, public or private	Refusal is itself an offence
3	Medical examination and forensic sampling	Registered medical practitioner	As soon as possible, ideally within twenty four hours
4	Report recording findings and the survivor's account, without opinion on consent	Examining doctor	**The per vaginum two-finger test is prohibited**
5	Psychiatric assessment, support and follow-up	Psychiatrist and counsellor	Repeat questioning minimised

The 2013 amendments after the Justice Verma Committee widened the definition of rape, created offences of stalking, voyeurism and disrobing, and imposed the duty of free treatment; a child complainant is dealt with under the POCSO Act 2012.

- **In the survivor.** An acute reaction of fear, numbing and disorganisation, then post-traumatic stress disorder, depression, substance use, sexual dysfunction, self-harm and, in children, developmental and behavioural regression.
- **In the accused.** Assess paraphilic disorder, intellectual disability, psychosis, substance intoxication and personality disorder; then fitness to stand trial and responsibility, which are separate questions.

WHAT THE EXAMINATION DOES NOT DECIDE

It never decides consent, and it never opines on whether the offence occurred: both are for the court. A paraphilic diagnosis is not a defence, and the absence of injury is not evidence that nothing happened.

WHAT EARNS THE MARKS

- **Consent taken separately for each part of the examination.**
- **The prohibited two-finger test, named as prohibited.**
- **Survivor and accused answered separately.**
- **The refusal to opine on consent,** which is the boundary of the role.

SEE ALSO Slot III - 28 the POCSO Act in practice SPRINT Day 25, Mental health legislation and forensic psychiatry

III - 06

Discuss the assessment of risk for violence. Discuss nonpharmacological and pharmacological management of a patient with high risk of violence. [4+3+3]

4 + 3 + 3 | CRIMINAL AND CIVIL FORENSIC PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	**Assessment**	Three factor classes, and the method that beats intuition
3	**Non-pharmacological**	Environment, de-escalation, restraint under law
2	**Pharmacological**	Rapid tranquillisation, then the disorder itself
1	**Afterwards**	Monitor, review, and write the risk plan down



Fig 006.1 Risk factors paired row by row with the intervention each one licenses, so that the static row is seen to buy observation rather than treatment.

- **The method.** Structured professional judgement beats both unaided clinical opinion and a pure actuarial score: a validated checklist completed in full, then a formulation. Historical, clinical and risk-management items are rated, and short-term inpatient risk is tracked with a brief daily checklist.
- **Rapid tranquillisation.** Intramuscular lorazepam alone where the cause is unclear; add or substitute an antipsychotic where psychosis drives it. Do not give intramuscular olanzapine within an hour of a parenteral benzodiazepine. Monitor pulse, blood pressure, respiration, temperature and consciousness.

WHAT EARNS THE MARKS

- **Static, dynamic and protective factors named as three classes.**
- **Structured professional judgement named,** with a validated instrument.
- **De-escalation placed before any drug,** as the figure orders it.
- **Restraint tied to its statutory limits,** not offered as a treatment.

SEE ALSO Slot III - 02 fitness to stand trial **SPRINT** Day 25, Mental health legislation and forensic psychiatry

III - 07

Critically discuss the role of Psychiatrists in the court of law as expert witness in criminal offence committed by the mentally ill. [10]

10 CRIMINAL AND CIVIL FORENSIC PSYCHIATRY

RGUHS, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What an expert is	Opinion evidence, and the statute that admits it
4	The steps	Instruction to testimony, with the duty at each
2	The two questions	Fitness now, responsibility then
2	The critique	Bias, retrospection, and the limits of the science

- **The expert witness.** An ordinary witness speaks to facts he perceived; an expert may give an opinion, because the law admits opinion on a point of science from a person specially skilled in it. The psychiatrist is the court's helper, not the party's advocate, whoever pays the fee.

STEP	WHAT IS DONE	WHO ACTS	THE DUTY AT THIS STEP
1	Accept the instruction and identify who is asking and for what	Court, prosecution or defence	**Declare any prior treating role**
2	Tell the accused the purpose and that this is not treatment	Psychiatrist	No confidentiality is promised
3	Examine, and gather records, police papers and family accounts	Psychiatrist	Multiple sources, not the in-terview alone
4	Form and write the opinion, with reasoning and alternatives	Psychiatrist	**State the degree of certainty**
5	Testify and be cross-examined	The court	Concede what is not known

The two questions are separate and must be answered separately: fitness to stand trial is about the accused today, and responsibility is about his mind at the moment of the act.

- **The critique.** Retrospective opinion on a mental state months past is inference, not measurement; the adversarial setting invites partisan drift; and the court is never bound by the opinion, which is advisory only.

WHAT THE EXPERT DOES NOT DO

He does not decide guilt, does not opine that the accused is legally insane, and does not treat the person he is assessing. Medical insanity is his field; legal insanity is the court's conclusion drawn from it.

WHAT EARNS THE MARKS

- **Expert opinion distinguished from factual testimony,** with its statutory basis.
- **The warning that confidentiality does not apply,** given before the examination.
- **Fitness and responsibility answered as two questions.**
- **A genuine critique,** since the stem says critically.

SEE ALSO Slot III - 08 the McNaughton rule SPRINT Day 25, Mental health legislation and forensic psychiatry

III - 08

Discuss the McNaughton's rule. [10]

10 CRIMINAL AND CIVIL FORENSIC PSYCHIATRY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The origin	The 1843 case, and the question the Lords answered
3 The rule	Defect of reason, disease of mind, two alternatives
3 In India	The penal provision, the burden, and the case law
2 Criticism and rivals	What the rule leaves out, and the tests that answer it

- **The rule, 1843.** Daniel McNaughton, intending to kill the Prime Minister, killed his secretary and was acquitted as insane. The Lords answered: to establish the defence it must be clearly proved that, at the time of the act, the accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of his act, or, if he did know it, that he did not know that what he was doing was wrong.

STEP	WHAT MUST BE SHOWN	WHO MUST SHOW IT	STANDARD OR NOTE
1	A defect of reason arising from disease of the mind	The accused	**At the time of the act, not at trial**
2	Either not knowing the nature and quality of the act	The accused	The first alternative
3	Or not knowing that it was wrong, or contrary to law	The accused	**India adds contrary to law**
4	Proof to the civil standard, the presumption of sanity displaced	The accused	Preponderance of probabilities
5	Verdict of acquittal by reason of unsoundness, then safe custody	The court	Not a simple release

In India the rule is enacted as Section 84 of the Indian Penal Code, now Section 22 of the Bharatiya Nyaya Sanhita, and the courts have repeatedly held that legal insanity, not medical insanity, is what excuses.

- **Criticism, and the rivals.** It is a purely cognitive test: it excuses the man who did not know, never the man who knew and could not stop. Hence the irresistible impulse test, the Durham product test, and the Model Penal Code's substantial capacity test, none of which India has adopted.

WHAT THE RULE DOES NOT COVER

Volition, emotion and diminished responsibility, which English law added by statute and India has not. Intoxication is dealt with separately, and a diagnosis by itself proves nothing: the illness must have produced the specified defect at the specified moment.

WHAT EARNS THE MARKS

- **The rule quoted with both alternatives,** not paraphrased into one.
- **Section 84 named, with the Sanhita renumbering noted.**
- **Legal insanity against medical insanity,** the line Indian courts insist on.
- **The cognitive limit and one named rival test.**

SEE ALSO Slot III - 07 the psychiatrist as expert witness **SPRINT**

Day 25, Mental health legislation and forensic psychiatry

III - 09

Adoption. [10]

10 CRIMINAL AND CIVIL FORENSIC PSYCHIATRY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	A permanent transfer of parentage, in law
3	The legal routes	Two statutes, two different things they do
3	Eligibility and process	Who may adopt, who is adoptable, who decides
2	Psychiatric aspects	Telling, attachment, and what follows placement

THE DEFINITION

Adoption is the process by which a child is permanently separated from his biological parents and becomes the lawful child of his adoptive parents, with all the rights, privileges and responsibilities that attach to a biological child. It is a change of status, not a placement.

ROUTE	WHAT IT PROVIDES	WHO IT SERVES
Juvenile Justice Act 2015	Full adoption, secular and available to all	**The general route, through the central adoption authority**
Hindu Adoptions and Maintenance Act 1956	Full adoption under personal law	Hindus, Buddhists, Jains and Sikhs
Guardians and Wards Act 1890	Guardianship only, ending at majority	**Not adoption, and confers no inheritance**

Under the 2015 Act a child is declared legally free for adoption by the Child Welfare Committee, prospective parents are registered and home-studied, and the adoption order is now passed by the District Magistrate.

- **Eligibility, in outline.** Physically, mentally and financially fit parents in a stable relationship of at least two years; at least twenty five years between child and parent; a single woman may adopt any child, a single man may not adopt a girl; the permitted composite age of the parents falls as the child's age rises.
- **Psychiatric aspects.** Disclosure told early and repeatedly rather than once; attachment difficulty and developmental delay after institutional care; identity questions in adolescence; grief in the adoptive parents where infertility preceded; and a raised rate of referral, which reflects both risk and help-seeking.

WHAT EARNS THE MARKS

- **Adoption defined as a change of legal status,** not as care of a child.
- **Guardianship separated from adoption,** which is the commonest confusion.
- **The two firm numbers,** the twenty five year gap and the single man rule.
- **Disclosure and attachment,** which is where the psychiatrist actually contributes.

SEE ALSO Slot III - 27 the Juvenile Justice Act SPRINT Day 25, Mental health legislation and forensic psychiatry

III - 10

A psychiatric patient is referred to you to determine if he has the capacity to make a will. What steps you will take to know his capacity to make a will and how will you report. [5+5]

5 + 5 | CRIMINAL AND CIVIL FORENSIC PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1	**Clarify the referral**	Who asks, why now, and what is being disposed
4	**The steps**	In order, with the condition attached to each
3	**The report**	Six headings, and the opinion in one sentence
2	**Limits**	What the report must decline to say

STEP	WHAT IS DONE	WHO ACTS	CONDITION
1	Confirm the instruction, and explain to the patient that this is an assessment, not treatment	Psychiatrist	**No confidentiality is promised**
2	Optimise first: correct delirium, sensory impairment, sedation, pain	Treating team	Assess at his best, not his worst
3	Take history, records and collateral, including the solicitor's instructions	Psychiatrist	Beneficiaries interviewed separately
4	Test the four limbs by open question, and screen cognition	Psychiatrist, patient alone	**Record the answers verbatim**
5	Re-examine on a second occasion where the state fluctuates	Psychiatrist	As close to execution as possible

The report: identifying data and instruction; sources used; the examination and the verbatim answers; findings on each of the four limbs; the opinion with its reasoning; and the limits of that opinion.

- **The opinion.** One plain sentence saying whether, in your opinion and on the balance of probabilities, the patient had testamentary capacity at the time of examination, with the reasons that led there and the alternative you rejected.

WHAT THE REPORT DOES NOT COVER

It does not declare the will valid, does not rule on undue influence, and does not certify capacity for any other decision or any other date. Capacity is decision-specific and time-specific, and the report should say so in terms.

WHAT EARNS THE MARKS

- **Optimising the patient before testing him,** which most answers skip.
- **Verbatim recording,** because the record is what the court reads.
- **A report with named headings,** not a narrative letter.
- **The stated limits,** which is the second half of the question.

SEE ALSO Slot III - 01 testamentary capacity **SPRINT** Day 25, Mental health legislation and forensic psychiatry

AREA 02 OF 13 · P3-A02

Intellectual disability and learning disorders

10	10.9%	III-11	III-20
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 SINGLE 9

REVISION ORDER

Definition and classification first: five of the ten slots open with them. Certification and IDEAS next, because they repeat verbatim in the legislation area. The named syndromes and the test list last.

WHAT IS IN THIS AREA

Definition and classification of intellectual disability (three slots), disability certification and its regulations (two), specific learning disorder and scholastic backwardness, the intelligence tests used in India, Fragile X, and stereotypic movement disorder.

III - 11

Fragile X syndrome. [10]

10 INTELLECTUAL DISABILITY AND LEARNING DISORDERS

KNRUHS, NTRUHS, RGUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2	The genetics	Gene, repeat, methylation, and the protein lost
2	Physical phenotype	Face, ears, jaw, testes, joints, valve
4	Behaviour and cognition	The named cluster, and the autism overlap
2	Diagnosis and care	The right test, then what actually helps the family

THE DEFINITION

The commonest inherited cause of intellectual disability, and the commonest single-gene cause of autism. An expanded CGG repeat in the FMR1 gene on the long arm of the X chromosome silences the gene by methylation once the full mutation is reached. Loss of its protein removes a brake on synaptic signalling, leaving long thin immature dendritic spines.

DOMAIN	FULL MUTATION, MALE	FEMALE AND PREMUTATION	WHAT SETTLES IT
Repeat size	Over 200 repeats, methylated, gene silent	55 to 200 is a premutation, gene still active	Repeat sizing, not a karyotype
Face and body	Long face, large ears, prominent jaw; after puberty large testes and lax joints	Milder or absent, by X inactivation	Suggestive only
Cognition	Mild to moderate disability, verbal better than visuospatial	Learning difficulty or normal range	Variable, so test the gene
Carrier illness	Not applicable	Tremor and ataxia in older men, early ovarian failure in women	Premutation, not full mutation

Amber cells are the two facts that decide the answer: size the repeat, and keep premutation illness separate from the syndrome itself.

- **The behavioural cluster.** Gaze aversion with social interest preserved, hand flapping and hand biting, shyness and social anxiety, hyperactivity and inattention, perseverative repetitive speech, sensory hyperarousal and tantrums on change. Autism is diagnosed in a substantial minority.
- **Diagnosis and management.** Order FMR1 repeat testing, not a karyotype. No treatment corrects the defect. Early intervention, speech and occupational therapy, a stimulant for inattention and a serotonin reuptake inhibitor for anxiety, plus predictable low-stimulus routines.
- **The family, not just the child.** Anticipation runs through the transmitting mother, so counsel and offer cascade testing. Certify under the Rights of Persons with Disabilities Act 2016 once intellectual disability is established.

WHAT EARNS THE MARKS

- **Gene, repeat threshold and methylation in one line.** Saying X-linked without the repeat is a half mark.
- **The premutation row.** Tremor ataxia and ovarian failure belong to carriers, and most answers never separate them.
- **Repeat testing named as the test.** Ordering a karyotype is the classic wrong answer here.

SEE ALSO Slot III - 12 aetiology of intellectual disability Slot III - 89 clinical features of autism

III - 12

Provide classification of Intellectual disability (Mental retardation) based on etiology. [10]

10 INTELLECTUAL DISABILITY AND LEARNING DISORDERS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1	The axis	State it: when the insult acted, not how severe
4	Prenatal	Genetic and acquired kept apart, with examples
3	Perinatal and postnatal	In order, and which of them India can prevent
2	Honest limits	Severity is a second axis; many causes go unfound

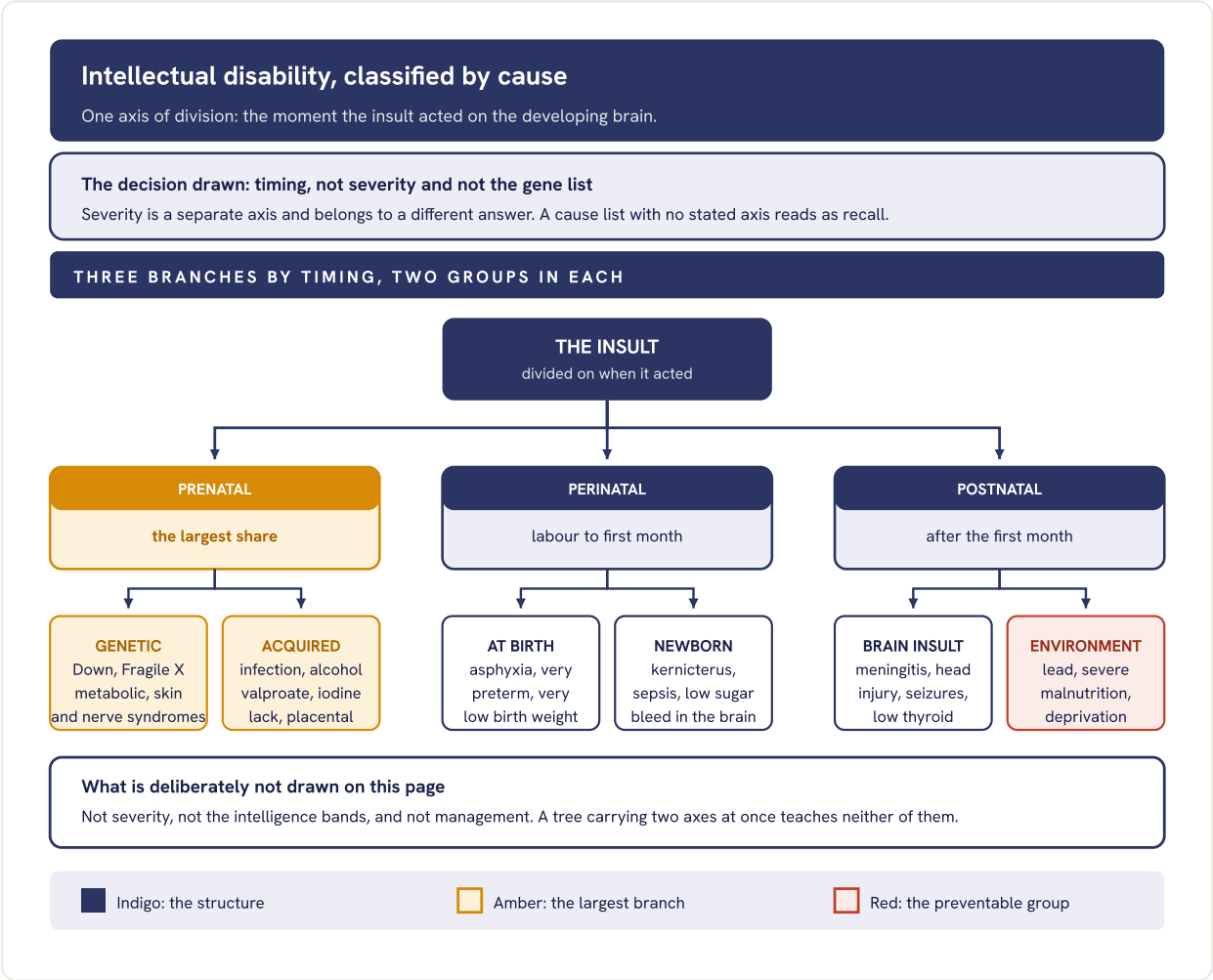


Fig 012.1 Aetiology divided on the timing of the insult, with a genetic and an acquired group inside each branch. The red chip marks the causes public health can remove.

- **What India can prevent.** Iodine fortification, rubella immunisation, folic acid, anaemia and nutrition programmes, skilled birth attendance and newborn resuscitation, newborn screening for low thyroid, lead control, and deworming. Newborn and early childhood screening runs through the national child health screening programme.
- **The honest limit.** No cause is found in roughly a third to a half of cases, and the yield falls further as severity falls. Chromosomal microarray and exome sequencing raise it, but neither is routinely available across Indian districts.

WHAT EARNS THE MARKS

- **The axis stated in one line before the tree.** Timing, and only timing.
- **Genetic separated from acquired inside the prenatal branch.** Merging them is the commonest structural loss.
- **Every branch of the plate reproduced,** with at least two named examples in each.
- **The unfound-cause line.** It reads as command of the field rather than a memorised list.

SEE ALSO Slot III - 11 Fragile X syndrome Slot III - 14 definition and certification

Slot III - 16 neurodevelopmental disorders and prevention

III - 13

Enumerate common disability certifications in psychiatry. Discuss existing certification regulations. How to assess specific learning disability (SLD)? What are the assistance provided by the Government for a SLD patient? [2+4+2+2]

2 + 4 + 2 + 2 | INTELLECTUAL DISABILITY AND LEARNING DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What is certifiable	The psychiatry entries in the schedule, named
4	The route	Who applies, who assesses, which scale, how long
2	Assessing learning disability	Exclusions first, then the index and the age rule
2	What the child gets	Exam provisions, reservation, schemes

- **Certifiable in psychiatry.** Of the twenty-one disabilities specified by the Rights of Persons with Disabilities Act 2016, five are ours: mental illness, intellectual disability, specific learning disabilities, autism spectrum disorder, chronic neurological conditions.
- **The instrument decides the number.** Mental illness scores on the Indian Disability Evaluation and Assessment Scale; intellectual disability on an intelligence test with an adaptive scale; learning disability on the NIMHANS Index. Benchmark disability begins at forty percent.

STEP	WHAT HAPPENS	WHO ACTS	CONDITION OR INTERVAL
1	Application with identity and address proof	Parent or guardian, on the national disability identity portal	Any time after the assessable age
2	Assessment against the 2018 gazette guidelines	Notified medical authority, as a board	Board must include the relevant specialist
3	Scoring on the prescribed instrument	Psychiatrist, clinical psychologist, special educator	**Percentage, not a diagnosis alone**
4	Certificate and identity card issued; appeal lies against the percentage	Certifying authority, then the district appellate authority	**Permanent if the condition is; otherwise reassessed**

WHAT THE ACT DOES NOT COVER

A certificate is an entitlement document, not a diagnosis and not a treatment order: rights to care come from the Mental Healthcare Act 2017 instead. Learning disability is not certified before about eight years and the third class, because the achievement gap cannot be measured earlier, and no certificate waives a course's core competency requirement.

WHAT EARNS THE MARKS

- **Five psychiatry entries named from the schedule,** not a general statement that mental illness is covered.
- **One instrument tied to each condition.** The scale is what turns a diagnosis into a percentage.
- **Steps in order with the actor at each one,** and the forty percent threshold stated.
- **The exclusions strip.** The age and class rule for learning disability is the line most answers miss.

SEE ALSO Slot III - 14 certification in intellectual disability Slot III - 18 assessment of specific learning disorder

SPRINT Day 25, Mental health legislation and forensic psychiatry

III - 14

Define intellectual disability and give its classification. Discuss disability certification in intellectual disability and the benefits being provided by the Government of India. [1+3+3+3]

1 + 3 + 3 + 3 | INTELLECTUAL DISABILITY AND LEARNING DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1	Definition	Three criteria, and the domain word that carries them
3	Classification	Four grades set by adaptive function, tests named
3	Certification	The steps, in order, with actor and interval
3	Benefits	Money, guardianship, education, employment

- **Definition, three criteria.** Deficits in intellectual function on testing; deficits in adaptive function across the conceptual, social and practical domains; onset in the developmental period. The domain triad is the part most answers drop.
- **Classification.** Mild, moderate, severe and profound, graded on adaptive function rather than the intelligence quotient alone. Indian tests: Binet Kamat, Malin's Intelligence Scale for Indian Children, Seguin Form Board, and the Vineland Social Maturity Scale for social age.

STEP	WHAT HAPPENS	WHO ACTS	CONDITION OR INTERVAL
1	Application on the national disability identity portal	Parent or legal guardian	From about three years, once testing is valid
2	Intelligence testing with an adaptive behaviour scale	Clinical psychologist	**Both, never intelligence alone**
3	Medical board assessment against the 2018 guidelines	Notified medical authority	Percentage recorded on the certificate
4	Certificate and identity card issued; guardianship applied for where capacity is absent	Certifying authority, then the local level committee	**Permanent, since the condition is lifelong**

Benefits follow at forty percent: disability pension, income tax relief under Sections 80DD and 80U, National Trust health insurance, four percent of government posts, five percent of higher education seats, aids, scholarships, travel concession.

WHAT THE ACT DOES NOT COVER

Intellectual disability is not a mental illness under the Mental Healthcare Act 2017 unless a mental illness coexists, so that Act's admission machinery does not follow from this certificate. Guardianship is not a nominated representative, and a percentage is not a capacity finding.

WHAT EARNS THE MARKS

- **All three definition criteria, with the three adaptive domains named.**
- **Grading tied to adaptive function,** with at least two Indian tests named.
- **Steps in order with the actor at each,** and the forty percent threshold before any benefit is listed.
- **The exclusions strip.** Separating the two Acts is what marks a top answer here.

SEE ALSO Slot III - 13 disability certification in psychiatry Slot III - 15 severity grades and pension schemes Slot III - 17 diagnosis and psychosocial management

III - 15

Define intellectual disability according to DSM-5. Compare various degrees of severity of intellectual disability. What are the various benefits and pension schemes for persons with intellectual disability in India? [1+5+4]

1 + 5 + 4 | INTELLECTUAL DISABILITY AND LEARNING DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1 Definition	Three criteria, then name the axis of grading
5 The grid	Four grades across the three adaptive domains
2 Pension and insurance	The named schemes, with the qualifying threshold
2 Other benefits	Tax, reservation, education, aids, travel

THE DEFINITION

Onset in the developmental period, with deficits in intellectual function on clinical assessment and standardised testing, together with deficits in adaptive function limiting independence across the conceptual, social and practical domains. Severity is set by adaptive function, not by the intelligence quotient.

GRADE	CONCEPTUAL	SOCIAL AND PRACTICAL	WHAT SETTLES IT
Mild	Academic difficulty from school age; abstract thinking, money and time weak	Immature judgment, easily led; self-care intact, needs help with complex tasks	Lives independently with intermittent support
Moderate	Primary school academics at best; slow acquisition throughout	Simple spoken language; self-care learned with long teaching and reminders	Needs daily prompting; supervised unskilled work
Severe	Little grasp of written language, number, quantity or time	Single words and phrases; supported for every daily activity	Supervision at all times
Profound	Understanding is of the physical world; may match and sort objects	Very limited symbolic communication; motor and sensory impairment common	Dependent for all personal care

Amber cells are the support statements. Grading on support need rather than on a test score is the change from the earlier manuals, and saying so is itself a mark.

- **Pension and insurance.** Benefits begin at forty percent. The national social assistance disability pension covers adults below the poverty line, with state pensions alongside. National Trust insurance covers intellectual disability, autism, cerebral palsy and multiple disability.
- **The rest of the package.** Tax relief under Sections 80DD and 80U, four percent of government posts and five percent of higher education seats, scholarships, aids and appliances, travel concession, and guardianship through the local level committee.

WHAT EARNS THE MARKS

- **A grid, not four paragraphs.** The comparison has to be readable across a row.
- **Adaptive function named as the grading axis** before the grid begins.
- **The forty percent threshold stated once,** before any scheme is listed.
- **Schemes named,** not described as government help in general terms.

SEE ALSO Slot III - 14 definition and certification Slot III - 17 diagnosis and psychosocial management

III - 16

a) What are the neurodevelopment disorders in children? b) What are the preventive measures of neurodevelopment disorders? c) Discuss schizophrenia as a neurodevelopment disorder. [2+3+5]

2 + 3 + 5 | INTELLECTUAL DISABILITY AND LEARNING DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The six families	Named as a group, with the shared feature stated
3	Prevention	Primary, secondary, tertiary, with Indian programmes
3	The evidence	Obstetric, premorbid, structural, genetic
2	The model and its limit	Two hits, and what the model does not explain

FAMILY	MEMBERS	CORE DEFICIT
Intellectual	Intellectual disability, global developmental delay	Reasoning and adaptive function
Communication	Language, speech sound, fluency, social pragmatic	Producing or using language
Autism spectrum	One category, severity graded on support	**Social communication plus repetitive behaviour**
Attention deficit	Inattentive, hyperactive impulsive, combined	Regulating attention and activity
Specific learning	Reading, written expression, mathematics	**One skill, intelligence in range**
Motor	Coordination, stereotypic movement, tic disorders	Acquiring or controlling movement

- **Prevention in three tiers.** Primary: folic acid, salt iodisation, rubella immunisation, no alcohol or valproate in pregnancy, nutrition programmes, genetic counselling, skilled birth attendance. Secondary: newborn thyroid screening, national child health screening, developmental surveillance. Tertiary: early intervention, remediation, inclusive schooling.

SCHIZOPHRENIA AS A NEURODEVELOPMENTAL DISORDER

Four lines of evidence. Obstetric complications, prenatal famine and prenatal infection raise risk. Premorbid milestone delay, lower childhood intelligence and poor social adjustment precede onset by years. Ventricular enlargement is present at first episode, with disordered cytoarchitecture and no gliosis, so a developmental lesion and not a degenerative one. Risk genes overlap with autism, and excessive adolescent pruning supplies the second hit.

- **What the model does not explain.** Why onset waits for late adolescence in most cases, why some patients have no premorbid abnormality at all, and why the illness can progress after onset. Say this, and the answer reads as understanding rather than recall.

WHAT EARNS THE MARKS

- **Six families, one member each, and the shared feature stated:** a deficit in acquiring a function, never in losing one.
- **Prevention split into three tiers,** with named Indian programmes rather than general advice.
- **Four separate lines of evidence for the schizophrenia part,** then the limit of the model.

SEE ALSO Slot III - 12 aetiology of intellectual disability

Slot III - 55 neurobiology of attention deficit hyperactivity disorder

III - 17

a) How to diagnose Mental retardation? b) Enumerate any four intelligence tests used commonly for child and adolescent. c) What are the principles for psycho social management of persons with intellectual disability? d) What are the benefits offered by Government of India for person with intellectual disability? [2+2+4+2]

2 + 2 + 4 + 2 | INTELLECTUAL DISABILITY AND LEARNING DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Making the diagnosis**	Three criteria, then the workup that finds the cause
2	**Four tests**	Named, with the age band each covers
4	**Psychosocial principles**	The flowchart: assess, teach, shape, protect
2	**Government benefits**	The threshold, then the named schemes

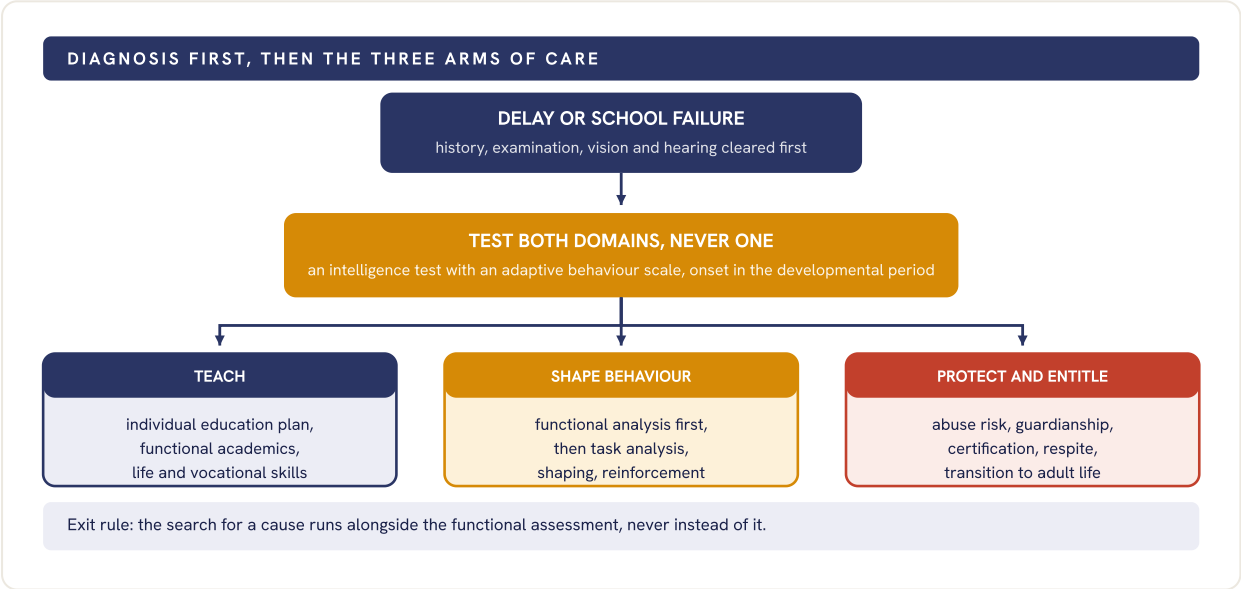


Fig 017.1 Diagnosis made on both domains together, then the three arms of psychosocial care, with the search for a cause carried along the foot.

- **Four tests, with their range.** Binet Kamat, three years upward; Malin's Intelligence Scale for Indian Children, six to fifteen; Wechsler Intelligence Scale for Children, Indian adaptation; Seguin Form Board for younger and non-verbal children. Adaptive function is rated separately, commonly on the Vineland Social Maturity Scale.
- **Benefits, at forty percent.** Disability pension, National Trust insurance and guardianship, tax relief under Sections 80DD and 80U, four percent of posts, five percent of seats, aids, travel concession.

WHAT EARNS THE MARKS

- **Both domains tested, written as the diagnostic step.** Resting on the intelligence quotient alone loses here.
- **Four tests named with their age bands,** the adaptive scale kept separate.
- **Functional analysis before any behavioural technique,** and the forty percent threshold before the benefits.

SEE ALSO Slot III - 14 definition and certification Slot III - 15 severity grades and pension schemes

III - 18

Enumerate the causes of Scholastic backwardness. Discuss in detail about the epidemiology, clinical features, assessment and management of Specific Learning Disorder. [10]

10 | ASKED AT 15 MARKS | INTELLECTUAL DISABILITY AND LEARNING DISORDERS

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Causes of backwardness	Six groups, each with the clue that points to it
2	Epidemiology and features	Prevalence range, the three types, what travels with them
3	Assessment	Exclude, then test, then the Indian index
2	Management	Remediate, accommodate, certify, treat what coexists

GROUP	CAUSES	THE CLUE THAT POINTS THERE
Cognitive	Intellectual disability, learning disorder, borderline intelligence	All subjects, or only one
Sensory and medical	Vision, hearing, epilepsy, sedating drugs, anaemia, worms	Copying, repetition, drowsiness
Mind and mood	Attention deficit, autism, depression, anxiety, bullying, substance use	A dated fall in the record
Home and school	Poverty, labour, migration, violence; teaching, class size, language	The whole class fails

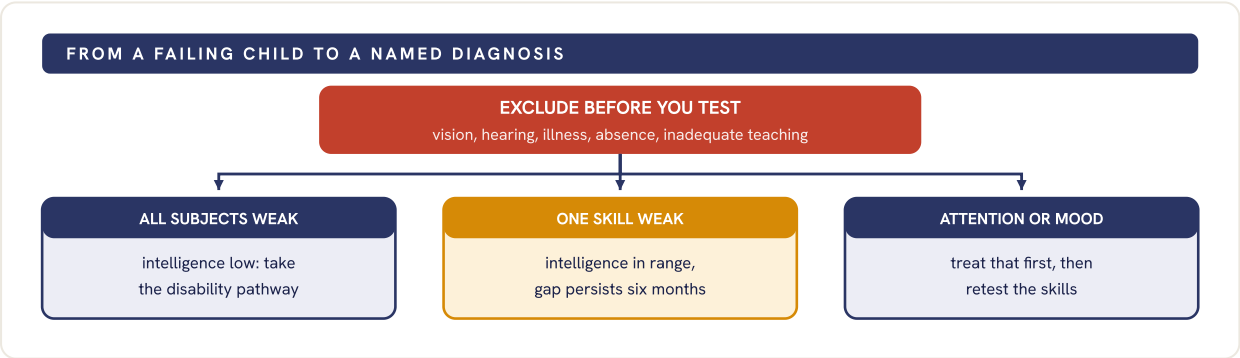


Fig 018.1 Exclusion first, then the three profiles a failing child resolves into. Only the amber lane is a learning disorder.

- **Epidemiology and features.** Indian school surveys report roughly five to fifteen percent, the spread reflecting the instrument rather than the population. Three types: reading, written expression, mathematics.
- **Assessment and management.** Achievement testing with an intelligence test, then the NIMHANS Index for certification from about eight years. Multisensory remediation, exam accommodations, and progress judged on curriculum based measurement.

WHAT EARNS THE MARKS

- **Causes grouped, not listed flat,** each group with the clue that points to it.
- **The exclusion step written before any testing.**
- **Prevalence as a range with the reason for it,** then remediation and accommodation named separately.

SEE ALSO Slot III - 13 assessing specific learning disability Slot III - 20 impairment in written expression

III - 19

Define Stereotypic Movement Disorder as per DSM-5. How would you differentiate it from body-focused repetitive behavior disorders? Discuss the management of Stereotypic Movement disorder. [2+4+4]

2 + 4 + 4 | INTELLECTUAL DISABILITY AND LEARNING DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	Criteria, then the two specifiers that change management
4 The grid	Domain by domain, with the discriminators marked
2 Behavioural management	Function first, then the technique that fits it
2 Drugs and protection	Only for self injury, and only with a review date

THE DEFINITION

Repetitive, seemingly driven and apparently purposeless motor behaviour, such as rocking, hand waving, head banging or self biting, beginning in the early developmental period, interfering with activity or causing self injury, and not better explained by a substance, a neurological condition or another mental disorder. Specify with or without self injurious behaviour.

DOMAIN	STEREOTYPIC MOVEMENT	BODY-FOCUSED REPETITIVE BEHAVIOUR	WHAT SETTLES IT
Onset	Early developmental period, often before three	Around puberty, sometimes later	**Onset before three years**
Form	Rhythmic, bilateral, whole body or limb	Focused grooming of hair, skin or nails	**Rhythm against grooming**
Product	Purposeless, nothing removed	Tissue removed or damaged	Follows from form
Urge	Occurs when engrossed or stressed; distractible	Premonitory urge, then relief	**A named urge first**
What works	Reinforcement based behaviour work, enrichment	Habit reversal training	The retrospective test

- **Management, in order.** Treat pain and medical drivers first: dental disease, otitis, constipation, reflux, eye disease in the blind child. Then a functional analysis. Then reinforcement of a competing behaviour, response interruption and redirection, enrichment and sensory work, and parent training. Protective equipment is short term and reviewed, never a substitute for the analysis.
- **Drugs.** No drug treats the stereotypy itself. Where self injury is severe, risperidone or aripiprazole have the best evidence, borrowed from irritability trials in autism, with metabolic monitoring. Restraint used without a functional analysis increases the behaviour it is meant to stop.

WHAT EARNS THE MARKS

- **The self injury specifier named in the definition.** It is what changes the management plan.
- **A grid, with onset, form and urge marked as the discriminators,** and tics named as the third confusable.
- **Pain and medical causes searched before behaviour is analysed.**
- **Drugs placed last and tied to self injury only.**

SEE ALSO Slot III - 31 tics and Tourette syndrome Slot III - 39 pica

III - 20

a) What are Specific Learning Disorder? b) What are various types of Specific Learning Disorder. c) Write steps in assessment and management of Specific Learning Disorder with impairment in written expression. [2+3+5]

2 + 3 + 5 | INTELLECTUAL DISABILITY AND LEARNING DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Definition**	Persistence, the six-month rule, and the exclusions
3 **The three types**	With the sub-skills each specifier names
3 **Assessment steps**	The flowchart: clear, test, profile, certify
2 **Management steps**	Teach the skill, then remove the barrier

THE DEFINITION

Difficulty learning and using an academic skill, present despite targeted help, persisting at least six months, well below that expected for age. Not better explained by intellectual disability, uncorrected vision or hearing, another disorder, adversity, the language of instruction, or inadequate teaching.

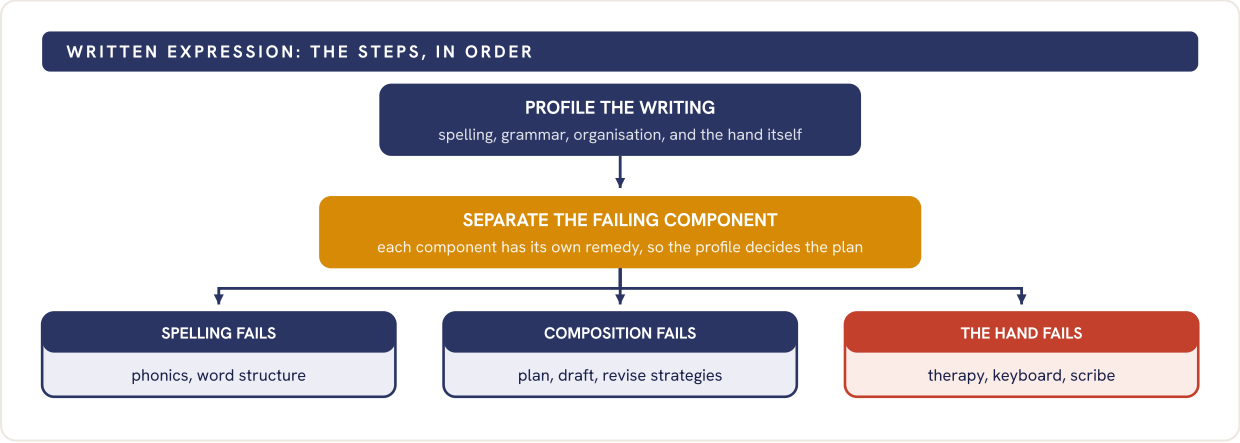


Fig 020.1 The writing profile split into its three failing components. Remediation teaches the skill, accommodation removes the barrier, and the child needs both.

- **The three types.** Reading: word accuracy, rate, comprehension. Written expression: spelling, grammar and punctuation, clarity of organisation. Mathematics: number sense, arithmetic facts, calculation, reasoning. Severity is graded mild, moderate or severe.
- **Steps that get skipped.** Test oral language too, since weak spoken language produces weak writing. Certify from about eight years for exam provisions: extra time, a scribe where the hand is the problem, spelling not penalised outside a spelling paper.

WHAT EARNS THE MARKS

- **The six-month persistence rule and the exclusion list.** A definition without them is half a definition.
- **Three specifiers with their sub-skills,** not three bare names.
- **The failing component identified before any remedy is chosen.**
- **Remediation separated from accommodation,** with the exam provisions named.

SEE ALSO Slot III - 18 scholastic backwardness and assessment Slot III - 13 assessing specific learning disability

AREA 03 OF 13 · P3-A09

Ethics, consent and child protection

10	10.7%	III-21	III-30
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 9

REVISION ORDER

Learn the four principles once, properly: they carry three slots and they frame the consent slot as well. Then the abuse and POCSO group, which shares a management spine. Boundaries last, and it is short.

WHAT IS IN THIS AREA

The four principles of ethics and their application (three slots), informed consent, boundary violations, and child sexual abuse with POCSO and the Juvenile Justice Act (four slots between them).

III - 21

Discuss the long-term consequences of child sexual abuse. [10]

10 ETHICS, CONSENT AND CHILD PROTECTION

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The frame	Not one syndrome; outcome is the rule, not the exception
4 Domain by domain	Psychiatric, developmental, interpersonal, physical
2 Mechanism	Why the sequelae are so wide, said in one line
2 What modifies it	The factors that make outcome better or worse

THE FRAME

There is no single post-abuse syndrome. Child sexual abuse is a non-specific risk factor of large effect, raising the likelihood of many adult disorders rather than producing one. A minority remain asymptomatic, and saying so is part of an accurate answer.

DOMAIN	WHAT APPEARS IN ADULT LIFE	THE CLINICAL MARKER
Psychiatric	Depression, post-traumatic stress disorder, anxiety, substance use, eating disorder	**Self-harm and suicide attempt rates rise sharply**
Personality and affect	Emotional dysregulation, dissociation, chronic shame, borderline pattern	Complex trauma presentation
Interpersonal	Insecure attachment, difficulty with trust and intimacy, revictimisation	**Revictimisation is the most robust finding**
Sexual and reproductive	Sexual dysfunction, early sexual activity, unplanned pregnancy	Often unvolunteered unless asked
Physical and social	Chronic pain, functional gastrointestinal disorder, poorer education and work outcome	Presents to medicine, not psychiatry

The amber rows are the two an examiner expects by name; the physical row is the one that shows the answer was learned from patients rather than a list.

- **Why so wide.** Abuse acts through disrupted attachment, altered stress response, traumatic sexualisation, stigmatisation and betrayal, and each of those routes has its own downstream disorders.
- **What modifies outcome.** Worse with penetrative and repeated abuse, an abuser close to the child, and disbelief on disclosure. Better with early disclosure that is believed, one supportive caregiver, and treatment received.

WHAT EARNS THE MARKS

- **Non-specificity stated at the top,** with the asymptomatic minority acknowledged.
- **Consequences given by domain,** not as an undifferentiated list.
- **A mechanism offered,** which separates the top band.
- **Moderators in both directions,** protective as well as adverse.

SEE ALSO Slot III - 25 assessment and management of abuse **SPRINT** Day 24, Child psychiatry

III - 22

Discuss principles of ethics and their implications for clinical practice. [10]

10 ETHICS, CONSENT AND CHILD PROTECTION

DNB, NTRUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	The four principles	Named, with the tradition they come from
4	What each demands	One concrete clinical act per principle
2	When they collide	No hierarchy; the collision is the work
2	The Indian frame	The code that actually binds a practitioner here

THE DEFINITION

Medical ethics is the systematic study of what a doctor ought to do. The working framework is the four principles of Beauchamp and Childress: respect for autonomy, beneficence, non-maleficence and justice, with veracity, fidelity and confidentiality as derived duties.

PRINCIPLE	WHAT IT DEMANDS AT THE BEDSIDE	HOW IT IS BREACHED IN PRACTICE
Autonomy	Informed consent, truth-telling, respecting a refusal	Consent taken as a signature, not a conversation
Beneficence	Acting in the patient's interest, not the family's or your own	Treating to relieve the relatives' distress
Non-maleficence	Weighing harm, monitoring, stopping what is not working	Polypharmacy continued without review
Justice	Fair use of scarce time, beds and money; no discrimination	Preferring the articulate or the paying patient

The four have no fixed rank. An answer that names a hierarchy is wrong; an answer that shows the collision being reasoned through is what is being marked.

- **The collision.** Autonomy against beneficence is the daily conflict of this specialty: the patient who refuses treatment he needs. It is resolved by testing capacity, not by overriding the refusal, and by using the least restrictive option that answers the risk.
- **The Indian frame.** Principles are argued, but a practitioner here is bound by the Medical Council code of ethics regulations of 2002, still the operative code, and by the rights chapter of the Mental Healthcare Act 2017.

WHAT EARNS THE MARKS

- **All four named and attributed.**
- **A concrete clinical act under each,** which is the implications half of the question.
- **The absence of a hierarchy, stated.**
- **An Indian instrument named,** not a purely Western answer.

SEE ALSO Slot III - 30 scope of the principles in mental health Slot III - 24 informed consent SPRINT Day 26, Forensic ethics, consent and legal procedure

III - 23

Describe the applications of ethics in mental health. What are the ethical issues to be considered in a drug trial? [5+5]

5 + 5 | ETHICS, CONSENT AND CHILD PROTECTION | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Why psychiatry is different	Capacity, coercion and stigma, all at once
3	The applications	Five settings where the principles actually bite
3	Trial ethics	Consent, vulnerability, placebo, compensation
2	The Indian regulation	Which committee, which guideline, which registry

SETTING	THE ETHICAL QUESTION	HOW IT IS ANSWERED
Involuntary care	Whose judgement replaces the patient's	**Capacity test, least restrictive option, review**
Confidentiality	What may be told to a family that provides the care	Consent, and disclosure limited to what is needed
Risk to a third party	Whether to warn	**Weighed case by case; not an Indian statutory duty**
Boundaries and dual roles	Treating and assessing the same person	Declare the role before the interview
Telepsychiatry and records	Identity, privacy and the limits of remote care	Follow the 2020 telemedicine guidelines

Psychiatry differs because the illness can impair the very faculty consent depends on, and because the setting can be coercive even when nobody intends it to be.

- **In a drug trial.** Independent ethics committee approval before enrolment; capacity assessed for consent, and assent with a legally acceptable representative where it is absent; an independent witness or audiovisual recording for vulnerable participants; placebo only where no proven treatment is being withheld to the participant's harm; free treatment for adverse events and compensation for trial-related injury; the right to withdraw without any loss of usual care.
- **The Indian regulation.** The national ethical guidelines for biomedical and health research, the New Drugs and Clinical Trials Rules 2019, a registered ethics committee, and prior registration of the trial in the national registry.

WHAT EARNS THE MARKS

- **Why psychiatry is a special case,** stated before the list.
- **Applications given as settings,** each with its resolution.
- **Capacity for research consent treated separately** from capacity for treatment.
- **Compensation and registration,** which are the Indian specifics.

SEE ALSO Slot III - 24 informed consent SPRINT Day 26, Forensic ethics, consent and legal procedure

III - 24

What is informed consent? What are the various components of informed consent? Discuss its relevance in the patients with psychotic disorders as compared to medical disorders. [1+3+6]

1 + 3 + 6 | ETHICS, CONSENT AND CHILD PROTECTION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1 Definition	A process, not a signature
3 The components	Five, each of which can fail on its own
4 The comparison	Domain by domain, with what settles each
2 What follows	When capacity is absent, the law supplies the route

- **Informed consent.** A voluntary agreement to a specific intervention by a person with the capacity to decide, given after disclosure of its nature, benefits, risks and alternatives, including the alternative of no treatment. Five components: disclosure, understanding, capacity, voluntariness, and the authorisation itself.

DOMAIN	PSYCHOTIC DISORDER	MEDICAL DISORDER	WHAT SETTLES IT
Capacity	May be impaired, and may fluctuate within the day	Usually intact unless delirium or pain	**Test the four abilities, do not assume**
Understanding	Thought disorder and poor insight distort it	Limited by literacy and anxiety	Ask the patient to say it back
Voluntariness	Ward setting and family pressure are coercive	Pressure is mostly time and cost	**Who benefits if he agrees**
Refusal	Refusal is read as illness, wrongly	Refusal is respected readily	**An unwise choice is not incapacity**
If capacity is absent	Nominated representative, advance directive, supported admission	Next of kin, emergency doctrine	The Mental Healthcare Act 2017 supplies the route

Amber cells are the discriminators. The last one carries the most marks: the same refusal is treated differently in the two settings, and that asymmetry is what the question is really asking about.

- **The Indian standard.** Disclosure is judged by what a reasonably competent practitioner would tell, not by what a particular patient would want to know, and consent covers only the procedure consented to.

WHAT EARNS THE MARKS

- **Consent as a process, in the first line.**
- **All five components named.**
- **A grid, not two paragraphs,** with discriminators marked as discriminators.
- **The refusal asymmetry,** which is the heart of the six-mark part.

SEE ALSO Slot III - 43 capacity under the Act SPRINT Day 26, Forensic ethics, consent and legal procedure

III - 25

Discuss the assessment and management of a case of child sexual abuse. [5+5]

5 + 5 | ETHICS, CONSENT AND CHILD PROTECTION | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	First contact	Believe, do not interrogate, and report
3	Assessment	Medical, psychiatric and safety, in that order
3	Early management	Protect, treat, and prepare for the process
2	Longer term	Trauma-focused therapy and the family's own work

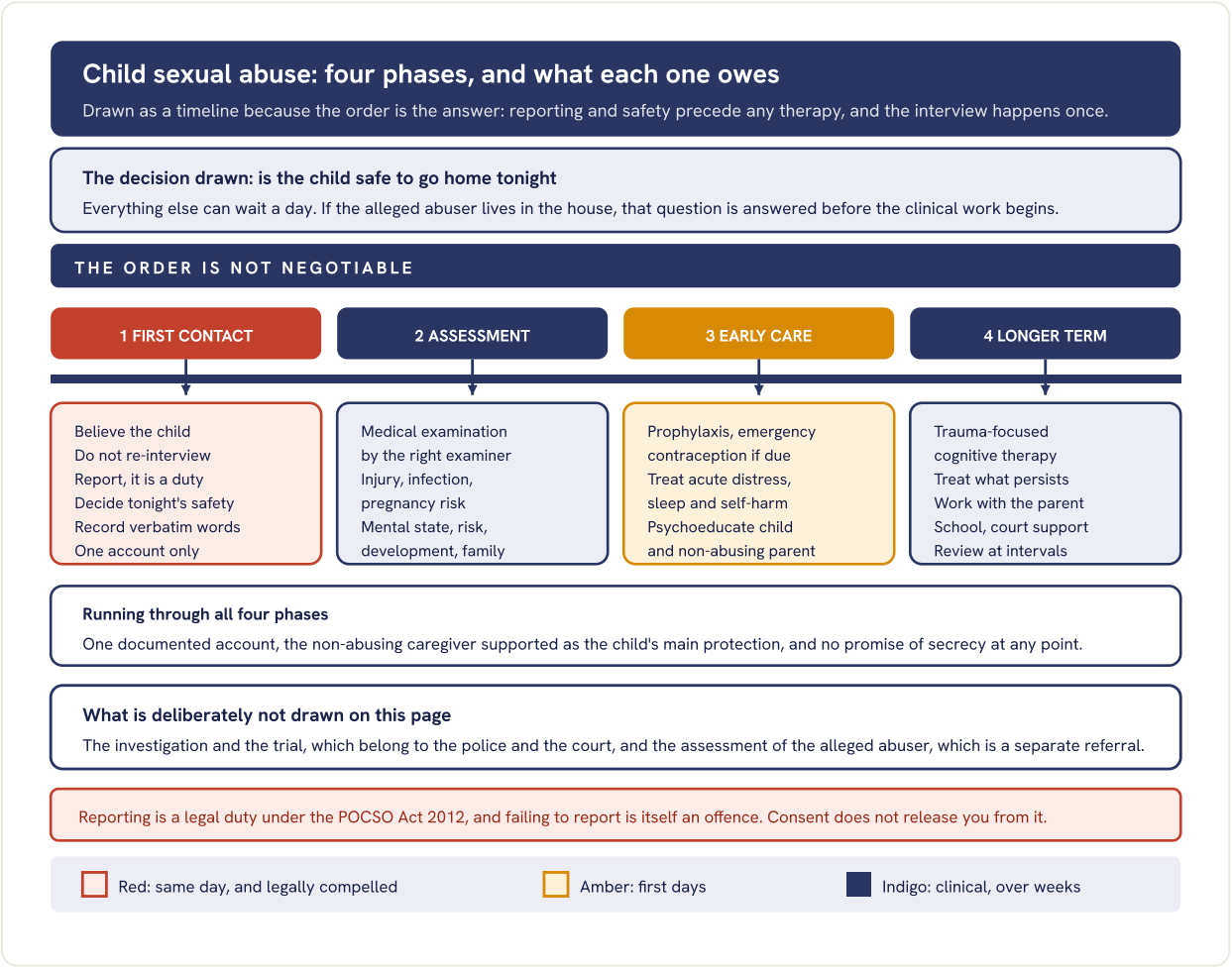


Fig 025.1 The four phases on one timeline, with the legally compelled same-day actions in red so that reporting and safety are never displaced by the therapy that follows them.

- **The one interview rule.** Every repetition costs accuracy and re-traumatises. Take one account in the child's own words, record it verbatim, and leave the forensic interview to the trained examiner rather than repeating it yourself.

WHAT EARNS THE MARKS

- **Safety and reporting placed before clinical work,** as the plate orders them.
- **Medical and psychiatric assessment kept as separate strands.**
- **The non-abusing caregiver named as the protective factor,** and worked with.
- **Trauma-focused cognitive therapy named,** not therapy in general.

SEE ALSO Slot III - 26 management under the POCSO Act Slot III - 21 long-term consequences **SPRINT** Day 24, Child psychiatry

III - 26

Discuss management of a child presenting with history of sexual abuse, especially in the background of the POCSO Act. [10]

10 | ETHICS, CONSENT AND CHILD PROTECTION | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The duty that starts it**	Mandatory reporting, and the penalty for silence
4 **The statutory steps**	In order, with who acts and by when
2 **Clinical care alongside**	It runs in parallel, never afterwards
2 **Child-friendly protections**	The provisions written to spare the child

- **Where it begins.** The Act makes reporting mandatory for any person who knows or apprehends that a sexual offence against a child has been committed, and failure to report is itself punishable. It is gender neutral, and a child is anyone below eighteen.

STEP	WHAT HAPPENS	WHO ACTS	TIME LIMIT OR CONDITION
1	Information given to the police or the child welfare unit, recorded in simple language	Any person who knows, doctor included	**A duty, not a discretion**
2	Child produced before the welfare committee, care and protection arranged	Police and Child Welfare Committee	Within twenty four hours
3	Medical examination, with a woman doctor for a girl and a trusted adult present	Registered medical practitioner	Free, and possible without a police report
4	Statement recorded by the Magistrate, at the child's home or a place of his choice	Magistrate, with a support person	**Recorded once, not repeatedly**
5	Trial in a designated Special Court, in camera, the child never facing the accused	Special Court	Evidence and trial are time-bound

The Act presumes the accused's culpable mental state, which reverses the usual burden; that is a feature of the statute and not a matter for the doctor's opinion.

- **Clinical care, in parallel.** Injury and infection treatment, emergency contraception where indicated, acute distress and self-harm risk, then trauma-focused cognitive behavioural therapy, work with the non-abusing caregiver, and school liaison. Court preparation is part of treatment, not separate from it.

WHAT THE ACT DOES NOT COVER

It does not make the doctor an investigator: no opinion on whether the offence occurred, no repeated interviewing, and no promise of confidentiality once reporting is triggered. Adolescent consensual activity below eighteen also falls within the Act, which is a recognised and unresolved difficulty.

WHAT EARNS THE MARKS

- **Mandatory reporting named first,** with the offence of not reporting.
- **The steps in order, with the actor at each.**
- **Clinical care shown running in parallel,** not after the legal process.
- **The child-friendly provisions,** which is where the Act earns its name.

SEE ALSO Slot III - 28 features of the POCSO Act **SPRINT** Day 24, Child psychiatry

III - 27

Juvenile justice act. [10]

10 ETHICS, CONSENT AND CHILD PROTECTION

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Two children, two systems	In conflict with law, and in need of care
2	The two bodies	One for each category, and who sits on them
4	The steps	Apprehension to disposal, including the 2015 change
2	The psychiatrist's part	The preliminary assessment, and what it is not

- **Two categories, two systems.** A child in conflict with law, who is below eighteen and alleged to have committed an offence, goes to the Juvenile Justice Board, a magistrate sitting with two social workers. A child in need of care and protection goes to the Child Welfare Committee. The Act's premise is care and reintegration, not punishment.

STEP	WHAT HAPPENS	WHO ACTS	CONDITION
1	Child apprehended and produced, never lodged in a police lock-up or jail	Special juvenile police unit	**Within twenty four hours**
2	Inquiry into the offence, the child kept out of the adult system	Juvenile Justice Board	Ordinarily completed in four months
3	For a heinous offence by a child of sixteen to eighteen, a preliminary assessment	Board, with a psychologist or expert	**Capacity, understanding of consequences, circumstances**
4	Board may transfer such a case to be tried as an adult	Children's Court	Only after that assessment
5	Disposal: counselling, community service, group home, or a place of safety	Board or Court	No death penalty, no life without release

The 2015 Act replaced the 2000 Act mainly to create this transfer route after public pressure; the same Act also carries the adoption provisions and the central adoption authority.

- **The preliminary assessment.** It asks whether the child had the mental and physical capacity to commit the offence, the ability to understand its consequences, and the circumstances in which it happened. It is not a trial, not a finding of guilt, and not a psychiatric diagnosis.

WHAT THE ACT DOES NOT COVER

Sexual offences against children, which go to the POCSO Act 2012 and its Special Court, and mental illness in the child, which goes to the Mental Health Care Act 2017. Nor does it validate the assessment it asks for: there is no established instrument for judging a sixteen year old's criminal maturity.

WHAT EARNS THE MARKS

- **The two categories separated at the start,** each with its own body.
- **The sixteen to eighteen transfer route,** which is the Act's defining feature.
- **The three limbs of the preliminary assessment.**
- **An honest word on its weak evidence base.**

SEE ALSO Slot III - 09 adoption **SPRINT** Day 24, Child psychiatry

III - 28

Define child sexual abuse. Discuss important features of the POCSO Act and its implication in psychiatric practice. [3+4+3]

3 + 4 + 3 | ETHICS, CONSENT AND CHILD PROTECTION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Definition	Contact and non-contact, and the two conditions
4	The Act's spine	Six features, each with what it changes
2	For the psychiatrist	What the Act requires of you specifically
1	The unresolved part	One honest limit, named

- **Child sexual abuse.** The involvement of a child in sexual activity that he does not fully comprehend, cannot give informed consent to, or is not developmentally prepared for, and which violates the law or the social taboos of society. It covers contact and non-contact acts, and consent is legally impossible below eighteen.

FEATURE	WHAT THE ACT DOES	WHAT IT CHANGES IN PRACTICE
Gender neutral, under eighteen	Protects any child from any offender	Boys are covered, which older law did not do
Graded offences	Penetrative, aggravated, sexual assault, harassment, pornography	Severity tracks the act and the offender's position
Mandatory reporting	Any person must report; silence is an offence	**Overrides clinical confidentiality**
Child-friendly procedure	Support person, statement recorded once, in camera trial	Reduces repeated retelling
Presumption of guilt	Culpable mental state presumed of the accused	**Reverses the ordinary burden**
Special Court, time-bound	Designated court, evidence and trial within fixed periods	Faster than the general system, in principle

- **For the psychiatrist.** Report on first suspicion; take one account in the child's words and record it verbatim; give no opinion on whether the offence occurred; prepare the child for court as part of treatment; and tell the family at the outset that confidentiality does not survive a disclosure of abuse.

WHAT THE ACT DOES NOT SETTLE

Consensual sexual activity between adolescents falls within it, so a sixteen year old's relationship is an offence, and clinicians are drawn into reporting where no exploitation exists. Courts and law reform bodies have repeatedly noted the problem, and it remains unresolved.

WHAT EARNS THE MARKS

- **A definition with both conditions,** comprehension and developmental readiness.
- **Mandatory reporting named as overriding confidentiality.**
- **The reversed burden,** which few candidates know.
- **The adolescent difficulty,** stated honestly rather than avoided.

SEE ALSO Slot III - 26 management under the Act SPRINT Day 24, Child psychiatry

III - 29

Discuss the sexual boundary violations and nonsexual boundary violations that are important in clinical practice in Psychiatry. [5+5]

5 + 5 | ETHICS, CONSENT AND CHILD PROTECTION | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Crossing and violation	The distinction the whole answer rests on
4	The non-sexual domains	Where they occur, with an example in each
2	Sexual violations	Absolute, and why the rule outlasts discharge
2	Prevention	The slippery slope, and what interrupts it

THE DISTINCTION

A boundary crossing is a departure from the usual frame that is brief, non-exploitative and may even help: seeing a distressed patient beyond the hour. A boundary violation exploits the patient and serves the therapist's needs. Crossings are judged by context; violations never are.

DOMAIN	THE VIOLATION	WHY IT HARMS
Role	Treating a friend or relative; becoming the patient's business partner	**Objectivity and the right to refuse both go**
Money and gifts	Accepting valuable gifts, loans, investment tips	Creates obligation the patient cannot refuse
Time and place	Repeated late sessions, meetings outside the clinic	The frame that makes therapy safe dissolves
Self-disclosure	Confiding personal problems to the patient	**Reverses the direction of care**
Confidentiality	Using the material socially, or in teaching without consent	Trust cannot be repaired once lost

The amber rows carry the marks because they show the mechanism: a violation is defined by whose needs are served, not by the act's outward appearance.

- **Sexual violations.** Absolutely prohibited, whatever the patient's apparent consent, because the power difference makes consent meaningless. The prohibition does not lapse on discharge; a relationship with a former psychotherapy patient remains unethical, and the conduct is professional misconduct before the State Medical Council.
- **Prevention.** Violations arrive gradually, so the interruption is early: notice the exception being made, document it, keep supervision and personal therapy available, and treat the wish to make an exception for one patient as the signal it is.

WHAT EARNS THE MARKS

- **Crossing separated from violation in the first line.**
- **Non-sexual violations given by domain,** which is the five-mark half.
- **The rule surviving discharge,** stated explicitly.
- **The gradual course,** which is what makes prevention possible.

SEE ALSO Slot III - 22 principles of ethics SPRINT Day 26, Forensic ethics, consent and legal procedure

III - 30

What are the principles of Ethics? Describe their scope and application in mental health. [4+3+3]

4 + 3 + 3 | ETHICS, CONSENT AND CHILD PROTECTION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	The principles	Four, plus the derived duties, each defined
3	Scope	Where they reach, and where the law takes over
2	Application	Four situations peculiar to this specialty
1	The limit	What principles cannot do on their own

THE PRINCIPLES

Autonomy, the right to decide for oneself. Beneficence, acting for the patient's good. Non-maleficence, above all doing no harm. Justice, fairness in distributing benefit and burden. From these follow veracity, fidelity and confidentiality, which are duties rather than principles.

WHERE IT APPLIES	THE MENTAL HEALTH QUESTION	WHAT GOVERNS IT HERE
Treatment decisions	Whether the patient may refuse	**Capacity, not diagnosis, decides**
Involuntary admission	Autonomy overridden to prevent harm	Statutory grounds, review, least restriction
Confidentiality	Family, employer, and a third party at risk	**Consent first; disclosure only as far as needed**
Research	Enrolling someone who cannot consent	Representative, assent, ethics committee
Resource use	Who gets the scarce bed or the long appointment	Need, not articulacy or ability to pay

Scope: principles guide, statute compels. Where the Mental Healthcare Act 2017 speaks, it settles the matter; where it is silent, the principles do the work.

- **The limit.** Principles do not rank themselves. They frame the argument and constrain the answer, but the decision in a hard case is made by reasoning through the conflict and recording that reasoning, which is also what protects the clinician afterwards.

WHAT EARNS THE MARKS

- **Four principles defined, not merely listed,** with the derived duties named as derived.
- **Scope answered as a boundary with the law,** which is what scope means here.
- **Applications specific to psychiatry,** not general medical examples.
- **The absence of a ranking,** and documentation as the practical answer.

SEE ALSO Slot III - 22 principles and clinical implications **SPRINT**

Day 26, Forensic ethics, consent and legal procedure

AREA 04 OF 13 · P3-A05

Childhood emotional, tic, attachment and elimination disorders

10	10.2%	III-31	III-40
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 · HIGH 2 · RECURRENT 1 · SINGLE 6

REVISION ORDER

Enuresis first and hardest: four of the ten slots, and the pharmacology is the part most answers get wrong. School refusal next. Tourette, anxiety, the mother infant slot and pica after that.

WHAT IS IN THIS AREA

Enuresis in four slots, school refusal in two, Tourette syndrome, childhood anxiety disorders, mother infant relationship disorders, and pica.

III - 31

Etiology, clinical features, differential diagnosis and treatment of Gilles de la Tourette syndrome. [10]

10 ASKED AT 7 MARKS

CHILDHOOD EMOTIONAL, TIC, ATTACHMENT AND ELIMINATION DISORDERS

DNB, KNRUHS, TN-MGRMU - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2 **Aetiology**	Genetic, circuit and chemical, in that order
3 **Clinical features**	The criteria, the urge, the course, the company kept
2 **Differential diagnosis**	Movement, drug and functional causes separated
3 **Treatment**	The stepped plate: educate, behaviour, drug, refractory

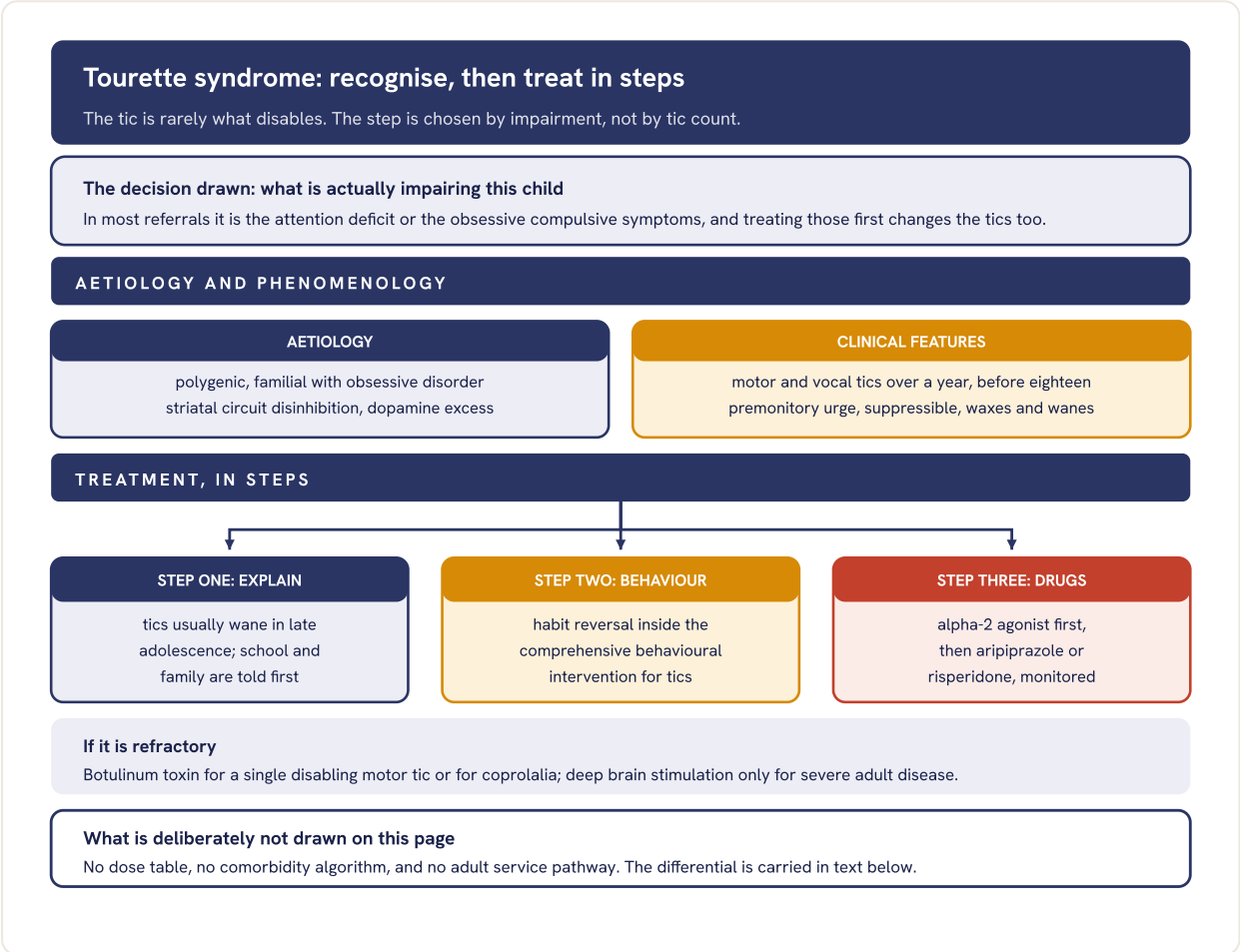


Fig 031.1 Aetiology and phenomenology in one band, then treatment as three steps, with the refractory options held back in a separate strip.

- **The company it keeps.** Attention deficit disorder in about half and obsessive compulsive symptoms in a third to a half, plus anxiety, rage outbursts, learning difficulty and poor sleep. Stimulants do not, on the evidence, worsen tics, so a coexisting attention deficit disorder should still be treated.

- **Differential diagnosis.** Provisional and persistent tic disorders, which do not meet the duration or the combination. Stereotypies, which are rhythmic and start earlier. Chorea in rheumatic fever or Wilson disease. Dystonia and myoclonus. Akathisia and tardive dyskinesia from antipsychotics. Compulsions, which are preceded by a thought rather than a bodily urge. Rapid-onset functional tic-like movements in adolescents.

PITFALL

Suppressibility is not proof of voluntariness. A child who holds tics through a clinic appointment and releases them at home has not been misbehaving, and telling a family otherwise is the commonest harm done in this condition.

WHAT EARNS THE MARKS

- **The premonitory urge named,** with suppressibility explained rather than treated as control.
- **A differential that crosses categories:** movement disorder, drug induced, and functional.
- **Behaviour therapy placed before medication,** with the named intervention.
- **The stimulant point.** Withholding stimulants for fear of tics is the classic wrong answer.

SEE ALSO Slot III - 19 stereotypic movement disorder Slot III - 50 management of attention deficit disorder **SPRINT** Day 23, Child behavioural, emotional & other disorders

III - 32

Define school refusal. What are the various causes of school refusal? Discuss the psychiatric aspects of management of a child presenting with the problem of school refusal. [2+4+4]

2 + 4 + 4 | CHILDHOOD EMOTIONAL, TIC, ATTACHMENT AND ELIMINATION DISORDERS

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	Four elements, and the line that excludes truancy
4 Causes	Child, school and family, each with an example
3 Management	The flowchart: treat, plan, return, hold
1 The urgency rule	One line on why time matters here

THE DEFINITION

Severe difficulty attending school, with marked emotional distress at the prospect of going, the child remaining at home with the parents' knowledge, and no significant antisocial behaviour. That last element separates it from truancy, where the absence is concealed and the child is elsewhere.

- **Causes, in three groups.** Child: separation anxiety in the younger, social anxiety and panic in the older, depression, specific phobia, and undiagnosed learning disorder or autism producing daily failure. School: bullying, punishment, examination pressure, an unfamiliar language of instruction. Family: parental anxiety or depression, overprotection, marital conflict, illness at home.

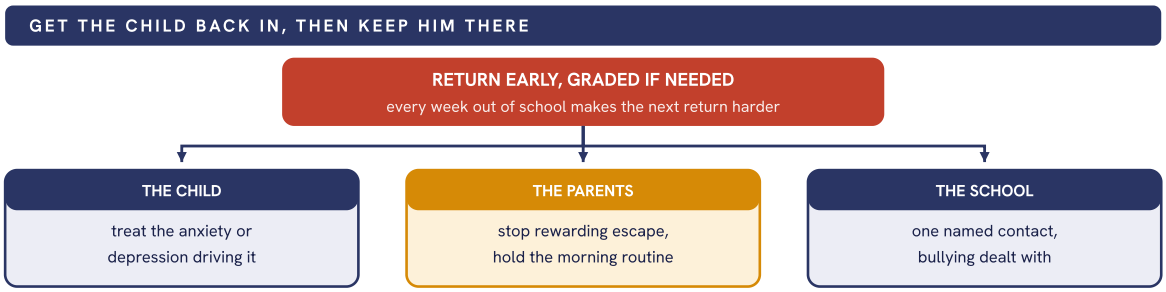


Fig 032.1 Early return drawn as the red first step, then the three parties who each own one part of the plan.

- **The psychiatric part.** Cognitive behavioural therapy with parent sessions alongside, graded exposure built into the return timetable, and a serotonin reuptake inhibitor where anxiety or depression is severe. Home schooling without a dated re-entry plan entrenches the problem.

WHAT EARNS THE MARKS

- **All four elements of the definition,** with truancy excluded by name.
- **Causes grouped as child, school and family,** not listed as one sequence.
- **Early return stated as the principle** before any therapy is described.
- **The parent behavioural work.** Treating only the child is the commonest incomplete answer.

SEE ALSO Slot III - 33 evaluation of school refusal Slot III - 35 childhood anxiety disorders **SPRINT** Day 23, Child behavioural, emotional & other disorders

III - 33

Discuss the evaluation of a child presenting with school refusal. [10]

10 CHILDHOOD EMOTIONAL, TIC, ATTACHMENT AND ELIMINATION DISORDERS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Frame the problem	Separate refusal from truancy and from withholding
5 The five sources	Child, parent, school, examination, instruments
2 Formulate the function	What the absence achieves for this child
1 Risk	The two enquiries that are never delegated

FRAME IT BEFORE YOU ASSESS IT

Three things look alike at the door. School refusal: the child stays home, the parents know, and there is distress. Truancy: the absence is concealed and the child is elsewhere. School withholding: the parents keep the child at home for their own reasons. The assessment differs completely, so the frame comes first.

SOURCE	WHAT TO COVER	WHAT IT SETTLES
The child, alone	Morning symptoms, fears, mood, sleep, self harm, bullying, abuse	**The private history the parent will not hear**
The parents	Timeline, what happens each morning, what the child does at home	Reinforcement, and secondary gain
The school	Attendance record, marks, peer group, a named teacher's account	**Whether the trigger is inside the school**
Examination	Physical review of the headache and abdominal pain	Rules out illness without endless tests
Instruments	Anxiety and depression rating scales; learning assessment where marks have fallen	Severity, and a baseline to measure

- **Formulate the function.** Four functions are described: avoiding what provokes distress at school, escaping social or evaluative situations, gaining attention at home, and pursuing something more rewarding outside. The first two are anxiety driven and the last is closer to truancy, and the function chosen decides the plan.
- **Risk, never delegated.** Ask the child directly and privately about self harm and about abuse. A sudden refusal in a previously well child is a safeguarding question until it is answered, and a child protection referral runs under the Juvenile Justice Act 2015 where neglect or abuse is found.

WHAT EARNS THE MARKS

- **The three-way frame at the start.** Refusal, truancy, withholding.
- **The child interviewed alone,** written as a step rather than assumed.
- **A named school contact treated as a data source,** not as an afterthought.
- **The function formulated,** because it is what turns an assessment into a plan.

SEE ALSO Slot III - 32 causes and management Slot III - 35 childhood anxiety disorders **SPRINT** Day 23, Child behavioural, emotional & other disorders

III - 34

What is enuresis? Classify enuresis. Describe the management of enuresis in a 15-year-old child and in a 70-year-old adult? [1+2+3+4]

1 + 2 + 3 + 4 | CHILDHOOD EMOTIONAL, TIC, ATTACHMENT AND ELIMINATION DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1	**Definition**	Frequency, duration, age, and the exclusion
2	**Classification**	Three axes: timing, primary or secondary, symptoms
3	**The adolescent**	Reassess first, then alarm, then a drug
4	**The older adult**	A different problem with a different framework

DEFINITION AND CLASSIFICATION

Repeated voiding into the bed or clothes, at least twice a week for three months or causing distress, at a chronological or developmental age of at least five, and not due to a substance or medical condition. Three axes: nocturnal, diurnal or both; primary if never dry for six months, secondary if dryness was lost; monosymptomatic, or with daytime symptoms.



Fig 034.1 The two ages split at the top because they are different problems. Only the left lane is enuresis as the definition above uses the word.

- **The adolescent.** After reassessment: fluid timing, void before bed, treat constipation. An alarm is first line and has the best sustained response. Desmopressin gives quick control for a camp or an examination, with fluid restricted around the dose. An anticholinergic if the bladder is overactive; imipramine last, with cardiac caution.
- **The older adult.** Work the reversible causes: delirium, infection, atrophic change, diuretics and sedatives, excess output from heart failure or diabetes, poor mobility, stool impaction. Then prostate, overactive bladder, dementia. Timed voiding, pelvic floor work, environment and aids come before any drug. Anticholinergics carry cognitive risk, and desmopressin is avoided over sixty-five.

WHAT EARNS THE MARKS

- **The definition with all four elements,** and the three classification axes named as axes.
- **The statement that the seventy year old does not have enuresis.** That one line is what the question is testing.
- **Alarm placed first for the adolescent,** desmopressin positioned as short term control.
- **Drug cautions specific to old age:** anticholinergic cognitive risk, and low sodium with desmopressin.

SEE ALSO Slot III - 36 treatment in detail Slot III - 38 aetiology **SPRINT**

Day 23, Child behavioural, emotional & other disorders

III - 35

Classify childhood anxiety disorders. Discuss principles of management of each of them. [4+6]

4 + 6 | CHILDHOOD EMOTIONAL, TIC, ATTACHMENT AND ELIMINATION DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The classification	Named, and what sits just outside the group
2	The common core	What every one of them is treated with first
4	Disorder by disorder	The specific twist each one needs
2	Medication	When it is added, which drug, and for how long

DISORDER	THE CORE FEAR	THE SPECIFIC TWIST IN MANAGEMENT
Separation anxiety	Loss of the attachment figure	**Graded separations, parent coached**
Selective mutism	Speaking where it is evaluated	**Shaping; never pressure to speak**
Specific phobia	One object or situation	Graded exposure, often one long session
Social anxiety	Scrutiny and humiliation	Exposure plus social skills
Panic and agoraphobia	The bodily sensations	Interoceptive, then situational
Generalised anxiety	Uncertainty across domains	Worry exposure, problem solving

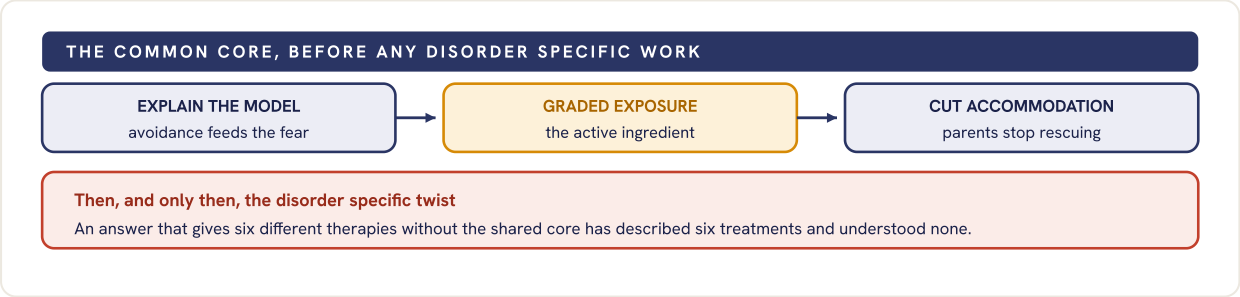


Fig 035.1 Three shared steps run before any disorder specific work, which is why the table's last column is a twist rather than a separate treatment.

- **Medication.** A serotonin reuptake inhibitor, usually fluoxetine or sertraline, when the disorder is moderate to severe or therapy alone has failed. Start low, review within a fortnight for activation and suicidal thinking, continue six to twelve months after response. Benzodiazepines are avoided in children.

WHAT EARNS THE MARKS

- **The classification named,** with obsessive compulsive and post-traumatic disorders placed outside the group but managed on the same exposure principle.
- **The shared core stated once** before the disorder by disorder section.
- **Selective mutism handled correctly.** Shaping, never pressure to speak, is the discriminating detail.
- **Drug placed second line with a duration attached.**

SEE ALSO Slot III - 32 school refusal Slot III - 33 evaluation of school refusal **SPRINT**

Day 23, Child behavioural, emotional & other disorders

III - 36

What is nocturnal enuresis? Describe its pharmacological and non-pharmacological management in detail. [2+4+4]

2 + 4 + 4 | CHILDHOOD EMOTIONAL, TIC, ATTACHMENT AND ELIMINATION DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it is	Definition, natural course, and the three mechanisms
4	Non-pharmacological	General measures, then the alarm protocol in full
3	Pharmacological	Each drug with its rule and its risk
1	Relapse	What is done when it comes back

WHAT IT IS, AND WHY IT HAPPENS

Bedwetting at or beyond five years, at least twice a week for three months or causing distress. A substantial proportion remit each year without treatment. Three mechanisms combine: more urine at night than the bladder holds, a small functional capacity with an overactive detrusor, and a high arousal threshold.

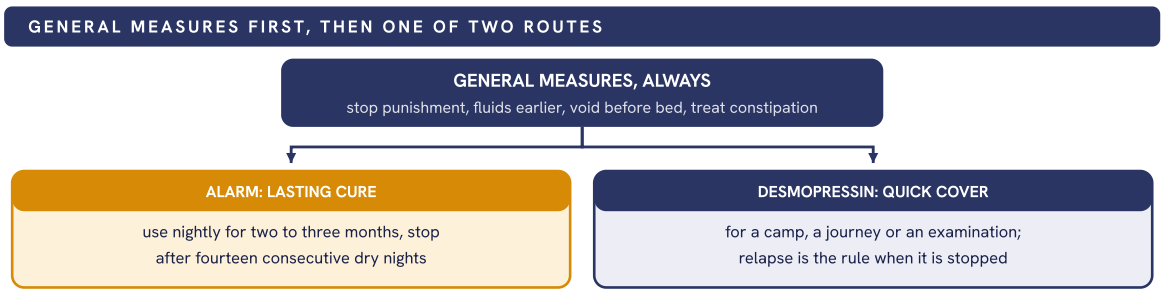


Fig 036.1 The two routes answer different questions. The alarm is chosen for a lasting result, desmopressin for cover on a known date, and the pair can be combined when either alone fails.

- **The alarm, in detail.** It conditions waking to bladder fullness. Reward the behaviours the child controls, getting up and resetting it, never a dry night. Overlearning, extra fluid at bedtime once dry, reduces relapse. A family too tired to wake with it is the commonest reason it fails.
- **The drugs, with their rules.** Desmopressin in the evening, with fluid restricted from an hour before to eight hours after, and withheld during vomiting or diarrhoea, because water intoxication is the serious harm. An anticholinergic where daytime urgency suggests an overactive bladder. Imipramine last, with cardiac review and a real overdose risk in the home.

WHAT EARNS THE MARKS

- **The three mechanisms named,** because each treatment maps onto one of them.
- **The alarm protocol with its stopping rule,** not just the word alarm.
- **Rewarding behaviour rather than dry nights.** It is the detail that shows the method is understood.
- **The fluid restriction with desmopressin,** and the reason for it.

SEE ALSO Slot III - 38 aetiology and management Slot III - 40 choosing the first treatment **SPRINT**

Day 23, Child behavioural, emotional & other disorders

III - 37

Describe the clinical features and management of mother infant relationship disorders. [10]

10 CHILDHOOD EMOTIONAL, TIC, ATTACHMENT AND ELIMINATION DISORDERS

KUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What the disorder is	The dyad is the patient, not the mother alone
4 Clinical features	On the mother's side and on the infant's side
3 Management	The flowchart: safety, mother, dyad, support
1 Safeguarding	The threshold at which the law is involved

PATTERN	IN THE MOTHER	IN THE INFANT
Emotional rejection	Delayed or absent warmth, no pleasure in the baby, avoidance	Gaze avoidance, reduced social smile, frozen watchfulness
Hostility	Anger at the infant, thoughts or threats of harm	Distress on approach; injuries requiring explanation
Anxious overinvolvement	Hypervigilance, constant checking, cannot let others hold	Feeding and sleep disturbance, irritability
Neglect	Care given mechanically or not at all	Poor weight gain, developmental delay



Fig 037.1 The safety question comes first because it decides whether the infant may be left unsupervised, and only then do the three arms of treatment begin.

- **Assessment.** Watch a feed and a period of play rather than relying on report. Rate maternal mood and bonding on the standard questionnaires. Ask about the pregnancy being wanted, pressure over the infant's sex, violence at home, and who else can help.
- **Safeguarding.** Where there are thoughts of harm, the infant is not left alone with the mother until the risk is managed, and admission with the infant is arranged where a facility exists. Significant neglect makes this a child in need of care and protection under the Juvenile Justice Act 2015.

WHAT EARNS THE MARKS

- **The dyad named as the patient.** An answer that only treats maternal depression has answered a different question.
- **Infant signs described,** not merely implied from the mother's behaviour.
- **Observation of a feed as the method,** and the safeguarding threshold with its statute.

SEE ALSO Slot III - 100 adverse childhood experiences Slot III - 98 temperament

III - 38

Etiology and management of enuresis. [4+6]

4 + 6 | CHILDHOOD EMOTIONAL, TIC, ATTACHMENT AND ELIMINATION DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Primary aetiology	Genetic and physiological, the three mechanisms
2	Secondary causes	Medical and psychological, each with its clue
4	Management by cause	Treat the cause found, then the symptom
2	The general programme	What every child gets whatever the cause

CAUSE	THE CLUE THAT POINTS THERE	WHAT IT CHANGES
Genetic and maturational	A wet parent, primary and monosymptomatic	Reassure, then the general programme
Constipation	Infrequent hard stools, soiling, a palpable mass	**Treat it and a third become dry**
Sleep disordered breathing	Snoring, mouth breathing, large tonsils	**Refer; surgery can resolve the wetting**
Urinary or metabolic	Dysuria, thirst, weight loss, poor stream	**Urine test and glucose before anything else**
Psychological	Secondary onset after a birth, a move, a loss or abuse	Address the event, not only the bed
Neurological	Sacral dimple, gait change, continuous dribbling	Imaging and specialist referral



Fig 038.1 Cause before symptom, with the general programme running underneath the whole sequence rather than at the end of it.

- **Primary aetiology.** Strongly familial, with concordance far higher in identical twins. Three mechanisms combine: a blunted night-time rise in vasopressin so more urine is made, a small functional bladder capacity with an overactive detrusor, and a raised arousal threshold so a full bladder does not wake the child. Attention deficit disorder and developmental delay are associated.

WHAT EARNS THE MARKS

- **The three mechanisms named,** because the treatments map onto them one for one.
- **Constipation identified as the highest-yield reversible cause.**
- **Secondary onset treated as a signal,** with abuse included in what is asked about.
- **Cause before symptom, stated as the order of work.**

SEE ALSO Slot III - 36 treatment in detail Slot III - 40 choosing the first treatment SPRINT
Day 23, Child behavioural, emotional & other disorders

III - 39

PICA. [10]

10 CHILDHOOD EMOTIONAL, TIC, ATTACHMENT AND ELIMINATION DISORDERS

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Definition**	Four criteria, and the cultural exclusion
2 **Who gets it, and why**	The populations, then the causes behind them
3 **Complications**	What the substance does once swallowed
3 **Assessment and management**	Investigate, correct, then change the behaviour

THE DEFINITION

Persistent eating of non-nutritive, non-food substances for at least a month, inappropriate to developmental level, and not part of a culturally supported or socially normative practice. Where it occurs alongside another disorder such as intellectual disability, autism or pregnancy, it is diagnosed separately only when severe enough to warrant clinical attention in its own right.

SUBSTANCE	WHERE IT POINTS	WHAT IT DOES
Soil and clay	Iron deficiency, poverty, cultural practice	**Worm infestation, lead, obstruction**
Ice	Iron deficiency anaemia	Dental damage; resolves with iron
Paint and plaster	Old housing, unsupervised toddler	**Lead poisoning, itself causing disability**
Hair	Trichotillomania, intellectual disability	**Trichobezoar, obstruction, surgery**
Paper, cloth, chalk	Understimulation, neglect, autism	Obstruction, malnutrition

- **Assessment.** Developmental and dietary history, who supervises the child, and what is available to eat. Examine for pallor and an abdominal mass. Check haemoglobin, ferritin and zinc, a lead level where exposure is plausible, and stool for ova and parasites. Image the abdomen if obstruction is suspected.
- **Management.** Correct iron and zinc, deworm, and remove the source: lead abatement, supervision, and enough food. Then behavioural work: reinforcing an alternative, discrimination training, blocking the response, and enriching the environment. No drug treats pica. Under national programmes, iron and folic acid supplementation and periodic deworming already reach many of these children.

WHAT EARNS THE MARKS

- **The cultural exclusion in the definition.** Geophagia that is normative in a community is not a disorder.
- **The one month duration and the developmental age rule.**
- **Complications tied to the specific substance,** not listed as one general set.
- **Deficiency corrected before behaviour work,** because in many children that alone ends it.

SEE ALSO Slot III - 19 stereotypic movement disorder Slot III - 12 aetiology of intellectual disability SPRINT Day 23, Child behavioural, emotional & other disorders

III - 40

Causes and treatment of Nocturnal Enuresis. [10]

10 CHILDHOOD EMOTIONAL, TIC, ATTACHMENT AND ELIMINATION DISORDERS

RGUHS, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	The first split	Monosymptomatic or not, and why it decides everything
3	Causes	The three mechanisms, then the red flags
4	Choosing the treatment	Which child gets the alarm, which gets the drug
1	Review	When to stop, and when to refer onward

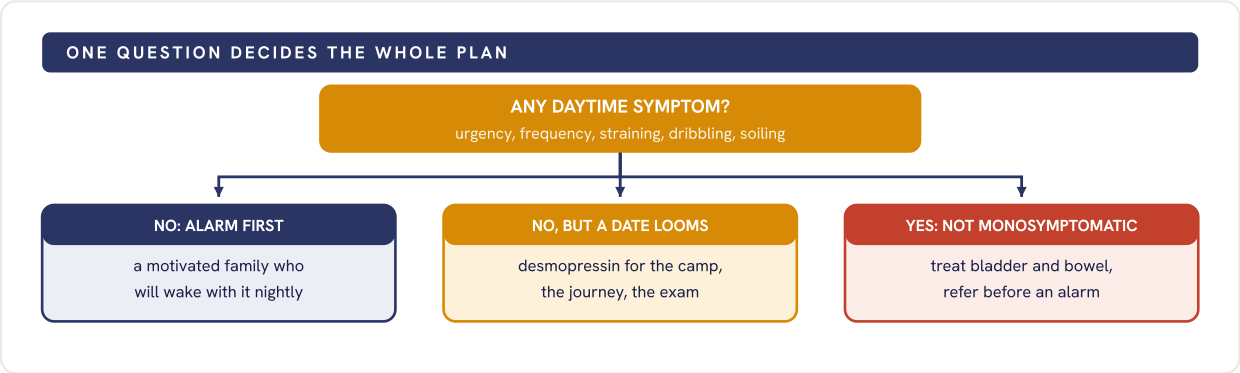


Fig 040.1 The daytime question is asked first because an alarm started in a child with an overactive bladder or constipation fails, and the failure is then blamed on the child.

- **Causes, in one line each.** More urine at night than the bladder holds, from a blunted night-time vasopressin rise. A small functional capacity with an overactive detrusor. A high arousal threshold. Layered on those: constipation, urinary infection, sleep disordered breathing, diabetes, and a psychological trigger where the wetting is secondary.
- **Red flags that redirect.** Continuous dribbling, a poor stream, a sacral dimple or hair tuft, gait change, new daytime wetting after years of dryness, weight loss with thirst, or any concern about abuse. These go to paediatrics, urology or safeguarding before any enuresis treatment begins.

WHAT EARNS THE MARKS

- **The monosymptomatic question asked first,** with the reason it comes first.
- **Alarm and desmopressin matched to different children,** rather than ranked against each other.
- **The red flag list, with where each one goes.**
- **Family capacity treated as part of the choice.** An alarm nobody wakes for is not a treatment.

SEE ALSO Slot III - 36 treatment in detail Slot III - 38 aetiology and management **SPRINT**
Day 23, Child behavioural, emotional & other disorders

AREA 05 OF 13 · P3-A07

Mental health legislation and disability law

9	9.6%	III-41	III-49
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 9

REVISION ORDER

The Act is five of the nine slots, so read the Act itself once rather than five summaries of it. Then RPWD 2016. Then the two disability assessment slots, which reuse the certification material from the intellectual disability area.

WHAT IS IN THIS AREA

The Mental Healthcare Act 2017 across five slots (advance directive, nominated representative, capacity and admission, the definition clauses, admission of a minor), the Rights of Persons with Disabilities Act 2016, disability assessment and IDEAS.

III - 41

Critically analyze the various aspects of Mental Health Care Act 2017. [10]

10 MENTAL HEALTH LEGISLATION AND DISABILITY LAW

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	What it replaced	The 1987 Act, the treaty behind it, the rights charter
3	The decision machinery	Capacity, advance directive, nominated representative
2	Admission and oversight	Four routes, Boards in place of Magistrates
1	The critique	What is enacted but not yet delivered

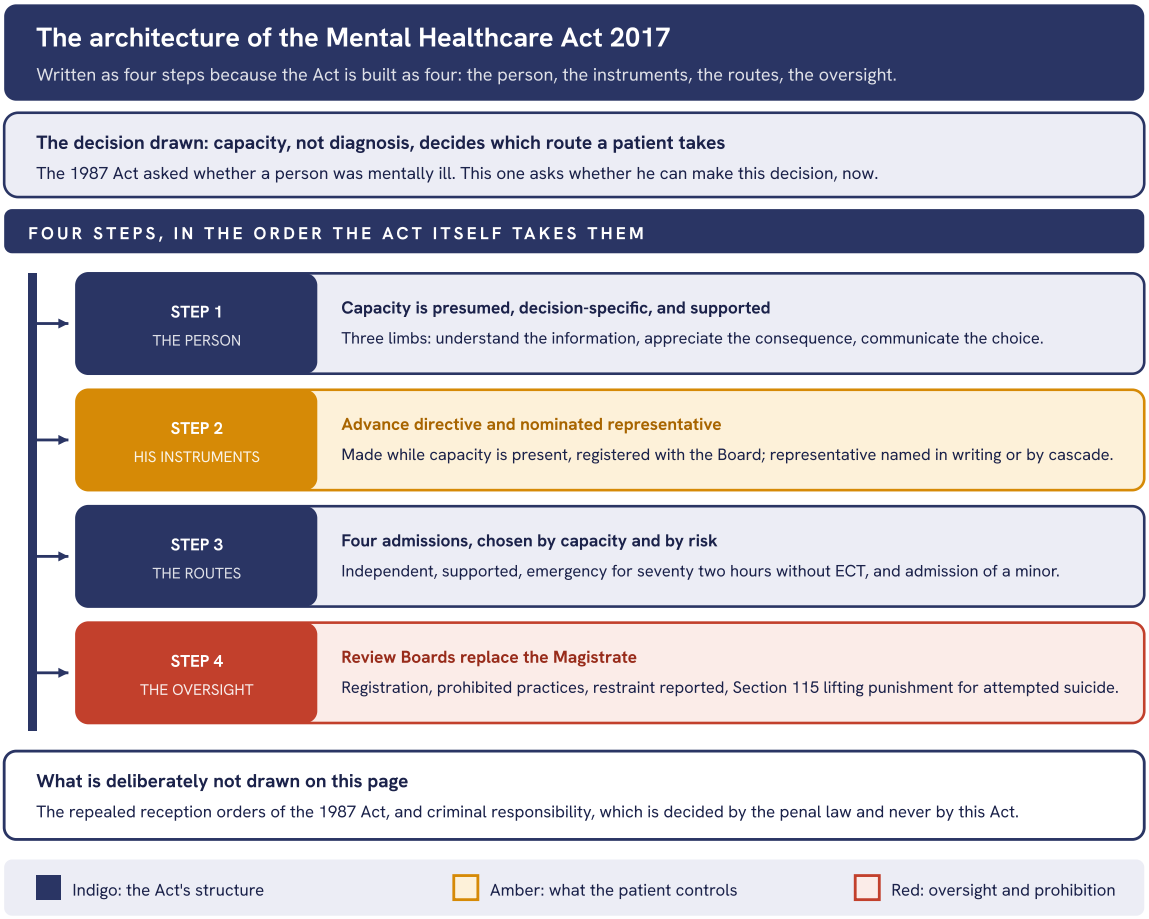


Fig 041.1 The Act drawn as the four steps it takes in its own order, with the two instruments the patient controls kept in amber.

- **What it gets right.** It converts a custodial statute into a rights statute: care as an entitlement, community living, confidentiality, free care for the poor, and insurance cover for mental illness on the same terms as physical illness.
- **Where it falls short.** Boards are unconstituted or under-resourced in many States, the advance directive register is largely unbuilt, capacity assessment is demanded of services without the staff for it, and insurance parity has needed repeated prodding.

WHAT THE ACT DOES NOT COVER

Intellectual disability by itself, excluded from the definition of mental illness and left to the Rights of Persons with Disabilities Act 2016. Criminal responsibility, which the penal law decides. And no right to any particular treatment.

WHAT EARNS THE MARKS

- **Capacity named as the organising idea,** not a list of chapters.
- **Both instruments the patient controls,** the directive and the representative.
- **A real critique, not a summary.** The stem says critically, and the implementation gap answers it.

SEE ALSO Slot III - 43 capacity at admission and discharge Slot III - 46 the advance directive **SPRINT** Day 25, Mental health legislation and forensic psychiatry

III - 42

Define nominated representative and his/her role in mental health care as defined in the MHCA, 2017. Discuss challenges in managing a wandering mentally ill patient, where nominated representative is not known or available. [2+2+6]

2 + 2 + 6 | MENTAL HEALTH LEGISLATION AND DISABILITY LAW

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Who the representative is	Appointed in writing, or the statutory cascade
2	What the role carries	Support the decision, do not replace the person
4	The wandering patient	The steps, in order, with the actor at each
2	The challenges	Identity, collateral, consent, and afterwards

- **Nominated representative.** A person appointed in writing by an adult to support his mental healthcare decisions. Where none is appointed, the Act supplies a cascade: the person named in an advance directive, then a relative, then a caregiver, then a person appointed by the Board.
- **The role.** To support, not to substitute: consider the person's expressed wishes, consent where he cannot, receive information, apply for supported admission, seek Board review, and prefer community care.

STEP	WHAT HAPPENS	WHO ACTS	TIME LIMIT OR CONDITION
1	Person found wandering at large and unable to care for himself is taken under protection	Officer in charge of the police station	**Section 100 duty, not a discretion**
2	Conveyed to the nearest public mental health establishment for assessment	Police officer	**Within twenty four hours**
3	Assessment of mental illness, capacity and immediate risk	Psychiatrist at the establishment	On arrival
4	No representative traceable, so one is appointed for the patient	Mental Health Review Board	On the establishment's application
5	Treatment on the supported route, or emergency treatment meanwhile	Psychiatrist with the appointed representative	Thirty days, then Board review

The amber cells are the two an examiner looks for: the police duty is mandatory, and it is time-bound.

WHAT THE ACT DOES NOT COVER

Tracing identity: there is no statutory mechanism, so the work falls to photographs, missing-person records and voluntary bodies. Nor does the Act fund long stay for the unclaimed, or settle repatriation across States.

WHAT EARNS THE MARKS

- **The cascade written out in order,** not merely the phrase appointed in writing.
- **Support rather than substitution,** stated as the character of the role.
- **Police duty and its twenty four hour limit,** with the word Section used for the provision.
- **The Board naming a representative** as the answer to the missing one.

SEE ALSO Slot III - 41 the Act as a whole **SPRINT** Day 25, Mental health legislation and forensic psychiatry

III - 43

Discuss capacity assessment and its implications during admission and discharge of psychiatric patients as per MHCA, 2017. [5+5]

5 + 5 | MENTAL HEALTH LEGISLATION AND DISABILITY LAW

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What capacity is	Three limbs, presumed until assessed
3	How it is assessed	Decision-specific, time-specific, supported first
3	At admission	Which route the finding opens, and for how long
2	At discharge	Who may leave, and what may briefly hold them

- **Capacity, in three limbs.** The ability to understand the information relevant to the decision, to appreciate any reasonably foreseeable consequence of taking it or not taking it, and to communicate the decision by any means. All three are needed, and each is judged for this decision at this time.

STEP	WHAT HAPPENS	WHO ACTS	TIME LIMIT OR CONDITION
1	Capacity is presumed and support is offered before any conclusion	The treating team	Until assessed otherwise
2	The three limbs are tested against this specific decision	Psychiatrist	**Recorded with the reasons**
3	Capacity present: independent admission on the person's own request	The person himself	Discharge on request
4	Capacity absent plus a stated risk ground: supported admission	Nominated representative, two assessments	**Up to thirty days**
5	Continuation beyond that period, or any dispute	Mental Health Review Board	Board review before extension

The risk grounds are stated, not assumed: recent harm or threat of harm to self, violence to others, or an inability to care for oneself that places one at risk.

- **At discharge.** An independent patient may leave on request, subject only to a brief statutory hold while the establishment assesses whether the supported criteria are met. A supported patient is discharged when capacity returns or the risk ground lapses, with a discharge plan.

WHAT THE ACT DOES NOT COVER

Capacity is not global, not permanent, and not a diagnosis. The Act does not permit incapacity to be inferred from the illness, from an unwise choice, or from refusal of the recommended treatment; and it gives no power to detain an independent patient who retains capacity.

WHAT EARNS THE MARKS

- **All three limbs named,** with the decision-specific and time-specific qualifiers.
- **The finding tied to a route,** so capacity is shown to have a consequence.
- **Risk grounds stated separately from incapacity.** Supported admission needs both.
- **Refusal distinguished from incapacity,** as the exclusions strip does.

SEE ALSO Slot III - 45 admission routes compared **SPRINT** Day 25, Mental health legislation and forensic psychiatry

III - 44

Disability assessment in mental illness. [10]

10 MENTAL HEALTH LEGISLATION AND DISABILITY LAW

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The frame	Impairment, activity limitation, participation restriction
2	The statute	RPWD 2016, the schedule, and the benchmark
4	The instrument	IDEAS: four domains, duration, conversion
2	What follows	The certificate, the card, the entitlements

THE DEFINITION

Disability assessment quantifies the loss of function that a mental illness causes, not the illness itself. The frame is the international classification of functioning: impairment of the mind, limitation of activity, and restriction of participation. Forty per cent or more is a benchmark disability under the Rights of Persons with Disabilities Act 2016.

DOMAIN SCORED	WHAT IS RATED	SCALE
Self care	Hygiene, dressing, feeding, health upkeep	Nought, none, to four, profound
Interpersonal activities	Relations within the family and outside it	Nought to four
Communication and understanding	Comprehension, expression, everyday reasoning	Nought to four
Work	Employment, housework or schooling, as applies	Nought to four
Duration of illness	Added to the four domain scores	A separate score, then converted

The amber row is the step most candidates omit: the four domain scores are summed, the duration score is added, and only then is the total converted to a percentage by the notified table.

- **Who certifies.** A medical authority notified by the State, in practice a board with a psychiatrist, issuing a certificate with a stated validity and a review date where improvement is expected.
- **What it unlocks.** The unique disability identity card, reservation in higher education and government employment, income tax relief, travel concession, and access to limited guardianship rather than the plenary guardianship the Act abolished.

WHAT EARNS THE MARKS

- **Function separated from diagnosis** in the opening line.
- **All four IDEAS domains, plus duration as a fifth added score.**
- **Forty per cent named as the benchmark,** with the statute it comes from.
- **The entitlements,** which are the reason the assessment is requested at all.

SEE ALSO Slot III - 48 IDEAS and the RPWD categories Slot III - 47 the RPWD Act in psychiatry SPRINT Day 25, Mental health legislation and forensic psychiatry

III - 45

a) What is mental illness and who are mental health professionals as per the MHCA, 2017? b) How the admission and discharge procedures as per the MHCA, 2017 are different from Mental Health Act,1987? c) What are the guidelines for ECT in the MHCA, 2017? [2+2+4+2]

2 + 2 + 4 + 2 | MENTAL HEALTH LEGISLATION AND DISABILITY LAW

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Two definitions**	What is in, what is expressly out, who qualifies
4	**The routes, then and now**	Route by route, with the 1987 equivalent named
2	**What actually changed**	Capacity, the Board, and discharge on request
2	**ECT**	Three rules, one of them absolute

- **Two definitions.** Mental illness is a substantial disorder of thinking, mood, perception, orientation or memory that grossly impairs judgment, behaviour, capacity to recognise reality or the ability to meet the ordinary demands of life. It includes conditions associated with alcohol and drug abuse, and excludes intellectual disability. A mental health professional is a psychiatrist, psychologist, psychiatric social worker or nurse registered with the State Authority.

ROUTE UNDER THE 2017 ACT	WHO APPLIES	DURATION	ITS 1987 PREDECESSOR
Independent admission	The person himself, having capacity	Until he asks to leave	Voluntary admission
Supported admission	Representative, two assessments	**Thirty, then ninety, then a hundred and twenty days**	Special circumstances; reception order
Emergency treatment	Any registered medical practitioner	Seventy two hours, and no ECT	No true equivalent
Admission of a minor	Nominated representative only	Reported to and reviewed by the Board	Admission by the guardian

What changed: capacity replaced diagnosis, the Board replaced the Magistrate, and the independent patient gained a right to leave.

- **ECT, three rules.** Unmodified ECT, without muscle relaxant and anaesthesia, is prohibited absolutely. ECT for a minor requires both the guardian's informed consent and the Board's prior permission. ECT can never be given as emergency treatment.

WHAT THE ACT DOES NOT COVER

It sets no upper limit on the number of treatments, prescribes no technique or electrode placement, and regulates private practice only through registration of the establishment. Those are matters of clinical guideline, not of statute.

WHAT EARNS THE MARKS

- **The express exclusion of intellectual disability** from the definition.
- **Routes matched to their 1987 predecessors,** which is what the question asks.
- **The three ECT rules, with the absolute one named as absolute.**

SEE ALSO Slot III - 49 admission of a minor **SPRINT** Day 25, Mental health legislation and forensic psychiatry

III - 46

What is advance directive? Discuss the concept with critically examining its utility and limitations. What is the legal status of advance directive in Psychiatry in India. [2+4+4]

2 + 4 + 4 | MENTAL HEALTH LEGISLATION AND DISABILITY LAW

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	A choice recorded while capacity is present
3 The steps	Make, register, invoke, review, revoke
3 Utility and limits	Both sides, because the stem says critically
2 Legal status in India	Statutory since 2018, and distinct from a living will

- **Advance directive.** A written statement by an adult, made while he has the capacity to make mental healthcare decisions, saying how he wishes to be cared for and treated if he later loses that capacity, and who shall be his nominated representative.

STEP	WHAT HAPPENS	WHO ACTS	CONDITION
1	The directive is written and signed	The person, having capacity	**Capacity at the time of making**
2	It is registered and made available	Mental Health Review Board	Held on the Board's register
3	It governs treatment once capacity is lost	Treating psychiatrist	A duty to follow it
4	It may be reviewed, altered or cancelled on application	The Board, on any party's application	**The Board may override it**
5	It may be revoked or amended by the maker	The person himself	Only while capacity is present

It does not apply to emergency treatment, so a directive refusing admission cannot block seventy two hours of emergency care.

- **Utility.** It extends autonomy across an episode, reduces family conflict at the worst moment, records a preference between agents already tried, and suits relapsing illness.
- **Limits.** Registers are largely unbuilt, awareness is low, capacity at the time of making is rarely documented, the Board can set it aside, and it cannot demand a treatment that is not indicated.

WHAT THE ACT DOES NOT COVER

It is not a living will. End-of-life refusal of life-sustaining treatment rests on Common Cause, 2018, and the procedure the Supreme Court simplified in 2023; the psychiatric advance directive is statutory, and the two should never be run together in an answer.

WHAT EARNS THE MARKS

- **Capacity at the time of making,** named as the condition that validates it.
- **Both sides argued,** because the stem says critically.
- **The emergency exception,** which is the commonest omission.
- **Separated from the living will,** with the Indian authority for each.

SEE ALSO Slot III - 42 the nominated representative SPRINT Day 25, Mental health legislation and forensic psychiatry

III - 47

Discuss the Rights of Persons with Disabilities Act 2016 in relation to Psychiatry. [10]

10 | ASKED AT 15 MARKS | MENTAL HEALTH LEGISLATION AND DISABILITY LAW

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it is for	The treaty behind it, and the 1995 Act it replaced
3	Who it covers	Twenty one conditions; five of them psychiatric
3	The rights that bite	Legal capacity, healthcare, education, employment
2	Certification and redress	Benchmark, authority, Commissioner, offences

- **Why it matters to psychiatry.** It gives the treatment of chronic mental illness a legal destination beyond symptom control: certification, entitlement and enforceable non-discrimination. Passed to give effect to the disability rights convention, it replaced the 1995 Act and widened the schedule from seven conditions to twenty one.

PROVISION	WHAT IT GIVES	WHERE IT BITES IN PRACTICE
The schedule	Mental illness, intellectual disability, specific learning disability, autism and chronic neurological conditions	**Five of the twenty one are ours**
Legal capacity	Equal recognition before law, with support	**Limited guardianship replaces plenary**
Healthcare	Free or affordable care, insurance, early detection	Grounds a claim, not merely a policy
Education and employment	Free education to eighteen, reservation in higher education and in government posts	Applies at benchmark disability
Enforcement	Chief and State Commissioners, penalties for atrocity and for false certification	A forum the 1995 Act lacked

Benchmark disability is forty per cent or more of a specified condition, certified by the notified medical authority, and it is the threshold most entitlements are keyed to.

WHAT THE ACT DOES NOT COVER

It is not a treatment statute: admission, capacity to consent to treatment and involuntary care remain with the Mental Healthcare Act 2017. It does not displace the National Trust Act 1999 for guardianship in autism, cerebral palsy and intellectual disability, and it creates no right to a cure.

WHAT EARNS THE MARKS

- **The psychiatric entries counted out of the twenty one.**
- **Limited guardianship named as the change,** not just legal capacity as a phrase.
- **Forty per cent tied to the entitlements it unlocks.**
- **The boundary with the 2017 Act,** as the exclusions strip draws it.

SEE ALSO Slot III - 44 disability assessment in mental illness Slot III - 48 IDEAS and the RPWD categories **SPRINT** Day 25, Mental health legislation and forensic psychiatry

III - 48

What is IDEAS, its domains and scoring? Discuss the various categories of psychiatric disorders included as per the Rights of Persons with Disability Act? [5+5]

5 + 5 | MENTAL HEALTH LEGISLATION AND DISABILITY LAW

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What IDEAS is	Who made it, what it measures, why it exists
3	Domains and scoring	Four domains, duration added, then converted
3	The RPWD categories	The psychiatric entries in the schedule
2	The certificate	Benchmark, authority, validity, appeal

- **IDEAS.** The Indian Disability Evaluation and Assessment Scale, developed by the Indian Psychiatric Society and gazetted as the instrument for quantifying disability in mental illness. It rates current functioning, not symptoms, and it needs an illness that has been diagnosed and treated, not a single cross-sectional interview.

STEP	WHAT IS DONE	SCALE	CONDITION
1	Rate self care, interpersonal activities, communication and understanding, and work	Each nought to four	Current function, all four rated
2	Sum the four to a global disability score	Nought to sixteen	No domain omitted
3	Add the score for duration of illness	A further nought to four	**The step most often forgotten**
4	Convert the total by the notified conversion table	To a percentage	**Forty per cent is the benchmark**
5	Certificate issued, with validity and a review date	Notified medical authority	Appeal lies against the finding

The psychiatric entries in the schedule: mental illness; intellectual disability, which carries specific learning disability and autism spectrum disorder alongside it; and chronic neurological conditions, named there as multiple sclerosis and Parkinson's disease.

WHAT THE INSTRUMENT DOES NOT COVER

IDEAS is for mental illness only. Intellectual disability is quantified by intelligence and adaptive testing, and autism by the notified Indian scale, each with its own certification route. Using IDEAS for those is a procedural error that invalidates the certificate.

WHAT EARNS THE MARKS

- **All four domains named, in the scale's own words.**
- **Duration added as a separate score before conversion.**
- **The schedule entries listed,** not a general statement that mental illness is covered.
- **Instrument matched to condition,** as the exclusions strip does.

SEE ALSO Slot III - 44 disability assessment in mental illness **SPRINT**

Day 25, Mental health legislation and forensic psychiatry

III - 49

Admission of Minor as per Mental Health Care Act (MHCA) 2017. [10]

10 | ASKED AT 7 MARKS | MENTAL HEALTH LEGISLATION AND DISABILITY LAW

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Who is a minor here**	Under eighteen, and who represents him
4 **The steps**	Application, admission, report, review, discharge
2 **The protections**	Separate space, an attendant, and the ECT bar
2 **Why it is separate**	A minor cannot consent, so oversight replaces consent

- **The principle.** A minor cannot give the consent that makes an adult admission independent, so the Act does not ask him to. It substitutes an application by his nominated representative, who is his legal guardian unless the Board orders otherwise, and then supplies external review in place of the consent that is missing.

STEP	WHAT HAPPENS	WHO ACTS	TIME LIMIT OR CONDITION
1	Application for admission of the minor	Nominated representative, ordinarily the guardian	**No other route exists**
2	Two independent assessments support the need for inpatient care	Psychiatrist and a second professional	Care not possible in the community
3	Admission is reported to the Review Board	Medical officer in charge	**Within seventy two hours**
4	Continued stay is reviewed	Mental Health Review Board	Where the stay passes thirty days
5	Discharge on the representative's request, with a plan	Representative and treating team	Unless the Board directs otherwise

The two amber cells carry the marks: the application route is exclusive, and the clock for reporting starts at admission, not at review.

- **The protections.** The minor is accommodated separately from adults; a parent, guardian or suitable attendant stays with him, and a female attendant for a girl; and electroconvulsive therapy needs both the guardian's informed consent and the Board's prior permission.

WHAT THE ACT DOES NOT COVER

It does not create a minor's right to admit himself, and it does not answer the case of a child brought by one parent against the other's objection, which goes to the Board. Protection from abuse and neglect is the business of the Juvenile Justice Act 2015, not of this one.

WHAT EARNS THE MARKS

- **The exclusive application route,** stated before anything else.
- **Both time limits,** the report and the review of a longer stay.
- **The three physical protections,** which are cheap marks most answers skip.
- **The ECT bar for minors,** with both conditions named.

SEE ALSO Slot III - 45 admission routes compared Slot III - 27 the Juvenile Justice Act **SPRINT**

Day 25, Mental health legislation and forensic psychiatry

AREA 06 OF 13 · P3-A04

ADHD and disruptive behaviour disorders

9	9.5%	III-50	III-58
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 · HIGH 3 · RECURRENT 1 · SINGLE 4

REVISION ORDER

ADHD management first, it is the highest-scoring question in this book. Conduct disorder next, since three slots ask it three ways. Then the single-drug slot and the two newer diagnostic entities.

WHAT IS IN THIS AREA

ADHD management and neurobiology, adult ADHD, atomoxetine, conduct disorder in three slots, oppositional defiant disorder and disruptive mood dysregulation disorder.

III - 50

Discuss pharmacological and nonpharmacological management of Attention Deficit Hyperactivity Disorder. [10]

10 ADHD AND DISRUPTIVE BEHAVIOUR DISORDERS

DNB, KUHS, NTRUHS, RGUHS, TN-MGRMU - 12 APPEARANCES, 12 OF 78 SESSIONS

SKELETON

2	The principle	Age decides which arm goes first, severity decides speed
3	Non-drug management	Parent, classroom, child, in that order of evidence
3	Drug management	First line, second line, and what each is chosen for
2	Monitoring	What is measured at every visit, and what stops the drug

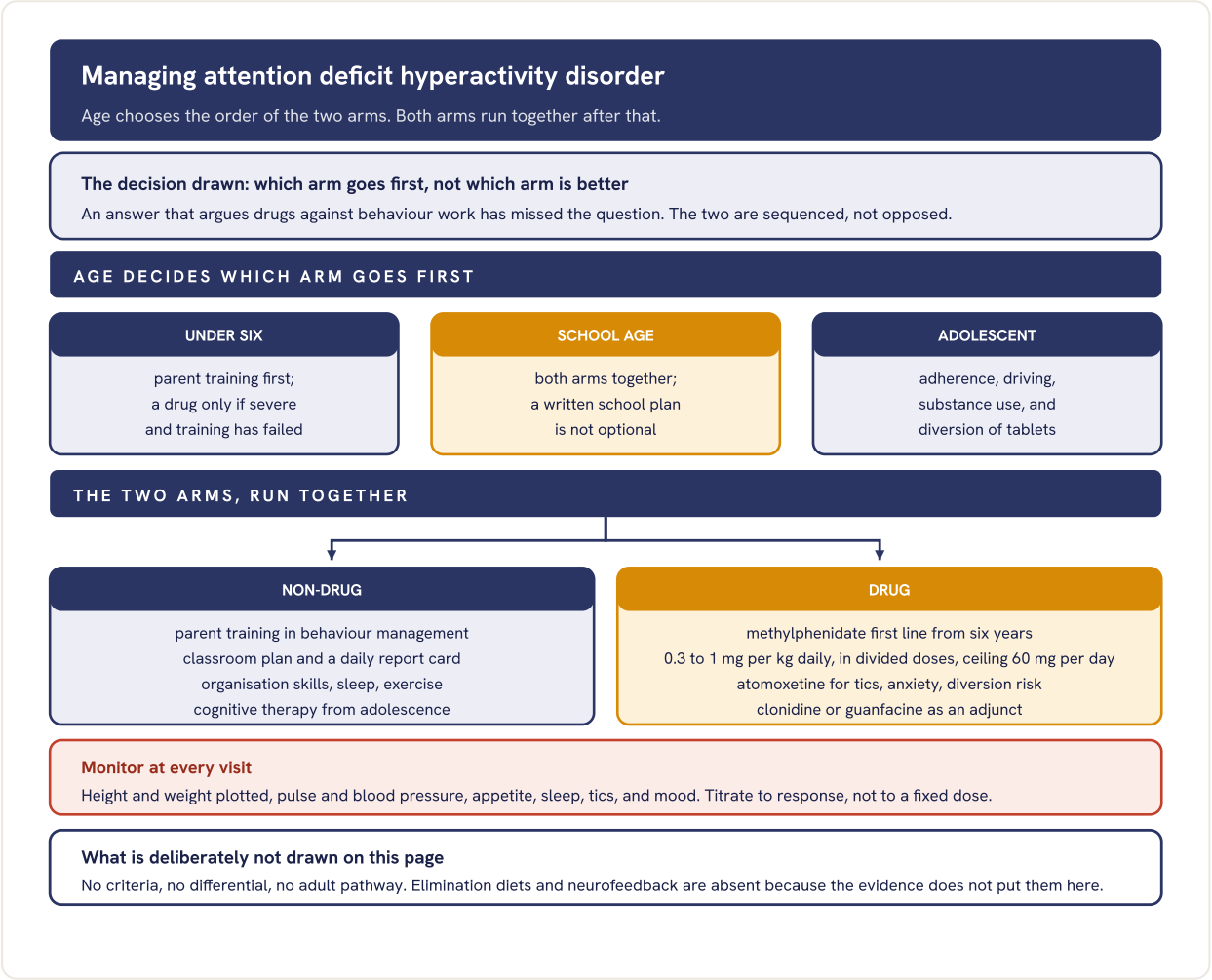


Fig 050.1 Age chooses the order, then both arms run together, with monitoring carried as a red strip because it is the part that is skipped.

- **Choosing between the drugs.** Stimulants act within days and have the larger effect. Atomoxetine takes two to six weeks, is not a controlled drug, and is preferred where there are tics, marked anxiety, or a diversion risk. Alpha-2 agonists help tics and sleep as an adjunct.
- **Indian practice.** Methylphenidate is a controlled substance, so prescribing follows the narcotic rules and long acting preparations are not reliably available. Large classes make a daily report card harder, raising the value of parent training.

PITFALL

A drug holiday is not a routine. It is considered only for growth faltering, and it costs the child the school terms it covers.

WHAT EARNS THE MARKS

- **Age stated as the sequencing rule** before either arm is described.
- **Both arms present.** An answer that is only pharmacology cannot pass a question that names both.
- **One weight-based dose band, with titration to response.**
- **The monitoring list.** Height, weight, pulse, blood pressure, appetite, sleep, tics, mood.

SEE ALSO Slot III - 53 atomoxetine Slot III - 54 adult attention deficit disorder Slot III - 55 neurobiology

III - 51

Discuss briefly the etiology, clinical features, prognosis and treatment of oppositional defiant disorder. [2+4+2+2]

2 + 4 + 2 + 2 | ADHD AND DISRUPTIVE BEHAVIOUR DISORDERS

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Aetiology	Child, parenting and context, named as an interaction
4	Clinical features	Three symptom clusters, the count, and the setting rule
2	Prognosis	Each cluster predicts a different adult outcome
2	Treatment	Parent first, child second, drugs almost never

- **Aetiology, as an interaction.** Difficult temperament and poor effortful control, moderate heritability, low verbal ability and coexisting attention deficit disorder, meeting harsh or inconsistent parenting. The coercive cycle described by Patterson is the mechanism: the child escalates, the parent gives way, and both are rewarded.
- **Clinical features.** Three clusters: angry and irritable mood; argumentative and defiant behaviour; vindictiveness. Four or more symptoms for at least six months, with someone who is not a sibling. Under five they occur on most days, from five at least weekly. Severity is graded by how many settings are affected.

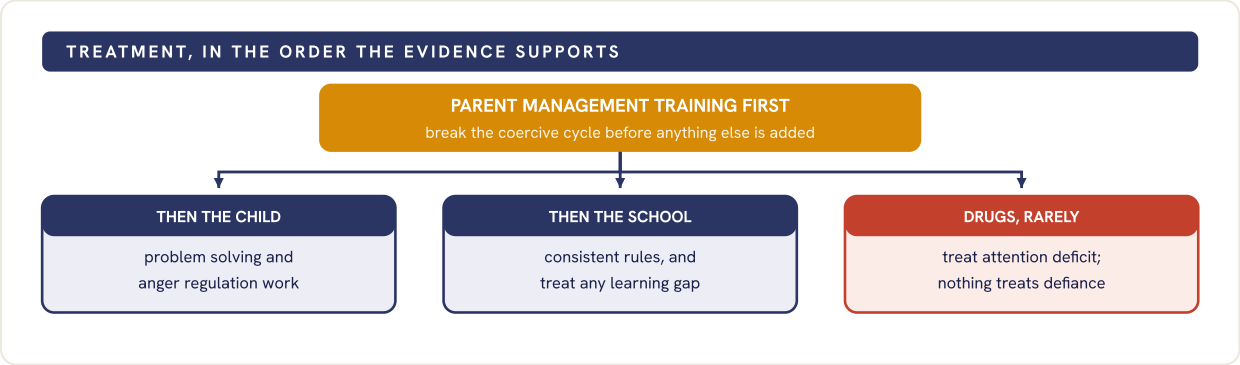


Fig 051.1 Parent work first, then the child and the school, with drugs as a red lane because they treat what coexists rather than the disorder itself.

PROGNOSIS, AND THE PITFALL INSIDE IT

Most children improve. The three clusters predict differently: the irritable cluster predicts later depression and anxiety, the headstrong cluster predicts attention deficit disorder and conduct problems, and the vindictive cluster carries the highest risk of conduct disorder. Treating all defiance as pre-conduct disorder misses the children whose outcome is a mood disorder.

WHAT EARNS THE MARKS

- **Aetiology written as an interaction,** with the coercive cycle named as the mechanism.
- **The count, the non-sibling rule and the age-based frequency.** These turn behaviour into a diagnosis.
- **Prognosis split by cluster,** not given as one figure for the whole disorder.

SEE ALSO Slot III - 57 conduct disorder Slot III - 56 disruptive mood dysregulation disorder **SPRINT**

Day 23, Child behavioural, emotional & other disorders

III - 52

Describe the course and outcome with management of Conduct disorders. [10]

10 ADHD AND DISRUPTIVE BEHAVIOUR DISORDERS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The two trajectories**	Age at onset is the strongest prognostic fact
3 **Outcome**	Named outcomes, and the honest proportion
3 **Management by age**	Who is the target of treatment at each stage
2 **What makes it worse**	The interventions that harm, and the legal interface

TRAJECTORY	WHAT IT LOOKS LIKE	WHERE IT TENDS TO GO
Childhood onset	Before ten years, aggression, often with attention deficit disorder	**Persistent; a minority reach antisocial personality disorder**
Adolescent onset	After ten, peer group offending, less aggression	**Most desist in early adult life**
Limited prosocial emotions	Callous, unremorseful, shallow affect	Worst outcome at any onset age
Girls	Relational aggression, later onset, more covert	More depression, anxiety and victimisation

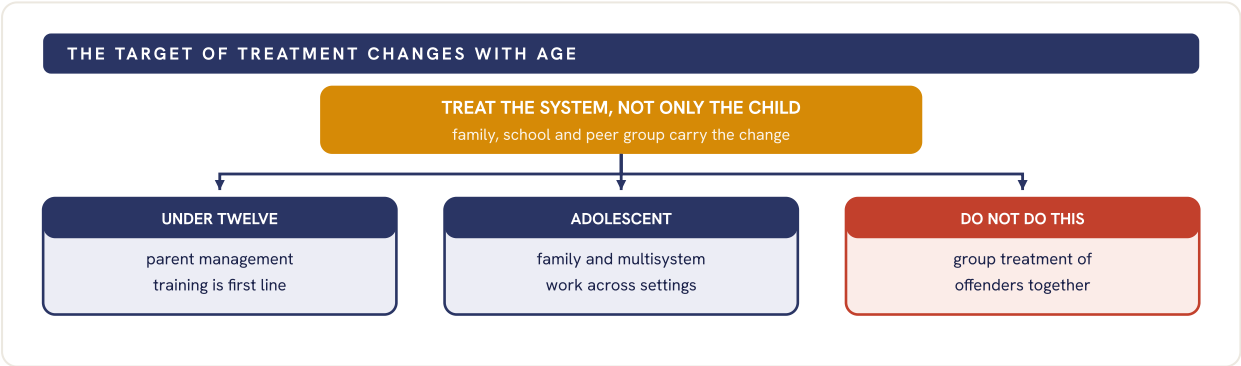


Fig 052.1 The target of treatment moves outward with age, and the red lane names the intervention that reliably makes outcomes worse.

- **Outcome, and the legal interface.** School dropout, substance use, injury, early parenthood, unemployment and offending. Most do not develop antisocial personality disorder, and saying so is a mark. A child in conflict with law is dealt with by the juvenile justice board under the Act of 2015, where the psychiatrist assesses and does not adjudicate.

WHAT EARNS THE MARKS

- **Age at onset named as the strongest prognostic fact,** before any outcome is listed.
- **The honest proportion.** A minority, not most, reach antisocial personality disorder.
- **Group treatment named as harmful,** and the juvenile justice role kept to assessment.

SEE ALSO Slot III - 57 features and aetiology Slot III - 58 definition and comorbidity **SPRINT**
Day 23, Child behavioural, emotional & other disorders

III - 53

Atomoxetine. [10]

10 ADHD AND DISRUPTIVE BEHAVIOUR DISORDERS

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What it is	Mechanism, and why the prefrontal effect matters
2	When it is chosen	The four situations that select it over a stimulant
4	Dose and adverse effects	Weight banded dosing, then the warnings
2	Monitoring	What is checked, and what stops the drug

WHAT IT IS

A selective noradrenaline reuptake inhibitor licensed for attention deficit hyperactivity disorder. Blocking the noradrenaline transporter also raises dopamine in the prefrontal cortex, where that transporter does most of the clearing. It is not a stimulant and not a controlled substance.

HEADING	WHAT TO WRITE	THE QUALIFIER
Chosen when	Tics, marked anxiety, substance misuse, or risk of tablets being diverted	Effect is smaller than a stimulant
Dose to 70 kg	Start 0.5 mg per kg daily, then target 1.2 mg per kg daily	Maximum 1.4 mg per kg or 100 mg
Dose over 70 kg	Start 40 mg daily, target 80 mg daily	Maximum 100 mg daily
Onset	Two to six weeks, full benefit later	Never used as needed
Common effects	Appetite loss, nausea, abdominal pain, sedation or insomnia, irritability	Usually settle with time

- **Stimulants and tics.** Stimulants are not absolutely contraindicated in tic disorders, so a coexisting attention deficit disorder is still treated, and atomoxetine is the preferred agent where tics are prominent or worsen on a stimulant.
- **The warnings.** Suicidal ideation in children and adolescents, especially in the first months. Rare liver injury, so stop for jaundice or dark urine. Small rises in pulse and blood pressure. Avoid in narrow angle glaucoma, phaeochromocytoma and serious cardiovascular disease, and within a fortnight of a monoamine oxidase inhibitor.
- **Monitoring and interactions.** Height and weight plotted, pulse and blood pressure, mood and suicidal thinking, and any liver symptom. Metabolised by cytochrome P450 2D6, so exposure rises with fluoxetine, paroxetine or bupropion, and in poor metabolisers.

WHAT EARNS THE MARKS

- **The prefrontal dopamine point.** It explains the effect without making it a stimulant.
- **Weight banded dosing with the two ceilings,** not a single adult figure.
- **The slow onset stated,** because it is why families stop the drug early.
- **Suicidality and liver injury named,** with what each one triggers.

SEE ALSO Slot III - 50 management of attention deficit disorder Slot III - 54 adult attention deficit disorder

III - 54

What are the manifestations of adult ADHD? How does it differ from childhood ADHD? Discuss its management in brief. [3+3+4]

3 + 3 + 4 | ADHD AND DISRUPTIVE BEHAVIOUR DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Adult manifestations	By domain, in the form adults actually present
3	The grid	Domain by domain, with the diagnostic rules marked
2	Non-drug management	Psychoeducation, skills, and the environment
2	Drug management	Sequencing around comorbidity, and what to watch

DOMAIN	IN CHILDHOOD	IN ADULT LIFE	WHAT SETTLES IT
Hyperactivity	Runs, climbs, leaves the seat	Inner restlessness, cannot sit through a meal or a film	Form changes, the trait persists
Inattention	Careless work, loses things, distracted	Procrastination, missed deadlines, disorganisation	**The domain that persists most**
Impulsivity	Blurts out, cannot wait a turn	Interrupts, impulsive spending, quits jobs, risky driving	Consequences, not the behaviour
Symptom count	Six or more in a domain	Five or more from seventeen years	**The threshold falls with age**
Onset evidence	Observed directly	Several symptoms before twelve, corroborated by an informant	**Retrospective, so seek records**
Company kept	Oppositional defiant disorder, learning disorder	Substance use, mood and anxiety disorder, personality difficulty	Changes the treatment order

- **Management, non-drug.** Psychoeducation for patient and partner, cognitive therapy adapted for adult attention deficit disorder, structured organisation and time management systems, environmental restructuring at work, sleep regularity, and coaching where available.
- **Management, drugs and order.** Methylphenidate is first line. Atomoxetine where there is substance misuse or a diversion risk. Stabilise an active substance disorder and any bipolar disorder before starting a stimulant, check cardiovascular history and blood pressure, and counsel on driving.

WHAT EARNS THE MARKS

- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **The two diagnostic rule changes marked:** five symptoms from seventeen, and onset before twelve.
- **Hyperactivity described as changing form,** not as disappearing.
- **Comorbidity sequenced before prescribing.** It is the line that separates a top answer.

SEE ALSO Slot III - 50 management of attention deficit disorder Slot III - 53 atomoxetine

III - 55

Neurobiology of attention deficit hyperactivity disorder. [10]

10 ADHD AND DISRUPTIVE BEHAVIOUR DISORDERS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Genetics and environment	Heritability, polygenic risk, the exposures that survive
3	Neurochemistry	Which transmitters, where, and how the drugs fit
3	Structure and networks	Maturation, volumes, and the circuits involved
2	Models and limits	Two or three pathways, then the honest caveat

THE ONE-LINE ACCOUNT

A highly heritable, polygenic disorder of catecholamine signalling in fronto-striatal and fronto-parietal circuits, expressed as delayed cortical maturation rather than as a lesion, with group differences that are real, small, and heavily overlapping with typical development.

LEVEL	THE FINDING	THE CAVEAT THAT MUST BE SAID
Genetic	Heritability around seventy to eighty percent, polygenic, many small effects	**No single gene, and no genetic test**
Environmental	Prematurity and low birth weight, lead, severe early deprivation	Prenatal smoking is largely confounded
Chemical	Dopamine and noradrenaline in prefrontal cortex and striatum	**An inverted U, so more is not better**
Structural	Delayed cortical maturation, small reductions in basal ganglia volume	Small effects, wide overlap
Network	Fronto-striatal, fronto-parietal, fronto-cerebellar; default mode intrusion	**Group level, never diagnostic**

- **How the drugs fit the biology.** Stimulants block the dopamine and noradrenaline transporters, atomoxetine blocks the noradrenaline transporter, and alpha-2A agonists strengthen prefrontal network signalling directly. All three converge on the same catecholamine deficit account.
- **Models, and the limit.** Executive dysfunction from weak behavioural inhibition, delay aversion from a hyposensitive reward system, and a timing pathway make up the dual and triple pathway models. No investigation is diagnostic, and the diagnosis remains clinical.

WHAT EARNS THE MARKS

- **Heritability quoted with the polygenic qualifier.** A number without it invites the wrong follow-up question.
- **Delayed maturation, not a lesion.** That phrase is what the question is testing.
- **Drugs mapped onto the chemistry,** which shows the biology is understood rather than listed.
- **The no-test caveat.** It is the last mark and most answers never reach it.

SEE ALSO Slot III - 50 management of attention deficit disorder Slot III - 16 neurodevelopmental disorders

III - 56

Disruptive Mood Dysregulation Disorder. [10]

10 ADHD AND DISRUPTIVE BEHAVIOUR DISORDERS

NTRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	The criteria	Outbursts, the mood between them, and every threshold
2	Why it exists	The problem it was written to solve
3	Differential	Four confusables, each with the deciding feature
2	Management	What the thin evidence actually supports

THE CRITERIA, WITH THE NUMBERS

Severe recurrent temper outbursts, verbal or behavioural, out of proportion to the provocation and to developmental level, on average three or more times a week. Between outbursts the mood is persistently irritable or angry, most of the day, nearly every day, and others can see it. Twelve months or more, never symptom free for three consecutive months, in at least two of three settings and severe in one. Onset before ten years; the diagnosis is not made before six or after eighteen.

CONFUSABLE	HOW IT DIFFERS	WHAT SETTLES IT
Bipolar disorder	Distinct episodes, with elation, reduced sleep need and grandiosity	Episodicity; here irritability is chronic
Oppositional defiant	Defiance directed at authority, outbursts less severe	Cannot be diagnosed alongside; this takes precedence
Intermittent explosive	Outbursts only, three months, mood normal in between	No irritability between episodes
Depression or attention deficit	Irritability confined to the mood episode, or driven by impulsivity	May coexist; both are allowed

- **Why it exists.** It was written to stop chronic non-episodic irritability being diagnosed as paediatric bipolar disorder. Follow-up studies show that this pattern predicts unipolar depression and anxiety in adult life rather than bipolar disorder, and treating it as bipolar exposed children to lithium and antipsychotics for years.
- **Management, honestly.** The evidence base is thin. Parent management training and anger regulation work first. Where attention deficit disorder coexists, a stimulant has the best evidence for the irritability itself. A serotonin reuptake inhibitor where depressive or anxiety features dominate. An antipsychotic only for severe aggression, briefly, with metabolic monitoring.

WHAT EARNS THE MARKS

- **Every threshold quoted:** three a week, twelve months, two settings, onset before ten, ages six to eighteen.
- **The mood between outbursts.** It is what separates this from every episodic diagnosis.
- **The exclusion with oppositional defiant disorder,** stated as precedence rather than as overlap.
- **The reason the category was created.** It is the two easiest marks on this page.

SEE ALSO Slot III - 51 oppositional defiant disorder Slot III - 57 conduct disorder

III - 57

Describe the clinical features, aetiology and management of Conduct Disorder. [10]

10 ADHD AND DISRUPTIVE BEHAVIOUR DISORDERS

NTRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	Clinical features	Four groupings, the count, and the two specifiers
3	Aetiology	Biological, family and social, as a chain not a list
3	Management	The algorithm: who is treated, at what age
1	Drugs	One line: what they treat, and what they do not

GROUPING	EXAMPLES FROM THE CRITERIA	THE RULE
Aggression to people and animals	Bullying, fights, weapon use, cruelty, robbery, forced sexual activity	Three or more criteria in twelve months
Destruction of property	Fire setting, deliberate damage	At least one in the past six months
Deceitfulness or theft	Breaking in, conning, shoplifting	Fifteen criteria in total
Serious rule violations	Staying out at night, running away, truancy before thirteen	Specify onset, and limited prosocial emotions

- **Aetiology, as a chain.** Heritable temperament with low autonomic arousal and reduced amygdala response to distress, plus weak verbal ability, meeting harsh parenting, maltreatment and poor supervision, and then deviant peers and school failure.

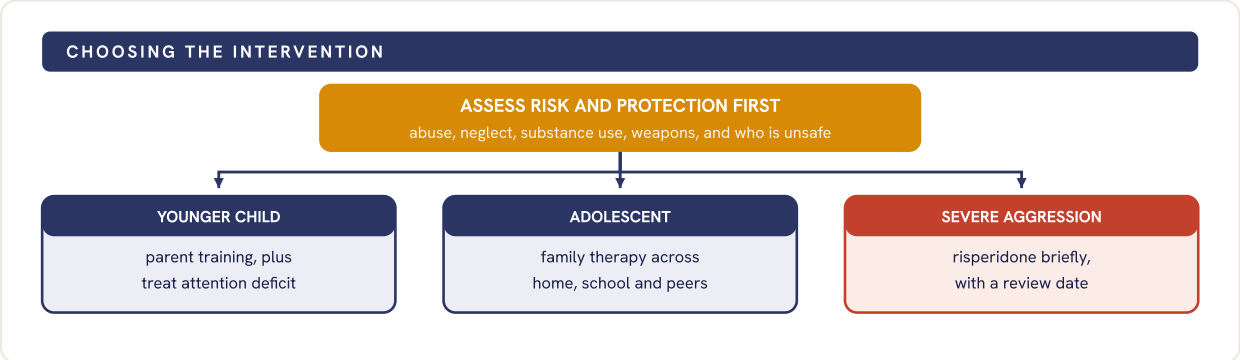


Fig 057.1 Risk assessed before treatment is chosen, then the intervention that matches the age, with medication as a red lane because it is symptomatic and temporary.

- **Drugs, in one line.** No drug treats conduct disorder. Stimulants treat coexisting attention deficit disorder and reduce aggression through it; risperidone reduces severe aggression short term with metabolic monitoring. Neither replaces the family and school work.

WHAT EARNS THE MARKS

- **Four groupings with the counting rule,** not fifteen behaviours in a flat list.
- **The limited prosocial emotions specifier named,** because it changes prognosis and treatment.
- **Aetiology written as a chain** from temperament through parenting to peers.
- **Risk assessment placed before treatment selection.**

SEE ALSO Slot III - 52 course and outcome Slot III - 58 definition and comorbidity **SPRINT**

Day 23, Child behavioural, emotional & other disorders

III - 58

Conduct disorders. [10]

10 ADHD AND DISRUPTIVE BEHAVIOUR DISORDERS

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	One sentence, with the duration and the count
3	Subtypes and specifiers	Onset, prosocial emotions, severity
3	Epidemiology and comorbidity	Rates as a range, then what travels with it
2	The Indian interface	Where the law meets the clinic, and our role in it

THE DEFINITION

A repetitive and persistent pattern of behaviour violating the basic rights of others or major age-appropriate norms, shown by three or more of fifteen criteria in the past twelve months, with at least one in the past six months, and causing clinically significant impairment.

AXIS	THE DIVISIONS	WHY IT MATTERS
Age at onset	Childhood onset before ten, adolescent onset, unspecified	The strongest prognostic split
Prosocial emotions	With limited prosocial emotions: remorse, empathy, concern, affect	Two of four, twelve months, more than one setting
Severity	Mild, moderate, severe, by number of criteria and harm caused	Sets intensity of the response
Comorbidity	Attention deficit disorder, learning disorder, substance use, depression, trauma	Each is separately treatable

- **Epidemiology.** Community estimates of a few percent of children and adolescents, with boys diagnosed more often than girls. Indian school and community surveys report a wide range because instruments and thresholds differ, so a range with its reason is the honest answer rather than a single figure.
- **The Indian interface.** A child in conflict with law goes to the juvenile justice board under the Act of 2015, and a neglected or abused child to the child welfare committee as a child in need of care and protection. The psychiatrist assesses capacity, development and mental illness. The board decides, not the clinician.

WHAT EARNS THE MARKS

- **The definition with three of fifteen, twelve months and six months.** Naming behaviours without the rule is a half answer.
- **Both specifiers,** with the two of four rule on prosocial emotions.
- **Prevalence given as a range with its reason.**
- **The legal boundary.** Assessing, not adjudicating, is the line that reads as command of the role.

SEE ALSO Slot III - 52 course and outcome Slot III - 57 features and aetiology SPRINT

Day 23, Child behavioural, emotional & other disorders

AREA 07 OF 13 · P3-A13

Geriatric, consultation-liaison and transcultural psychiatry

8	8.7%	III-59	III-66
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 · RECURRENT 1 · SINGLE 6

REVISION ORDER

Take the two CL model slots together, they overlap almost completely. Then the two sleep slots, likewise. Elder abuse, geriatric depression and the imbalance slot are independent and can go in any order.

WHAT IS IN THIS AREA

Geriatric psychiatry in five slots (elder abuse, imbalance and its medicolegal aspects, sleep in two slots, geriatric depression) and consultation-liaison psychiatry in three (the Indian model, the models in general, the unconscious patient brought by police). The registered name of this area also carries transcultural psychiatry, and no transcultural question survived selection into it. Nothing on the pages that follow is transcultural, and the area is described here by what it actually holds.

III - 59

What are the different types of elderly abuse and neglect? Discuss the management of elderly abuse. [3+7]

3 + 7 | GERIATRIC, CONSULTATION-LIAISON AND TRANSCULTURAL PSYCHIATRY

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	The types	All five groups, including the two usually missed
2	Recognition	The signs that should raise it unprompted
4	The pathway	Safety, capacity, then the branch that follows
1	The Act	What the 2007 Act actually provides

- **Where it happens.** Most elder abuse in India is domestic and by family, so the relative who brings her is not automatically an ally. Institutional abuse also occurs, and is under-recognised because the complainant depends on the institution.
- **What should raise it unprompted.** Injuries that do not match the account, delayed presentation, dehydration and weight loss, pressure sores, poor hygiene, unexplained loss of money or a sudden transfer of property, repeated attendances, a carer who answers for her, and fear in his presence.

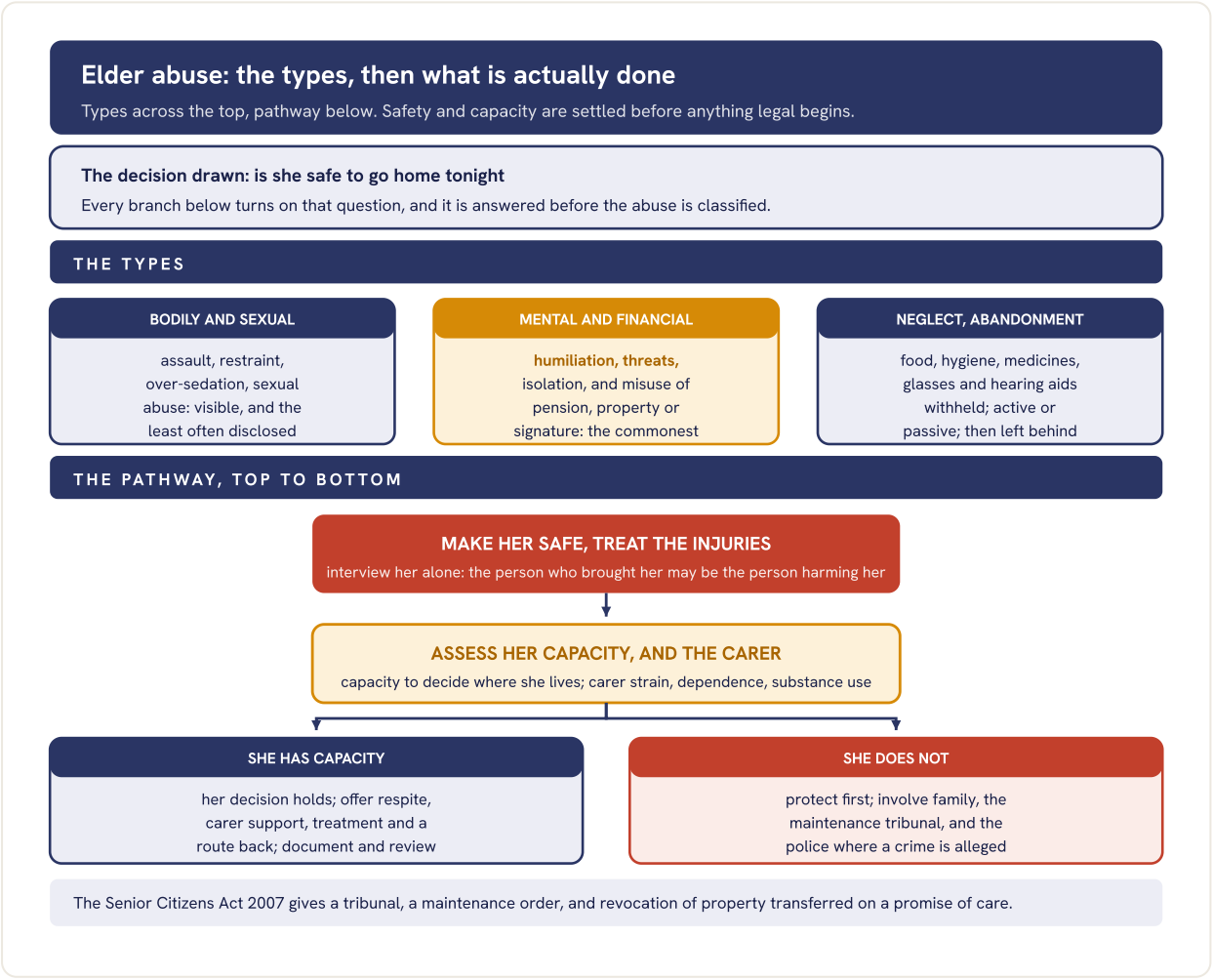


Fig 059.1 Types above, pathway below, with capacity as the single fork that decides everything after it.

PITFALL

Capacity is decision-specific and is presumed. An older person with capacity may choose to return to a household that is harming her, and the answer then is support, monitoring and an open route back, not removal. Treating a diagnosis of dementia as automatic incapacity is the commonest version of this error.

WHAT EARNS THE MARKS

- **All the types named,** financial abuse and neglect included, since those are the two most answers omit.
- **The alone interview written as a step,** with the reason given in the same line.
- **The Act named with what it actually provides,** not simply cited.

SEE ALSO Slot III - 61 medicolegal aspects of dementia Slot III - 66 geriatric depression **SPRINT** Day 29, consultation-liaison and geriatric psychiatry

III - 60

Elaborate on the model of consultation liaison psychiatry that is usually used in Indian set up. What are the common causes of referral from ICU to a psychiatrist for epilepsy patients? [6+4]

6 + 4 | GERIATRIC, CONSULTATION-LIAISON AND TRANSCULTURAL PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The Indian model	Named, then described in how it actually runs
3	Why that shape	Workforce, funding and referral culture, not preference
3	Referral causes	Delirium first, then drugs, then the psychiatric ones
1	The real question	What the team is actually asking for

THE MODEL IN INDIAN PRACTICE

The consultation model dominates: the psychiatrist is called after the problem is established, sees the patient once or twice, writes an opinion, and leaves the team to carry it. True liaison, where the psychiatrist joins the unit's rounds and sees patients before a crisis, survives only where a specialty funds it. The reason is workforce and funding, not preference. The taxonomy itself sits on III-65.

REFERRAL TRIGGER	WHAT IS USUALLY HAPPENING	WHAT TO CHECK	WEIGHT
Confusion after seizures	Postictal delirium, a prolonged postictal state, or non-convulsive status	Duration, fluctuation, electroencephalogram	**Non-convulsive status is the one that is missed**
Delirium from the illness	Sepsis, hyponatraemia, hepatic or renal failure, sedative or alcohol withdrawal	Sodium, cultures, drug and alcohol history	Delirium first, psychiatry second
Drug-related disturbance	Levetiracetam irritability and aggression, topiramate slowing, phenobarbitone sedation, steroid mood change	The drug chart against the symptom timeline	**The chart dates the symptom**
Psychiatric complication	Postictal psychosis, interictal depression, suicidality, functional seizures	Timing relative to the fit	Suicide risk is raised in epilepsy
Systems questions	Capacity and consent, breaking bad news, family distress, drug interactions	Enzyme induction, plasma levels	Half of intensive-care liaison work

- **The referral is often a proxy.** Teams ask for sedation when they mean distress, for capacity when they mean disagreement, and for depression when they mean pain. Clarifying the actual question is the first act of a good consultation.

WHAT EARNS THE MARKS

- **The Indian model named as the consultation model,** with the workforce reason attached rather than implied.
- **Delirium placed ahead of every psychiatric label** in the intensive-care setting.
- **The drug chart used as a diagnostic instrument,** with the agents named.

SEE ALSO Slot III - 65 models of consultation-liaison psychiatry Slot III - 62 the unresponsive patient **SPRINT**

Day 29, consultation-liaison and geriatric psychiatry

III - 61

What are the causes of imbalance in an elderly psychiatric patient? Discuss different medicolegal aspects of dementia. [4+6]

4 + 6 | GERIATRIC, CONSULTATION-LIAISON AND TRANSCULTURAL PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Imbalance: drugs**	Iatrogenic before neurological, every time
2	**Imbalance: the rest**	By system, ending with delirium itself
4	**The legal questions**	Each one separately: who assesses, who decides
2	**The gap**	What no Indian statute currently provides

- **Imbalance is iatrogenic before it is neurological.** Benzodiazepines, antipsychotics, tricyclics, anticholinergics and antihypertensives, with orthostatic hypotension as the mechanism most often missed. Review the whole chart, not the newest drug.
- **Then the rest, by system.** Neurological: parkinsonism including the drug-induced kind, cerebellar disease, normal pressure hydrocephalus, stroke, neuropathy, cervical myelopathy. Then vestibular, cardiac, metabolic (hyponatraemia on an antidepressant, hypoglycaemia, B12 and thyroid), sensory loss, sarcopenia, and delirium itself.

THE QUESTION	WHAT IS ASSESSED	WHO DECIDES	THE STANDARD
Testamentary capacity	Nature of the act, extent of the property, the claims of those who might benefit	Clinician advises, court decides	**Banks v Goodfellow, still the test**
Consent to treatment	Understand, retain, weigh, communicate, now	Treating clinician, recorded	Decision-specific, presumed
Property and affairs	Handling money, and vulnerability to undue influence	Civil court, on evidence	**Capacity and influence are separate**
Driving	Attention, reaction time, visuospatial ability, insight	Licensing authority, on advice	Advise, document, inform
Criminal responsibility	Unsoundness of mind at the time of the act	The court	The general exception, Penal Code into Sanhita

WHAT NO ACT COVERS

India has no general adult guardianship regime for acquired incapacity. The National Trust Act 1999 does not reach dementia. The advance directive under the Mental Healthcare Act 2017 must be made while capacity is retained, so it arrives too late for most people already diagnosed. The Senior Citizens Act 2007 gives maintenance, not treatment decisions. Families go to the civil court instead.

WHAT EARNS THE MARKS

- **Imbalance answered drugs first,** with orthostatic hypotension named as the mechanism.
- **Each legal question answered separately,** with its own assessor, decider and standard, rather than one paragraph about capacity.
- **The statutory gap named.** Listing provisions is the competent answer; naming what none of them covers is the top one.

SEE ALSO Slot III - 59 elder abuse and neglect Slot III - 62 the unresponsive patient and consent **SPRINT** Day 29, consultation-liaison and geriatric psychiatry

III - 62

What is consultation-liaison psychiatry? Discuss how to plan for an unconscious patient brought by local police from CL psychiatry point of view? [2+8]

2 + 8 | GERIATRIC, CONSULTATION-LIAISON AND TRANSCULTURAL PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	One block, then move: the marks are in part b
4 The sequence	Resuscitate, history from police, examine, investigate
2 Psychiatric causes	A diagnosis of exclusion, with its discriminating signs
2 Duties	Consent, documentation, and the police duty

THE DEFINITION

Psychiatry practised at the interface with the rest of medicine: psychiatric illness in the physically ill, physical illness presenting psychiatrically, and the behaviour, capacity and communication problems that a general hospital generates.

STEP	WHAT IS DONE	WHY	WEIGHT
Resuscitate	Airway, breathing, circulation, glucose, thiamine first where alcohol is likely, naloxone for an opioid picture	Nothing psychiatric happens before this	**Never psychiatric first**
History from the police	Where and how found, position, container or note, smell, time last seen	The only history available	Record it verbatim
Examine	Injuries, needle marks, pupils, tone, reflexes, neck stiffness, focal signs, toxidrome	Separates structural from toxic	Document injuries for the record
Investigate	Electrolytes, renal and liver function, gases, toxicology, imaging, and lumbar puncture or electroencephalogram if indicated	Encephalitis and non-convulsive status hide here	**Electroencephalogram when nothing else explains it**
Then psychiatry	Catatonic stupor, dissociative unresponsiveness, depressive stupor, malingering	Exclusion, in that order	The lorazepam challenge sits here

- **Signs that point away from a structural cause.** Resistance to eye opening, an intact eyelash reflex, avoidance of the falling hand, normal caloric responses and a normal electroencephalogram. These are described, and never used to accuse.
- **The duties around an unidentified patient.** Treat under the emergency doctrine, document everything including injuries, inform the police in writing, trace family through the hospital and the police, and record capacity and consent as they change. The Act also places a duty on a police officer to take a wandering person with apparent mental illness under protection and bring them for assessment.

WHAT EARNS THE MARKS

- **Definition in one line, then the answer moves.** Two marks do not deserve a paragraph.
- **Resuscitation and investigation placed ahead of every psychiatric hypothesis.**
- **The psychiatric differential given as exclusions,** with the discriminating signs named rather than gestured at.

SEE ALSO Slot III - 65 models of consultation-liaison psychiatry Slot III - 60 the Indian model and intensive-care referral

SPRINT Day 29, consultation-liaison and geriatric psychiatry

III - 63

What are the physiological changes in sleep in elderly persons? Discuss differential diagnosis of insomnia in the elderly. What are the nonpharmacological strategies to manage insomnia in elderly? [3+3+4]

3 + 3 + 4 | GERIATRIC, CONSULTATION-LIAISON AND TRANSCULTURAL PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Physiological change**	The baseline the differential is measured against
3	**The differential**	Four causes, worked as a sieve
3	**Non-pharmacological**	Ranked, with the first-line named
1	**What is not done**	One honest line about hypnotics

WHAT AGEING DOES TO SLEEP

The circadian signal weakens and advances: sleep and waking both come earlier. Slow wave sleep falls markedly, rapid eye movement a little and sooner, arousals multiply, efficiency drops. The need changes little; taking it in one block does.

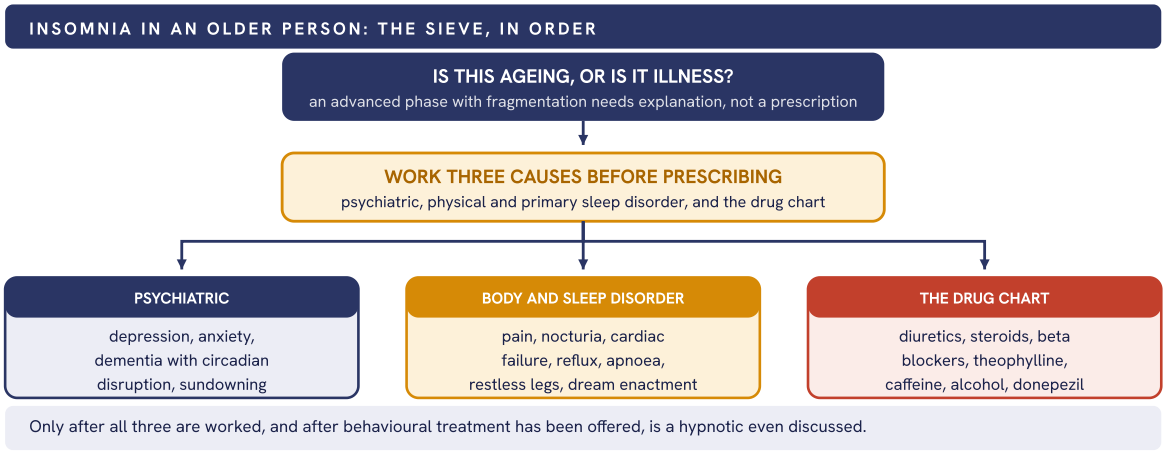


Fig 063.1 The drug chart gets a lane of its own, being the cause least often examined.

- **The non-pharmacological answer, ranked.** Cognitive behavioural therapy for insomnia first: stimulus control, sleep restriction, cognitive work and relaxation, with sleep hygiene as an adjunct and never as the whole treatment. Then timed evening light for an advanced phase, daytime activity, restricted napping, and treatment of pain and nocturia.
- **What is not done.** A hypnotic as the first act. Benzodiazepines and Z drugs in this age group buy a modest gain in sleep at the price of falls, fracture, confusion and dependence.

WHAT EARNS THE MARKS

- **Normal ageing described before any pathology.**
- **The differential worked as a sieve,** with the drug chart counted as a cause.
- **Behavioural therapy named as first-line,** with sleep hygiene demoted to an adjunct.

SEE ALSO Slot III - 64 delivering the behavioural sleep treatment Slot III - 66 geriatric depression **SPRINT** Day 29, consultation-liaison and geriatric psychiatry

III - 64

Discuss the sleep changes in elderly. Write the non-pharmacological approaches for management of sleep problems in them. [5+5]

5 + 5 | GERIATRIC, CONSULTATION-LIAISON AND TRANSCULTURAL PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Normal ageing**	Architecture, timing and continuity, each named
2	**Not ageing**	Three findings that are disorders, not decades
4	**Delivery**	The flowchart: diary, prescription, three components
1	**In dementia**	The same measures, delivered by the carer

THE QUESTION BEHIND THE QUESTION

Two things are being asked. Which sleep changes are ordinary ageing, and so need explanation rather than treatment; and what can be done without a drug. Answering the first well is what makes the second credible to a family expecting a tablet.

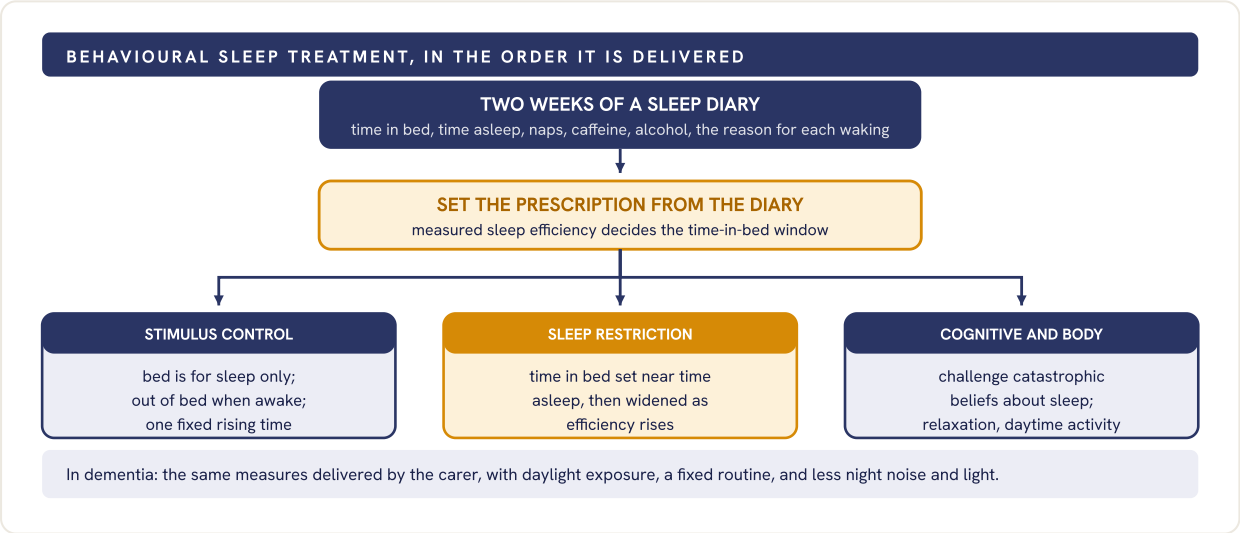


Fig 064.1 The diary comes before the prescription: the window is calculated from measured sleep.

- **Normal against pathological, stated first.** Earlier sleep and waking, less slow wave sleep, more arousals and lower efficiency are ageing. Loud snoring with witnessed apnoea, an urge to move the legs at night, and acting out dreams are disorders, and are treated rather than explained away.
- **Why this is not the soft option.** Behavioural treatment outperforms hypnotics at follow-up, carries no fall or fracture risk, and its gains persist after the course ends.

WHAT EARNS THE MARKS

- **Normal ageing separated from disorder,** with named disorders on the pathological side.
- **The components named individually,** not lumped together as "sleep hygiene".
- **The dementia and institutional adaptation included,** since that is where most of these patients are.

SEE ALSO Slot III - 63 the differential of insomnia in the elderly Slot III - 66 geriatric depression **SPRINT**

Day 29, consultation-liaison and geriatric psychiatry

III - 65

What is consultation-liaison psychiatry? Discuss the different models. [2+8]

2 + 8 | GERIATRIC, CONSULTATION-LIAISON AND TRANSCULTURAL PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The definition	One block, no more: eight marks are below it
2	The axis	State what the classification divides on
4	The models	Drawn, each with what it costs and what it buys
2	Which fits India	Name the constraint, not the ideal

THE DEFINITION

Psychiatry practised at the interface with the rest of medicine: psychiatric illness in the physically ill, physical illness presenting psychiatrically, and the behaviour, capacity and communication problems a general hospital generates.

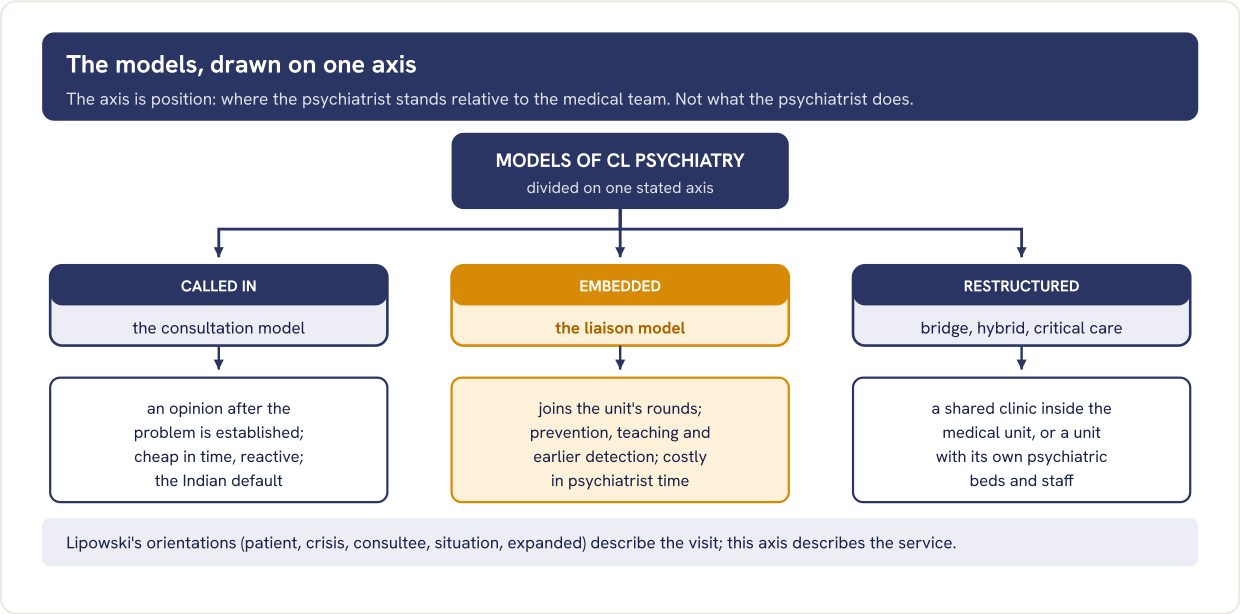


Fig 065.1 One axis, three positions, and under each the cost as well as the benefit. A tree here, so no table: the classification is the answer.

- **Which fits Indian practice.** The consultation model dominates, because one psychiatrist covers a whole hospital. Liaison survives where a specialty funds it, in oncology, transplant, obstetric and epilepsy services. The honest answer names the constraint rather than recommending the ideal.

WHAT EARNS THE MARKS

- **The axis stated before the models.** A list of model names with no axis reads as recall.
- **Every model given its cost as well as its benefit.** An incomplete tree loses more than a plain list would.
- **The Indian answer named, with the reason.** Recommending liaison without saying who would staff it is the weak ending here.

SEE ALSO Slot III - 60 the Indian model and intensive-care referral Slot III - 62 the unresponsive patient **SPRINT** Day 29, consultation-liaison and geriatric psychiatry

III - 66

Clinical features and challenges in management of geriatric depression. [4+6]

4 + 6 | GERIATRIC, CONSULTATION-LIAISON AND TRANSCULTURAL PSYCHIATRY

DNB, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	How it presents	Different from younger adults, in named ways
2	Two special forms	Cognitive impairment, and the vascular subtype
4	The pathway	The flowchart: screen, stratify, treat by severity
2	Prescribing	The challenge, stated as a principle

HOW IT PRESENTS DIFFERENTLY

Less complaint of sadness, more somatic preoccupation, anxiety, and agitation or retardation. Guilt and nihilistic delusions are commoner in late life. Early waking, weight loss, and a higher suicide rate with more lethal methods and less warning.

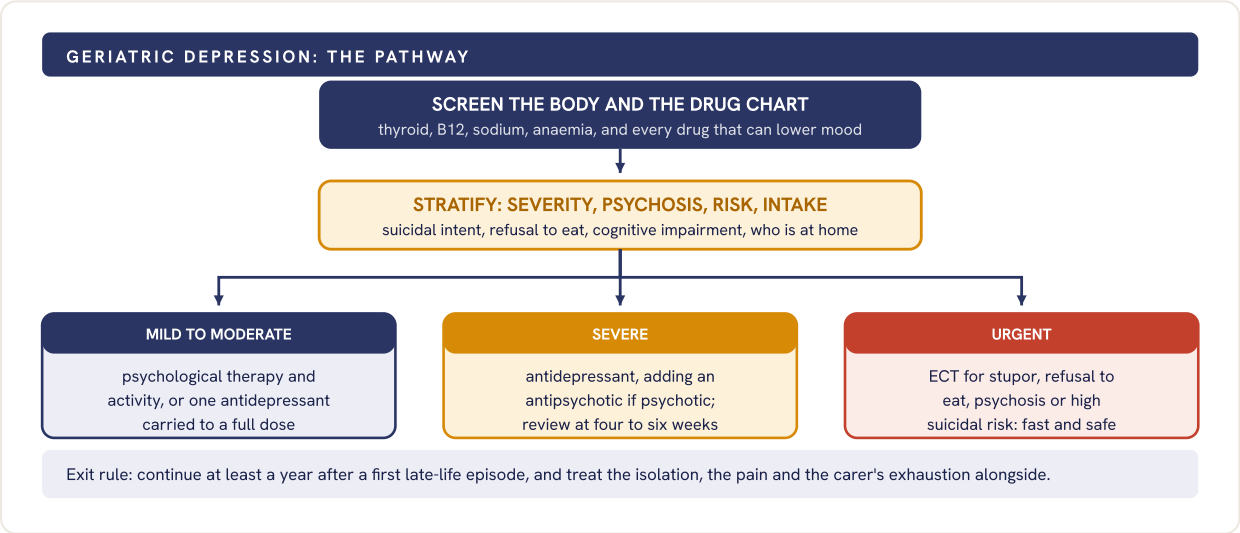


Fig 066.1 The body and the drug chart sit above the fork, not beside it.

- **Two forms worth naming.** Depression with reversible cognitive impairment, where effort is poor but recognition is preserved and the complaint is louder than the deficit; and vascular depression, of late onset, with executive dysfunction, white matter change and a poorer drug response.
- **The prescribing challenge, as a principle.** Start low and go slow, but arrive at a full dose for a full duration: undertreatment is the commoner error at this age. Watch hyponatraemia, falls, QT prolongation, and anticholinergic burden summed across the whole chart rather than drug by drug.

WHAT EARNS THE MARKS

- **The presentation described as different from younger-adult depression,** in named ways rather than as "atypical".
- **Undertreatment named alongside start low and go slow.** Quoting only the caution is the half answer.
- **ECT placed as fast and safe in late life,** not as a last resort after everything else has failed.

SEE ALSO Slot III - 63 insomnia in the elderly Slot III - 61 medicolegal aspects of dementia SPRINT
Day 29, consultation-liaison and geriatric psychiatry

AREA 08 OF 13 · P3-A10

ECT and neurostimulation

8	7.9%	III-67	III-74
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 7

REVISION ORDER

The two ECT slots first: they are the ones a board is most likely to set. Then the non-convulsive devices as one group, because they are asked as a group. Ketamine and the subspecialty slot last.

WHAT IS IN THIS AREA

ECT indications and contraindications, unilateral ECT and electrode placement, rTMS, transcranial direct current stimulation, vagus nerve and deep brain stimulation, ketamine, interventional psychiatry as a subspecialty, and ECT inside a prevention and rehabilitation frame.

III - 67

Discuss indications and contraindications of Electro convulsive therapy. [10]

10 | ECT AND NEUROSTIMULATION | KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What it is	The treatment defined, and the law it is given under
3 Indications	By urgency first, then by diagnosis
3 Contraindications	None absolute; the risk list and what each changes
2 What decides it	Speed, refractoriness, consent, the Act's limits

THE DEFINITION

A generalised cerebral seizure of adequate duration, induced by a brief pulse stimulus to the scalp under general anaesthesia and muscle relaxation. Modified ECT is the only lawful form: the Mental Healthcare Act 2017, Section 95, bars ECT without anaesthesia and relaxants, and bars it for a minor except with guardian consent and the Board's prior permission.

- **Urgent, where delay is itself the danger.** Sustained high suicidal intent, refusal of food and fluids, stupor, malignant catatonia, severe puerperal illness, and neuroleptic malignant syndrome.
- **Established by diagnosis.** Severe depression, especially with melancholic or psychotic features; mania that is unresponsive or exhausting; catatonia of any cause; schizophrenia with catatonic or affective features, or persistent drug resistance.
- **There is no absolute contraindication.** Raised intracranial pressure with a space-occupying lesion comes nearest. Everything else is a risk to be quantified with the anaesthetist and consented to, not a bar.

DOMAIN	THE CONCERN	WHY IT MATTERS	WHAT CHANGES
Intracranial	Space-occupying lesion, raised pressure	Surge in cerebral blood flow and pressure	Neurosurgical opinion first
Cardiac	Recent infarct, unstable angina, arrhythmia	Vagal bradycardia, then a sympathetic surge	Cardiology input, rate control
Vascular	Aortic or cerebral aneurysm	Transient rise in arterial pressure	Pressure controlled through the fit
Endocrine	Pheochromocytoma	Catecholamine release	Alpha blockade before treatment
Ocular, respiratory	Retinal detachment, unstable airway disease	Pressure change, anaesthetic risk	Specialist opinion, then optimise

WHAT EARNS THE MARKS

- **The Act named, not implied.** Unmodified ECT is unlawful, and a minor needs guardian consent and the Board's prior permission.
- **Indications ordered by urgency before diagnosis.** The emergency list separates a top answer from a recalled one.
- **"No absolute contraindication" written as a sentence,** with raised intracranial pressure named as the nearest thing to one, and the amber rows read as modifiers of preparation rather than bars.

SEE ALSO Slot III - 73 unilateral ECT and electrode placement Slot III - 70 ECT across prevention and treatment

SPRINT Day 27, ECT and neurostimulation

III - 68

Transcranial direct current stimulation; Discuss the current status with evidences. [10]

10 | ECT AND NEUROSTIMULATION | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it is	The method, the dose, and how it differs from rTMS
5	The evidence	Indication by indication, each with its study type
2	Where it stands	Regulation, safety, and Indian availability
1	What is unsettled	One honest sentence about the field

- **The method.** A weak constant current, usually 1 to 2 mA, passed between two scalp electrodes for 20 to 30 minutes daily. It does not induce a seizure and does not fire neurons; it shifts resting membrane potential, so the anode raises and the cathode lowers cortical excitability. Effects outlast the session through synaptic plasticity.
- **The usual montage.** Anode over the left dorsolateral prefrontal cortex, cathode over the right, given in a course of ten to twenty sessions. Portable, cheap, needs no anaesthesia, and can in principle be delivered outside a procedure room.

INDICATION	WHAT IS CLAIMED	EVIDENCE SO FAR	STATUS
Unipolar depression	Antidepressant effect as monotherapy or add-on	Several sham-controlled trials and meta-analyses of them	**Best supported use**
Auditory hallucinations	Fronto-temporal montage reduces voices	Small randomised trials, replication uneven	Promising, contested
Negative symptoms	Prefrontal anodal stimulation	Small trials, short follow-up	Trial stage
Craving in substance use	Reduced cue-induced craving	Short crossover studies, soft endpoints	Trial stage
Cognition, obsessive-compulsive disorder	Working memory, symptom reduction	Exploratory, heterogeneous montages	Promising only

STATUS: WHERE THIS SITS TODAY

Safety is the strong part of the record: no seizures at conventional dose, and adverse effects limited to itching, tingling and transient erythema under the pads. Efficacy is the weak part. Trials are small, montages and session counts differ, and blinding is imperfect. It is not an approved standalone treatment in Indian practice, and its place is adjunctive and largely within research and specialist settings. It does not displace ECT where speed matters, or rTMS where the evidence base is larger.

WHAT EARNS THE MARKS

- **Subthreshold, not suprathreshold.** Saying it modulates excitability rather than inducing firing is the sentence that shows the mechanism is understood.
- **Each indication tied to its study type.** A named use with no design behind it earns nothing on this archetype.
- **Status separated from promise,** and safety separated from efficacy.
- **One honest unsolved line:** heterogeneity of montage and dose is why the trials do not converge.

SEE ALSO Slot III - 69 rTMS principle and utility Slot III - 72 interventional psychiatry SPRINT Day 27, ECT and neurostimulation

III - 69

Discuss the principle of RTMS, indications and utility in clinical practice. [3+2+5]

3 + 2 + 5 | ECT AND NEUROSTIMULATION | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Principle	Induction, coil, depth, and how the dose is set
2 Indications	One established use, the rest ranked below it
3 Utility	Answered as a comparison with ECT, not as a list
2 Limits and safety	Seizure risk, the implant screen, durability

THE PRINCIPLE

A brief high current pulse through a coil on the scalp creates a rapidly changing magnetic field. The field crosses skull and scalp without attenuation and induces a secondary electric current in the cortex beneath it, which depolarises interneurons a few centimetres deep. This is Faraday induction, not conduction of electricity through the head. A figure of eight coil focuses the field, and the dose is set as a percentage of that person's resting motor threshold.

- **Frequency decides direction.** High frequency, 5 Hz and above, raises cortical excitability; 1 Hz lowers it. Intermittent theta burst is the short excitatory protocol, continuous theta burst the inhibitory one.
- **The depression protocol.** High frequency over the left dorsolateral prefrontal cortex, or 1 Hz over the right, on consecutive working days for four to six weeks. No anaesthesia, no seizure, no memory cost, so the patient drives home.
- **The one serious risk is a seizure.** Rare, and almost always during the session. Screen for ferromagnetic implants near the head, cochlear implants and implanted pulse generators. Hearing protection is worn.

USE	TARGET AND DIRECTION	EVIDENCE	WHERE IT SITS
Resistant depression	Left prefrontal, excitatory	Largest randomised base	**The established use**
Auditory hallucinations	Left temporoparietal, inhibitory	Several trials, modest effect	Adjunct
Obsessive-compulsive disorder	Supplementary motor, orbitofrontal	Smaller randomised trials	Adjunct in resistance
Negative symptoms, craving	Prefrontal, excitatory	Mixed and exploratory	Trial stage

Utility is judged against the alternative: slower and weaker than ECT, better tolerated, and useless when the patient cannot wait four weeks.

WHAT EARNS THE MARKS

- **Faraday induction named,** with the coil, the depth and the motor threshold. "Magnetic stimulation" without the induced current is a half answer.
- **Frequency mapped to direction of effect,** and the side that goes with each.
- **Utility written as a comparison with ECT.** Speed, severity and memory are the three axes an examiner is checking.
- **Seizure risk and the implant screen,** one line each, before the answer ends.

SEE ALSO Slot III - 68 transcranial direct current stimulation Slot III - 72 interventional psychiatry SPRINT Day 27, ECT and neurostimulation

III - 70

a) Describe terms prevention, treatment and rehabilitation in context of psychiatry. b) Impact of ECT in these terms. [5+5]

5 + 5 | ECT AND NEUROSTIMULATION | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The axis**	Defined by when a service acts
3 **What each level does**	The content of all three, with examples
3 **ECT at each level**	Drawn: none, most, and indirect
2 **The honest limit**	Where ECT is not prevention at all

- **The axis.** Caplan placed a service by the moment it acts. Primary acts before onset and lowers incidence; secondary acts early and lowers prevalence by shortening the episode; tertiary acts after it and lowers disability.
- **Treatment is not a fourth level.** It is the instrument secondary prevention uses, and it counts as prevention only because it shortens the episode and prevents the next.

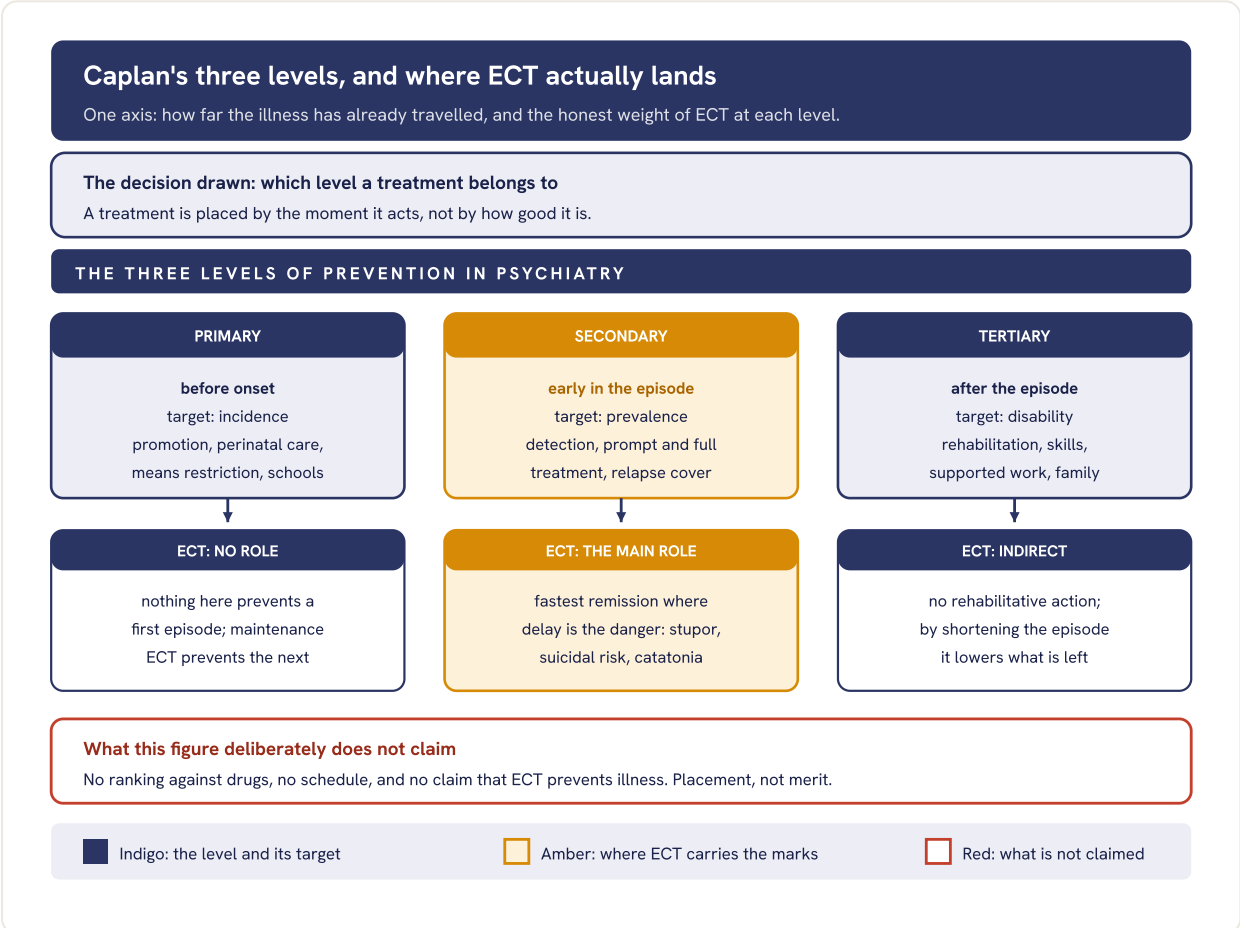


Fig 070.1 The levels read left to right as the illness advances, with ECT placed under each rather than praised in general.

WHAT EARNS THE MARKS

- **The axis stated before the levels, with Caplan named.**
- **ECT placed, not praised.** The answer that says it has no primary preventive role is the one that has understood part b.
- **Maintenance and tertiary answered honestly.** Maintenance ECT prevents the next episode, not the first, and ECT shortens the episode that rehabilitation must then repair.

SEE ALSO Slot III - 67 indications and contraindications of ECT Slot III - 73 unilateral ECT and electrode placement

SPRINT Day 39, community psychiatry and public health

III - 71

Vagus Nerve stimulation and Deep Brain Stimulation. [10]

10 | ECT AND NEUROSTIMULATION | RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Both defined**	Implanted, reversible, adjustable: that is the shared axis
3	**Vagus nerve stimulation**	Hardware, mechanism, use, adverse effects
3	**Deep brain stimulation**	Targets, use, adverse effects
2	**Selection and the law**	Who qualifies, and why this is not psychosurgery

THE DEFINITION

Two implanted neuromodulation treatments for severe treatment-resistant illness. Vagus nerve stimulation delivers chronic intermittent pulses to the left cervical vagus from a generator in the chest wall. Deep brain stimulation delivers continuous adjustable stimulation from electrodes placed stereotactically in a defined subcortical target. Neither destroys tissue, and both are reversible and adjustable. That is what separates them from the ablative operations they replaced.

FEATURE	VAGUS NERVE STIMULATION	DEEP BRAIN STIMULATION	WHAT FOLLOWS
Where it acts	Left cervical vagus, afferents to the brainstem and on to limbic cortex	Subcallosal cingulate, ventral capsule and striatum, nucleus accumbens	**Target choice is the whole of the second**
Onset	Slow: months, still improving at a year	Weeks, and adjustable at each visit	**Changes who can wait for it**
Psychiatric use	Adjunctive in resistant depression	Refractory obsessive-compulsive disorder best supported	Evidence is uneven in both
Adverse effects	Voice change, hoarseness, cough, breathlessness while stimulating	Haemorrhage, infection, hardware failure, hypomania at some targets	One is a nuisance, one is surgical
Neurological use	Refractory epilepsy	Parkinson disease, dystonia, tremor	Both entered psychiatry from neurology

- **Selection is the hard part.** Both require a documented, adequate trial of established treatments, a stable diagnosis, capacity, and formal multidisciplinary and ethical review. Neither answers a first refractory episode.
- **Not psychosurgery, in law.** The Mental Healthcare Act 2017, Section 96, reads on interventions designed to destroy brain tissue or its functioning, and requires informed consent and the Board's approval. Stimulation destroys nothing, so it falls outside that definition while still needing consent and oversight on ordinary grounds.

WHAT EARNS THE MARKS

- **Each defined by what is stimulated and where the hardware sits,** not by the abbreviation.
- **Reversible and adjustable, written as the reason these replaced ablative surgery.**
- **Onset separated:** months against weeks, and what that does to selection.
- **The legal line.** Destruction of tissue is what the psychosurgery provision turns on.

SEE ALSO Slot III - 72 interventional psychiatry Slot III - 69 rTMS principle and utility SPRINT
Day 27, ECT and neurostimulation

III - 72

Write briefly about Interventional Psychiatry as a subspecialty of psychiatry. [10]

10 ECT AND NEUROSTIMULATION WBUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Defined by how treatment is delivered, not by diagnosis
3 What it contains	Four families, an example in each
3 Why it separated out	Refractoriness, and what a procedure needs that a clinic lacks
2 What it demands	Skills, governance, and the honest evidence line

THE DEFINITION

The subspecialty that delivers device-based and procedure-based treatments for severe or treatment-resistant mental illness, in a setting built for anaesthesia, monitoring, consent and audit. Its unit of practice is a procedure with a dose, a checklist and a recovery period, rather than a prescription.

FAMILY	EXAMPLES	WHAT IS DELIVERED	SETTING
Convulsive	ECT, magnetic seizure therapy	An induced generalised seizure	**Anaesthesia and recovery**
Non-invasive modulation	rTMS, theta burst, transcranial direct current stimulation	Subconvulsive cortical modulation	Outpatient chair
Implanted modulation	Vagus nerve stimulation, deep brain stimulation	Chronic stimulation from implanted hardware	Theatre, then clinic
Procedural pharmacology	Intravenous ketamine, intranasal esketamine, intravenous clomipramine	A drug given under observation	**Day care with monitoring**

Amber marks the two families that cannot be delivered in an ordinary consulting room, which is the argument for a subspecialty.

- **Why it separated out.** A large share of depressive illness does not remit after two adequate drug trials. The treatments that answer that are procedures, and procedures need rooms, trained staff, checklists and outcome audit.
- **The failure mode is enthusiasm.** A device with a plausible mechanism and thin trials is still experimental. Ranking the families by evidence, rather than by novelty, is what a good answer does here.

WHAT EARNS THE MARKS

- **Defined by delivery, not by diagnosis.** Procedures rather than drug classes is the organising idea.
- **The four families named, with an example in each.** A list of devices with no grouping reads as recall.
- **The infrastructure argument.** Anaesthesia, monitoring, consent and audit is why this is a subspecialty and not a technique.
- **Evidence weight kept honest** across established, adjunctive and experimental.

SEE ALSO Slot III - 71 vagus nerve and deep brain stimulation Slot III - 74 ketamine in psychiatry **SPRINT** Day 27, ECT and neurostimulation

III - 73

Advantages of Unilateral Electroconvulsive Therapy and electrode placement for the procedure. [10]

10 ECT AND NEUROSTIMULATION RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Why unilateral**	Dominant hemisphere spared: the whole rationale
3 **The placements**	All three drawn, each with its surface landmark
3 **Advantages**	Cognitive, and stated as differences not as praise
2 **The condition**	Suprathreshold dosing, and when bitemporal still wins

THE RATIONALE

The right hemisphere is non-dominant for language in most people. Both electrodes over it still induce a generalised seizure, but current density over the left temporal structures that carry verbal memory is much lower. Same treatment, different cost to memory.

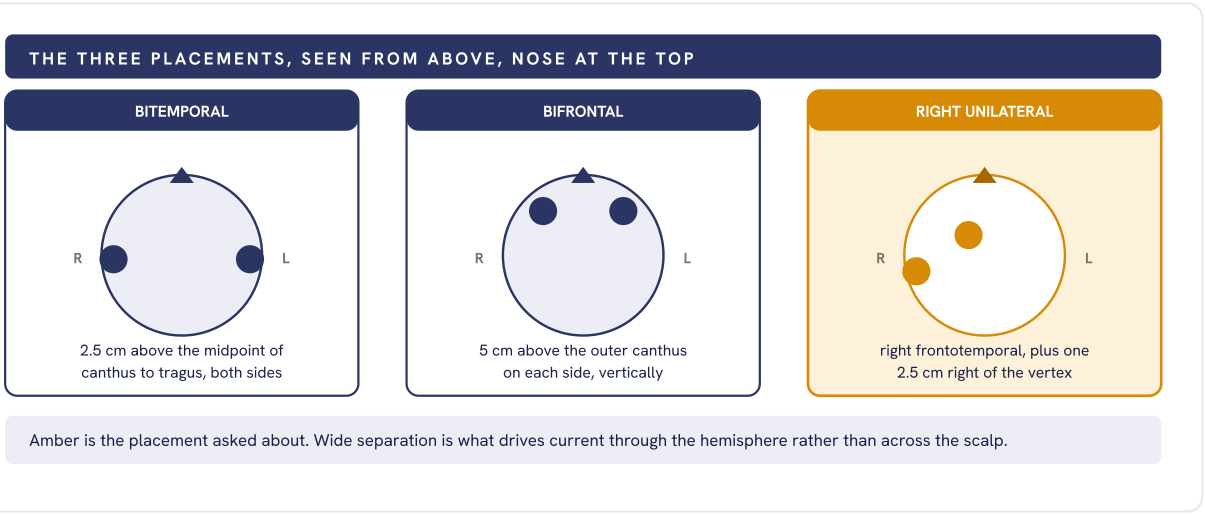


Fig 073.1 The d'Elia position is defined by the separation of the two electrodes as much as by either landmark.

- **The advantage is cognitive.** Less retrograde and anterograde memory impairment, shorter post-ictal disorientation and faster reorientation than bitemporal, at equal antidepressant efficacy when the dose is adequate.
- **The dose is the condition.** Unilateral works only well above seizure threshold, conventionally about six times it, so the threshold is titrated at the first session. Given near threshold it is a weak treatment with a good side effect profile, which is the worst of both. Bitemporal is still chosen where speed outweighs memory.

WHAT EARNS THE MARKS

- **Each placement given with its surface landmark,** not just its name.
- **The dose condition stated alongside the placement.** Unilateral without suprathreshold dosing is the commonest error here.
- **The advantage specified as memory and reorientation,** not vague tolerability.

SEE ALSO Slot III - 67 indications and contraindications of ECT Slot III - 70 ECT across prevention and treatment

SPRINT Day 27, ECT and neurostimulation

III - 74

Ketamine in Psychiatry. [10]

10 ECT AND NEUROSTIMULATION RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it is	Receptor, origin, and why it changed the field
3	Mechanism	Blockade, glutamate surge, synaptic repair
3	Uses, dose, route	Each use with its dose and its time course
2	Limits	Durability, adverse effects, and control status

THE DEFINITION

A non-competitive antagonist at the NMDA glutamate receptor, in anaesthetic use since the 1960s and repurposed as a rapid-acting antidepressant. One subanaesthetic dose lifts mood within hours in illness that has not responded to conventional drugs, which broke the assumption that antidepressant action must take weeks.

USE	DOSE AND ROUTE	TIME COURSE	EVIDENCE
Resistant depression	Racemic ketamine 0.5 mg per kg intravenously over 40 minutes	Response in hours, peak near 24 hours	**Several randomised trials**
Resistant depression	Esketamine, intranasal, alongside an oral antidepressant	Slower and steadier than intravenous	Registration trials
Acute suicidal ideation	Single infusion, within an admission	Ideation falls faster than mood	**Short follow-up only**
Anaesthesia for ECT	Anaesthetic dose, by the anaesthetist	Does not raise seizure threshold	Long anaesthetic record

- **The mechanism as taught.** Blockade on inhibitory interneurons releases a glutamate surge, which drives AMPA receptor throughput, brain-derived neurotrophic factor release and mTOR signalling, and restores prefrontal synaptic connections within hours.
- **The problem is durability, not response.** The effect fades over three to seven days, so treatment is a course, commonly twice weekly, and the answer must say what maintains the gain afterwards.

PITFALL

Dissociation, a rise in blood pressure and pulse, nausea and transient anxiety are expected effects of a monitored dose. Cystitis, hepatobiliary injury and dependence belong to heavy recreational use. Confusing the two lists, or forgetting that it is a controlled substance, both lose marks.

WHAT EARNS THE MARKS

- **The receptor named, and the surge that follows it.** Antagonism alone does not explain an antidepressant effect.
- **Dose, route and time course written down,** not gestured at.
- **The anti-suicidal effect kept separate from the antidepressant effect.**
- **Durability named as the limitation,** with a maintenance plan implied.

SEE ALSO Slot III - 72 interventional psychiatry Slot III - 67 indications and contraindications of ECT SPRINT
Day 12, mood disorder pharmacotherapy and resistance

AREA 09 OF 13 · P3-A11

Emergency psychiatry

7	6.5%	III-75	III-81
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 2 · RECURRENT 1 · SINGLE 4

REVISION ORDER

Suicide risk assessment first, and reuse its structure for the three other suicide slots. Catatonia and NMS next, as one differential. Restraint last, and it is a short structured answer.

WHAT IS IN THIS AREA

Suicide risk assessment, suicide in children, deliberate self-harm in adolescents, family and mass suicide, catatonia, neuroleptic malignant syndrome and restraint.

III - 75

Discuss the assessment of suicide risk. [10]

10 EMERGENCY PSYCHIATRY KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What is assessed	A formulation of drivers, not a probability
3 The interview	What is asked, and in what order
3 The domains	Static, dynamic, warning, contextual, protective
2 The output	Safety plan, means restriction, dated follow-up

WHAT IS BEING ASSESSED

Not a probability. A suicide risk assessment names this person's current drivers, their access to means, and what would have to change for them to be safer. The interview is itself the instrument, and asking directly about suicide does not plant the idea.

DOMAIN	WHAT IT COVERS	WHAT IT IS FOR	WEIGHT
Static	Previous attempt, chronic illness, family history, age and sex	Sets the baseline	**Previous attempt is the strongest single item**
Dynamic	Depression severity, hopelessness, psychosis, intoxication, pain, insomnia	Treatable	**Where the plan actually acts**
Warning signs	Rehearsal, final acts, giving things away, sudden calm, stated intent	Shifts over hours	Drives the immediate decision
Contextual	Access to means, isolation, recent loss, discharge, debt	Partly removable	Means restriction sits here
Protective	Dependants, responsibilities, faith, help-seeking, engagement	Modifies risk	Never a reason to discharge on its own

- **The interview, in order.** Normalise, then thoughts, then intent, then plan, then preparation and rehearsal, then access to means, then what has held them back so far. Corroborate with a relative wherever possible.
- **Instruments support, they do not decide.** Structured tools are described here, not reproduced. They organise the interview; they do not carry the decision.

PITFALL

Sorting people into low, medium and high risk does not predict who will die, and a low-risk label has been used to justify discharge. Assess drivers and modify them rather than grading and filing. A no-suicide contract is not a safety plan and has no evidence behind it.

WHAT EARNS THE MARKS

- **Risk written as a formulation with named drivers,** not as a category.
- **The direct questions written out in order,** with the myth about planting the idea answered.
- **Means restriction and a dated follow-up carried into the disposition.** An assessment that ends at a label is unfinished.

SEE ALSO Slot III - 80 adolescent with deliberate self-harm Slot III - 79 suicide in children **SPRINT** Day 18, impulse-control disorders and suicide

III - 76

Enumerate catatonic symptoms and describe the management of Catatonia. [10]

10 | EMERGENCY PSYCHIATRY | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The signs**	Enumerated in groups, not as one long list
2 **Recognition**	Screening, and the workup that must run alongside
4 **The pathway**	The flowchart: stop, challenge, titrate, escalate
2 **The malignant form**	What changes when fever and rigidity appear

- **Catatonia is a syndrome, not a diagnosis.** It occurs in mood disorder more often than in schizophrenia, and in medical, neurological and autoimmune illness, so it is screened for rather than waited for.
- **The challenge test is diagnostic and therapeutic at once.** A partial response within ten minutes of 1 to 2 mg of lorazepam supports the diagnosis and starts the treatment.

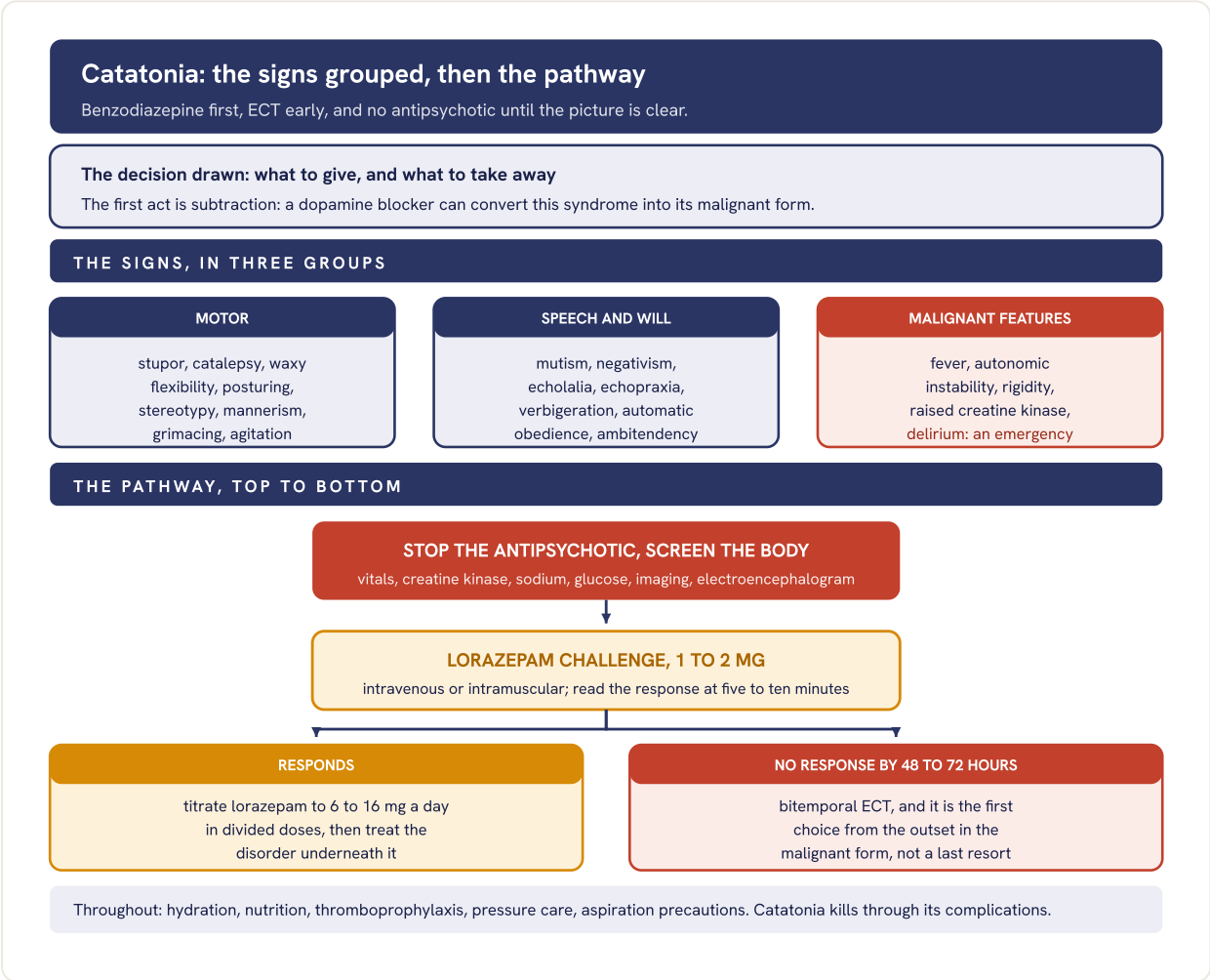


Fig 076.1 Signs above, pathway below, the two branches decided by one challenge dose.

PITFALL

Malignant catatonia and neuroleptic malignant syndrome overlap so closely that some regard them as one condition. With fever, rigidity and autonomic instability, stop the antipsychotic and move to ECT early.

WHAT EARNS THE MARKS

- **The signs enumerated in groups.** Twelve names in one undifferentiated list reads as recall.
- **The lorazepam challenge with its dose, route and reading time.**
- **ECT placed early and named as first choice in the malignant form,** with supportive care written down because immobility is what kills.

SEE ALSO Slot III - 77 neuroleptic malignant syndrome Slot III - 67 indications and contraindications of ECT **SPRINT** Day 10, other psychotic disorders and catatonia

III - 77

Neuroleptic malignant syndrome [10]

10 EMERGENCY PSYCHIATRY KUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 Definition	The tetrad, and its time relation to the drug
3 Risk and laboratory	Who gets it, and what supports the diagnosis
3 What must be excluded	There is no confirmatory test, so exclusion is the work
2 Management and after	In order, ending with the rechallenge rule

THE DEFINITION

An idiosyncratic and potentially fatal reaction to dopamine blockade: hyperthermia, generalised lead pipe rigidity, altered sensorium and autonomic instability, arising within two weeks of a dopamine antagonist being started or raised, or of a dopamine agonist being withdrawn.

AXIS	WHAT IS FOUND	WHAT IT IS FOR	WEIGHT
Risk	High potency or parenteral antipsychotic, rapid titration, agitation, dehydration, catatonia, prior episode	Dates the exposure	The drug history dates the syndrome
Laboratory	Creatine kinase often above 1000 units per litre, leucocytosis, low serum iron, myoglobinuria, acidosis	Supports	There is no confirmatory test
Complications	Rhabdomyolysis, acute kidney injury, aspiration, venous thromboembolism, coagulopathy	Sets the monitoring	These, not the fever, kill
Exclusions	Serotonin syndrome, malignant catatonia, malignant hyperthermia, anticholinergic toxicity, infection, heat stroke	Each is treated differently	Exclusion is the diagnosis

- **Management, in order.** Stop the antipsychotic and any lithium or anticholinergic; move to a monitored bed; cool, hydrate and correct electrolytes; add bromocriptine or amantadine, and dantrolene where rigidity and creatine kinase are extreme; ECT where the picture is refractory or overlaps with malignant catatonia.
- **Rechallenge is part of the answer.** Wait at least two weeks after full resolution, choose a lower potency agent, start low, titrate slowly, and monitor temperature and creatine kinase.

WHAT EARNS THE MARKS

- **The tetrad named as a tetrad,** with its time relation to the drug in the same sentence.
- **"No confirmatory test" written down,** with the laboratory findings placed as supporting rather than diagnostic.
- **The rechallenge rule,** which most answers never reach.

SEE ALSO Slot III - 76 catatonia and its management Slot III - 67 indications and contraindications of ECT SPRINT Day 9, antipsychotics and adverse effects

III - 78

Role of psychiatrist in family suicide. How it is different from mass suicide. What are preventive factors. [10]

10 EMERGENCY PSYCHIATRY MUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Both defined**	Name the axis, and say the boundary is contested
4	**The grid**	Domain by domain, with the discriminators marked
2	**The role in each**	Individual work against population work
2	**Prevention**	Population measures, means restriction named first

- **The terms, and the honesty they need.** Usage is not settled. Family suicide describes a household dying together, commonly with one dominant instigator and dependants who cannot truly consent. A suicide pact is a mutual agreement, usually between two adults. Mass suicide is a larger group outside a household, moving together under a belief or a leader. Saying the boundaries are contested is a mark, not a hedge.

DOMAIN	FAMILY SUICIDE	MASS SUICIDE	WHAT SETTLES IT
The group	One household, bound by kinship	A congregation or crowd, unrelated	**Kinship against ideology**
The driver	Debt, dishonour, illness in a dominant member, despair over dependants	Belief, charismatic leadership, group contagion	**Private shame against shared belief**
Consent	Children and dependants cannot consent, so the deaths are homicide in law	Adults usually consenting, coercion variable	The legal reading differs entirely
How it reaches care	Through an index patient or a survivor	Through media and community alarm	Different entry point
The role	Risk assessment, safeguarding the dependants, treating the index illness	Public health, media liaison, contagion control, postvention	**Individual work against population work**

PREVENTIVE FACTORS

Means restriction first: pesticide regulation and secure storage carry the largest effect in Indian data. Then debt relief and distress schemes where indebtedness is the driver; responsible media reporting, since detailed coverage of method and place drives imitation; gatekeeper training and accessible helplines; and the Mental Healthcare Act 2017, Section 115, which presumes severe stress in anyone who attempts suicide and removes the criminal shadow that kept families from asking for help.

WHAT EARNS THE MARKS

- **Both terms defined before the grid,** with the contested boundary stated in one line.
- **The consent difference made explicit.** Dependent children turn a family death into a homicide question, and that changes everything downstream.
- **Prevention answered at population level,** with means restriction named first rather than last.

SEE ALSO Slot III - 75 assessment of suicide risk Slot III - 79 suicide in children SPRINT

Day 18, impulse-control disorders and suicide

III - 79

Suicide in children. [10]

10 EMERGENCY PSYCHIATRY RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The developmental frame	Why it is rare early and rises through adolescence
3	Risk factors	By domain, and where each one is elicited
3	How it presents	Atypical, and what that does to the interview
2	What is done	Treat, restrict means, plan, refer, follow up

THE DEVELOPMENTAL FRAME

Suicide is rare before puberty and rises steeply across adolescence. The rarity is developmental rather than protective: a younger child's grasp of death as final and irreversible is incomplete, methods are less available, and intent is harder to hold over time. The rise tracks the arrival of mood disorder, impulsivity, autonomy and access.

DOMAIN	WHAT RAISES RISK	WHERE IT IS FOUND	WEIGHT
Psychiatric	Depression, previous attempt, substance use, conduct problems, neurodevelopmental conditions	Direct interview	**Previous attempt is the strongest item**
Family	Bereavement, parental mental illness, family history of suicide, conflict, harsh discipline	Carer interview	Corroboration is essential
Adversity	Physical, sexual and emotional abuse, neglect, bullying including online	Alone, without the carer	**Surfaces only in a private interview**
Situational	Examination failure, humiliation, relationship breakdown, pesticide or medicines at home	Home and school enquiry	Means restriction acts here

- **Presentation is atypical.** Irritability, behavioural change, falling school performance, somatic complaints and withdrawal more often than a stated wish to die. Direct enquiry is still needed, in the child's own words.
- **What is done.** Treat the disorder, restrict the means at home with the carer present, write a safety plan with both, involve the school, and refer for safeguarding where abuse is disclosed. Follow up within a week, not a month.

WHAT EARNS THE MARKS

- **The developmental frame stated before the risk list.** It converts a list into an explanation.
- **The child interviewed alone, written as a step.** Abuse and bullying surface nowhere else.
- **Means restriction at home and a named follow-up interval,** written into the plan rather than implied.

SEE ALSO Slot III - 80 adolescent with deliberate self-harm Slot III - 75 assessment of suicide risk SPRINT
Day 18, impulse-control disorders and suicide

III - 80

Evaluation of an adolescent with deliberate self-harm [10]

10 EMERGENCY PSYCHIATRY KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The sequence	Medical first, alone second, carer third
3	The act	Intent read from what was done, not from what is said
3	Person, context, function	Why this act, in this person, on this day
2	Disposition and duties	Admit or discharge, and what the law requires

THE SEQUENCE

Medical stabilisation first, and psychosocial assessment once the acute effects of the act have cleared. Interview the adolescent alone before interviewing the carer, and say at the start what will be kept private and what will not. An assessment done through a parent, or done while the patient is still drowsy, is not an assessment.

AXIS	WHAT IS EXAMINED	THE OPENING QUESTION	WEIGHT
The act	Planning, precautions against discovery, method, expectation of death, final acts, regret now	"What did you think would happen?"	Intent is read from the act
The person	Depression, psychosis, substance use, neurodevelopmental condition, previous self-harm	"Has this happened before?"	Repetition predicts repetition
The context	Family conflict, abuse, bullying, examination pressure, relationship breakdown	"What was happening that day?"	Where the plan will act
The function	Affect regulation, communication, escape, self-punishment	"What did it do for you?"	Separates self-harm from an attempt

- **Two constructs, not one.** Non-suicidal self-injury regulates affect and tends to repeat; a suicide attempt intends death. They coexist in the same adolescent, and the management differs, so the distinction is drawn explicitly rather than assumed.
- **The duties that follow.** The Mental Healthcare Act 2017, Section 115, presumes severe stress and bars prosecution. Sexual abuse disclosed in the interview triggers a mandatory report under the Protection of Children from Sexual Offences Act 2012. A child unsafe at home is a child protection referral, not a discharge.

WHAT EARNS THE MARKS

- **The order stated:** medical, then the adolescent alone, then the carer, with confidentiality framed at the start.
- **Intent inferred from the act,** with the markers named rather than gestured at.
- **Disposition written out:** means restriction, a carer conversation, a safety plan and a dated follow-up.

SEE ALSO Slot III - 75 assessment of suicide risk Slot III - 79 suicide in children SPRINT Day 18, impulse-control disorders and suicide

III - 81

Use of restraints in Psychiatric Emergencies. Advantages and Complications. [10]

10 | ASKED AT 7 MARKS | EMERGENCY PSYCHIATRY

TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **What restraint is**	Four kinds, named separately
2 **The legal frame**	What the Act bars outright, and what it permits
3 **Advantages**	Time-limited and purpose-bound, never general
3 **Complications**	Physical, psychological, and at service level

THE DEFINITION AND THE LAW

Restraint covers physical holding, mechanical devices, chemical restraint by rapid tranquillisation, and seclusion. The Mental Healthcare Act 2017 prohibits chaining in any form under Section 95, and under Section 97 bars seclusion and solitary confinement outright while permitting physical restraint only as the last means of preventing imminent harm, authorised by the psychiatrist in charge, recorded, reported to the Board, and never used for punishment or staff convenience.

AXIS	ADVANTAGE CLAIMED	COMPLICATION	WEIGHT
Immediate safety	Prevents assault, absconding and self-injury in the minutes before treatment acts	Positional asphyxia, aspiration, fracture, nerve and soft tissue injury during the hold	**Prone holding is the one that kills**
Enabling care	Permits examination, venous access and essential treatment	Rhabdomyolysis, venous thromboembolism, pressure injury, dehydration, hyperthermia	**Duration multiplies every risk**
Predictability	A rehearsed, time-limited procedure beats an unplanned struggle	Retraumatisation, loss of trust, disengagement, especially after abuse	Recorded in the relationship too
Service level	Buys time for de-escalation and for medication to work	Becomes a substitute for staffing, training and ward design	Rising rates are a service defect

- **What makes it defensible.** De-escalation attempted and recorded first; the least restrictive option; a named time limit with review; continuous observation with airway, circulation and posture monitored; the indication documented; and a debrief with the patient afterwards.
- **Chemical restraint is restraint.** Rapid tranquillisation carries over-sedation, respiratory depression, hypotension, dystonia and QT prolongation, and is governed by the same rules as a mechanical device. Calling it treatment does not exempt it.

WHAT EARNS THE MARKS

- **The four kinds named separately,** with the Act's prohibitions stated exactly rather than paraphrased as "restraint is discouraged".
- **Advantages written as time-limited and purpose-bound,** never as general benefits.
- **Physical complications separated from psychological ones,** with positional asphyxia named.

SEE ALSO Slot III - 76 catatonia and its management Slot III - 77 neuroleptic malignant syndrome SPRINT Day 25, Mental health legislation and forensic psychiatry

AREA 10 OF 13 · P3-A12

Perinatal and women's mental health

7	6.3%	III-82	III-88
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 6

REVISION ORDER

One classification of postpartum illness answers five of these seven slots, so build it once and reuse it. The bonding slot and the assessment slot are the two that need their own material.

WHAT IS IN THIS AREA

Postpartum psychosis in two slots, postpartum depression, postpartum blues, assessment in the postpartum period, the psychiatric disorders of pregnancy and childbirth, and the effect of postpartum illness on infant-mother bonding.

III - 82

Describe the assessment of mental illness during postpartum period. [10]

10 | PERINATAL AND WOMEN'S MENTAL HEALTH | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What is different here	Four things a general assessment does not carry
3	Timing and the body	Onset dates the syndrome; the puerperium is a medical risk period
3	The interview	The mother, the infant, and the household around them
2	Red flags	What moves this to the same day

WHAT MAKES THIS ASSESSMENT DIFFERENT

Four things a general psychiatric assessment does not carry: the timing of onset relative to delivery, a deliberate physical screen because the puerperium is a period of high medical risk, the infant as a second patient, and a safety triad of suicide, harm to the infant, and neglect.

AXIS	WHAT IS ASKED OR DONE	WHY	WEIGHT
Timing	Onset in the first days, within two weeks, or later in the first year	The syndromes separate on timing	**The first discriminator**
Physical	Temperature, pulse, pressure, bleeding, haemoglobin, thyroid, glucose, infection screen	Sepsis, eclampsia, thyroiditis and anaemia all present as mental change	**Confusion is organic until proved otherwise**
The infant	Feeding, weight, who is caring, bonding, and thoughts about the infant	The infant's safety is part of the assessment	Asked directly, without alarm
Screening	Brief postnatal instruments, described here and not reproduced	Finds what an open question misses	Never a substitute for the interview

- **The interview, adapted.** Ask about her sleep separately from the infant's; ask about intrusive thoughts of harm in a way that makes them safe to disclose; ask how she judges her own competence. Take a collateral history from the partner and the household, since symptoms are under-reported by the mother herself.
- **Red flags for same-day action.** New or rapidly changing symptoms, perplexity or confusion, pervasive guilt or hopelessness, estrangement from the infant, and any thought of harm to herself or the infant carrying intent.

WHAT EARNS THE MARKS

- **Timing used as the first discriminator,** before any symptom list.
- **The physical screen written as a step,** not assumed to have been done by obstetrics.
- **The infant assessed as a second patient,** with the direct question about harm asked and recorded.

SEE ALSO Slot III - 83 postpartum psychosis Slot III - 88 postpartum blues **SPRINT** Day 28, perinatal and women's mental health

III - 83

Postpartum Psychosis. [10]

10 PERINATAL AND WOMEN'S MENTAL HEALTH KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	Onset in days, and the frequency quoted as a range
3 Phenomenology	Fluctuating, perplexed, and often about the infant
3 Where it belongs	An affective illness, and who is most at risk
2 Differential and course	Delirium excluded; episode and illness judged apart

THE DEFINITION

An acute psychotic episode arising almost always within the first two weeks after delivery, most within the first few days, and classically quoted at one to two per thousand deliveries. It is a psychiatric emergency, because of the risk of suicide and of harm to the infant.

- **The picture is unstable.** Kaleidoscopic and changing hour to hour, with perplexity and clouding that resemble delirium, delusions and hallucinations often concerning the infant, and disorganisation. Insomnia and restlessness are usually the first signs the family notices.
- **It is mostly a bipolar illness.** Most episodes are affective, and most women either have or later declare bipolar disorder. Treatment and prophylaxis follow that reading rather than a schizophrenia reading, and saying so is the mark.

AXIS	WHAT IS FOUND	WHY IT MATTERS	WEIGHT
Risk	Bipolar disorder, previous puerperal episode, primiparity, sleep loss, family history, a stopped mood stabiliser	Decides who is watched	**Bipolar disorder carries the highest risk**
Differential	Delirium from sepsis, eclampsia, thyroid or electrolyte disturbance, substance withdrawal	The presentations overlap	**Confusion is investigated, not assumed**
Course	Onset in days, resolution over weeks with treatment, recurrence in a later puerperium	Sets prophylaxis	High enough to plan for
Outcome	Good for the episode, with continuing risk of further affective illness	Answers the prognosis part	Episode and illness differ

WHAT EARNS THE MARKS

- **Onset timed in days,** with the frequency given as a quoted range rather than a bare number.
- **The bipolar reading stated,** because it drives both treatment and prophylaxis.
- **Delirium excluded explicitly.** Perplexity is the shared feature, and the physical causes are the ones that kill quickly.

SEE ALSO Slot III - 85 aetiology and management of postpartum psychosis

Slot III - 82 assessment in the postpartum period SPRINT Day 28, perinatal and women's mental health

III - 84

Psychiatric disorders associated with pregnancy and child birth [10]

10 PERINATAL AND WOMEN'S MENTAL HEALTH KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The axis	A timeline from conception to the end of the first year
3 Antenatal	Half the answer, and the half most candidates skip
3 Postnatal	The three syndromes, separated on timing
2 Loss and context	Bereavement after loss, and the Indian risk picture

THE FRAME

Pregnancy is not protective. Ordering the answer by time, from conception to the end of the first postnatal year, turns a list into a map, and it is also how a clinician actually thinks: what is likely now, in this woman, at this point.

PERIOD	WHAT OCCURS	WHAT IS SPECIFIC	WEIGHT
Antenatal	Depression, anxiety, tokophobia, obsessive-compulsive disorder, relapse on stopping medication, substance use	As common as post-natal depression, less often detected	The commonest missed diagnosis
Around delivery	Acute stress and post-traumatic stress after a frightening or complicated delivery	The event drives it, not the hormones	Ask about the deliv-ery itself
First two weeks	Blues, and puerperal psychosis	Timing separates them	Confusion moves it out of blues
First year	Depression, anxiety with intrusive harm thoughts, bonding disorder	Overlaps with ordi-nary exhaustion	Function is the test
Pregnancy loss	Grief, depression and anxiety after miscar-riage, stillbirth or termination	Often unacknowl-edged by the family	Named loss, named grief

- **The Indian context belongs in this answer.** Son preference, intimate partner and dowry-related violence, early marriage, anaemia and undernutrition, and limited say in decisions all raise risk. The joint household can protect or crowd, depending on the relationships inside it.
- **The commonest error is starting at delivery.** Half the material is antenatal, and an answer that opens with the blues has already given away those marks.

WHAT EARNS THE MARKS

- **A timeline used as the organising axis,** stated before the first disorder is named.
- **Antenatal illness given real weight,** not one sentence on the way to the puerperium.
- **Pregnancy loss included,** since most answers stop at a live birth.

SEE ALSO Slot III - 86 postpartum depression Slot III - 88 postpartum blues SPRINT

Day 28, perinatal and women's mental health

III - 85

Describe the clinical features, aetiology and management of post-partum psychosis. [10]

10 PERINATAL AND WOMEN'S MENTAL HEALTH

KUHS, NTRUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Clinical features	One line each, ending with the two risks
3	Aetiology	Three groups, with the strongest predictor named
4	Management	The flowchart: admit, exclude, treat, escalate
1	Next time	Prophylaxis before the next delivery

- **The features, one line each.** Onset within days of delivery; insomnia and restlessness first; a fluctuating, perplexed picture with clouding; delusions and hallucinations often about the infant; disorganisation; and rapid change from hour to hour.
- **Two risks are asked about by name.** Suicide, and harm to the infant. Both are asked directly, recorded, and used to set the level of observation rather than left as an impression.

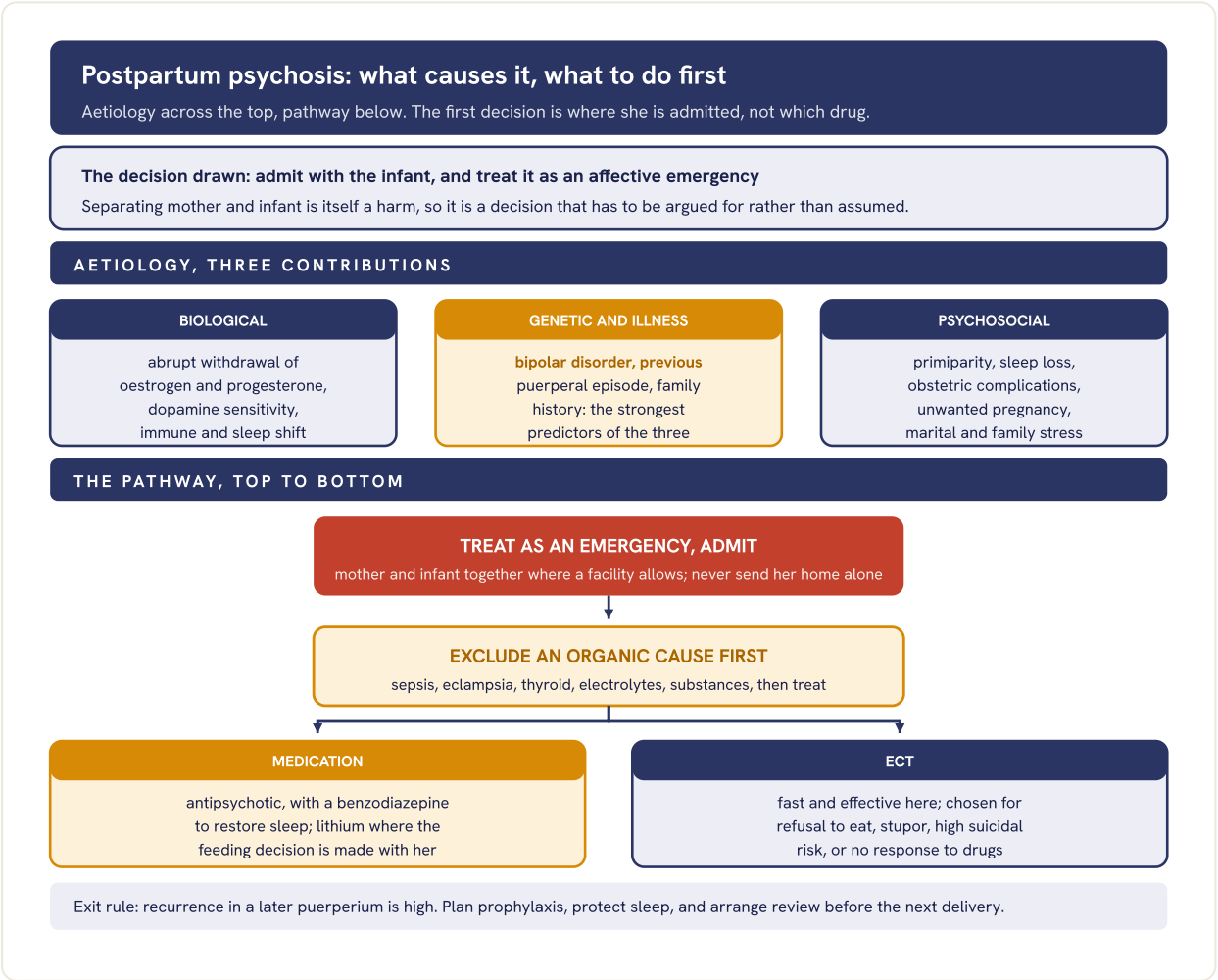


Fig 085.1 Aetiology above, pathway below, with medication and ECT as parallel options rather than a sequence.

WHAT EARNS THE MARKS

- **Aetiology answered in three groups,** with the bipolar loading named as the strongest single predictor.
- **Admission written as the first management step,** with the infant considered rather than assumed away.
- **ECT placed as a real early option,** not a footnote, and prophylaxis for the next pregnancy stated before the answer ends.

SEE ALSO Slot III - 83 postpartum psychosis as an entity Slot III - 86 postpartum depression **SPRINT** Day 28, perinatal and women's mental health

III - 86

Describe the clinical features and management of postpartum depression. [10]

10 | PERINATAL AND WOMEN'S MENTAL HEALTH | KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Clinical features**	The ones specific to this setting
2 **The neighbours**	Blues one side, psychosis the other
4 **Management**	The flowchart: screen, stratify, treat
2 **Feeding, follow-up**	Lactation principle, and how long to continue

WHAT SEPARATES IT FROM ITS NEIGHBOURS

Blues peaks around the fifth day and settles within a fortnight untreated. Depression persists past two weeks, impairs function, and carries guilt and unwanted intrusive thoughts about the infant. Psychosis adds perplexity, delusions and loss of insight.

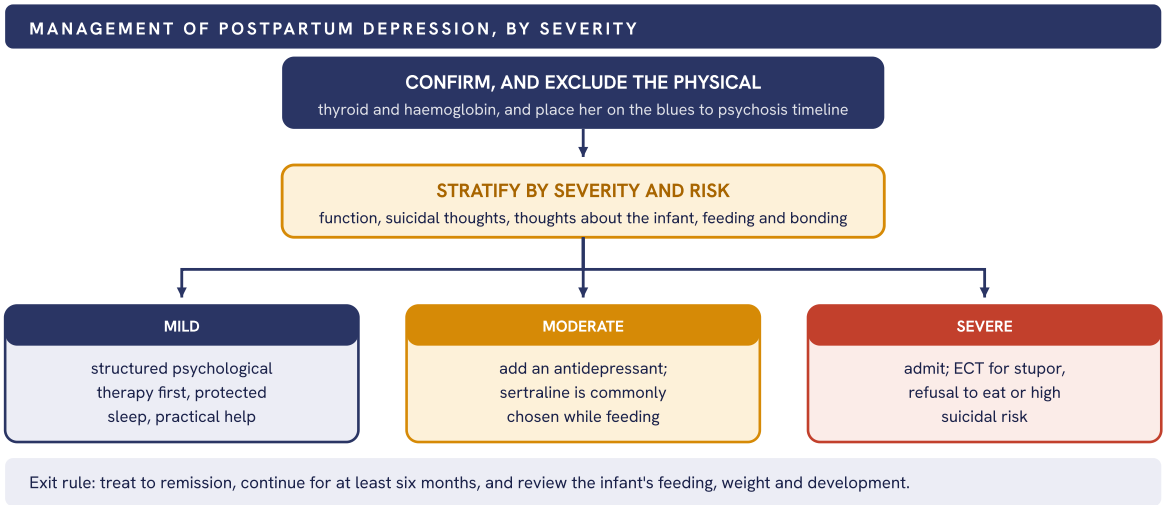


Fig 086.1 Severity chosen before treatment, with the physical screen above the fork so it cannot be skipped.

- **The features that are specific.** Guilt attached to being a bad mother; unwanted intrusive harm thoughts; sleep disturbance beyond what the infant explains; poor bonding; and reduced pleasure in the baby rather than in life generally.
- **The lactation principle, not a drug list.** Choose the agent with the most data and the lowest relative infant dose, use the lowest effective dose, and monitor the infant. A mother taken off treatment to protect feeding is the worse outcome for both.

WHAT EARNS THE MARKS

- **The three postnatal syndromes separated on timing and function** before treatment is named.
- **Severity used to choose the treatment,** with therapy first at the mild end rather than a drug for everyone.
- **Lactation answered as a principle with the infant monitored,** not as a memorised list.

SEE ALSO Slot III - 88 postpartum blues Slot III - 87 postpartum illness and bonding **SPRINT** Day 28, perinatal and women's mental health

III - 87

Influence of Postpartum psychiatric disorders on the process of Infant-Mother Bonding. [10]

10 PERINATAL AND WOMEN'S MENTAL HEALTH RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Bonding defined**	And separated from attachment, which is the infant's side
3	**Disorder by disorder**	What each one does to the interaction itself
3	**What the infant carries**	Attachment, development, behaviour, growth
2	**Buffer and repair**	What protects, and why treating the mother is not enough

THE TWO WORDS ARE NOT INTERCHANGEABLE

Bonding is the mother's emotional tie to her infant, present or absent from the first days. Attachment is the infant's developing tie to a caregiver, built over the first year. They run on different clocks, and postnatal illness can damage the first while the second is still being formed.

DISORDER	WHAT IT DOES TO THE INTERACTION	WHAT THE INFANT MEETS	WEIGHT
Depression	Withdrawal, flat affect, less looking, talking and contingent response, or an intrusive over-stimulating style	An unpredictable partner	**Reduced contingent response is the mechanism**
Anxiety and obsessional illness	Intrusive harm thoughts drive avoidance of bathing, changing, or being alone with the infant	A carer who withdraws from care	Avoidance, not aggression
Psychosis	Delusional misidentification, or delusional beliefs about the infant	Real risk, and a broken tie	**The only one with an immediate safety question**
Substance use, severe personality difficulty	Inconsistent availability and poor supervision	Neglect and unpredictability	Overlaps with safeguarding

- **What the infant carries forward.** Insecure and disorganised attachment, poorer cognitive and language development, more behavioural difficulty, and, where care is compromised, faltering growth. Severity and chronicity predict this better than the diagnostic label does.
- **What buffers, and what repairs.** A second responsive caregiver, marital support and freedom from poverty all buffer, and in a joint household that second caregiver is often the protective factor. Treating the mother's illness is necessary and not sufficient: add dyadic and video-feedback work, admit mother and infant together where possible, and involve the family.

WHAT EARNS THE MARKS

- **Bonding and attachment defined separately at the start.** Using them interchangeably costs the first two marks.
- **The mechanism named as reduced contingent responsiveness,** not as "the mother is sad".
- **The repair line.** Treating the illness alone does not restore the relationship, so dyadic work is named explicitly.

SEE ALSO Slot III - 86 postpartum depression Slot III - 85 aetiology and management of postpartum psychosis

SPRINT Day 28, perinatal and women's mental health

III - 88

Post partum blues. [10]

10 PERINATAL AND WOMEN'S MENTAL HEALTH RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition and timing	Three numbers: onset, peak, resolution
2	Frequency and cause	Common enough to be a variation, not a disorder
3	Features	Defined as much by what is absent as by what is present
3	Management and significance	No drug, and the safety net that follows

THE DEFINITION

A transient disturbance of mood in the first days after delivery, beginning around the third day, peaking near the fifth, and settling by the tenth to fourteenth without treatment. It is quoted classically as affecting between half and four fifths of mothers, which places it among normal variations rather than among disorders.

FEATURE	WHAT IS SEEN	WHAT IS NOT SEEN	WEIGHT
Mood	Tearfulness, lability, irritability, anxiety, feeling overwhelmed	Sustained low mood that does not lift with company or good news	Reactivity is preserved
Timing	Day three to five, gone by day ten to fourteen	Onset later, or lasting past two weeks	Duration is the discriminator
Function	Feeding, caring and sleeping continue	Inability to care for the infant or herself	Function stays intact
Cognition and risk	Insight retained throughout	Guilt, hopelessness, confusion, delusions, thoughts of harm	Any of these changes the diagnosis

- **Why it happens.** The abrupt fall in oestrogen and progesterone after placental separation, on a background of exhaustion, pain, disturbed sleep and a changed role. No treatment acts on this, and none is needed.
- **Management is explanation and support.** Normalise it, give the mother and the family the time course, protect her sleep, arrange practical help, and review. No antidepressant. Reassurance is offered in a way that still leaves her able to come back.

WHAT EARNS THE MARKS

- **The timing given as three numbers:** onset, peak and resolution. Vague timing is what separates a half answer here.
- **Defined by what it lacks** as much as by what it shows.
- **The safety net stated.** Blues that outlasts two weeks, deepens, or adds confusion is a different diagnosis, and blues is itself a risk marker for later depression.

SEE ALSO Slot III - 86 postpartum depression Slot III - 83 postpartum psychosis SPRINT Day 28, perinatal and women's mental health

AREA 11 OF 13 · P3-A03

Autism spectrum disorder

5	4.6%	III-89	III-93
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 · HIGH 1 · SINGLE 3

REVISION ORDER

Clinical features first, they carry three of the five slots between them. The pervasive developmental disorder classification next. Rett last, it is a named-syndrome answer.

WHAT IS IN THIS AREA

Clinical features of autism spectrum disorder, the pervasive developmental disorders, high functioning autism, the diagnostic criteria for childhood autism, and Rett syndrome.

III - 89

Describe the clinical features of autism spectrum disorder. [10]

10 AUTISM SPECTRUM DISORDER

KNRUHS, KUHS, TN-MGRMU, WBUHS - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2	The two domains	Named as a dyad, with the counting rule
4	Features by domain	Each strand, with the early sign to ask for
2	Severity and specifiers	Graded separately for each domain
2	What travels with it	The comorbidity that changes the plan

THE DYAD, IN ONE BLOCK

Persistent deficits in social communication and social interaction across contexts, with restricted, repetitive patterns of behaviour, interests or activities. All three social strands are required and two of the four repetitive strands. Onset is in the early developmental period, though it may be masked until demand exceeds capacity.

STRAND	WHAT IS SEEN	EARLY SIGN TO ASK FOR
Reciprocity	Little sharing of interest or affect, no back and forth conversation	**No bringing an object to show, by eighteen months**
Nonverbal	Reduced eye contact, few gestures, gaze and speech not integrated	**No pointing to share, by twelve months**
Relationships	Cannot adjust to context, no shared pretend play, few friendships	Little interest in other children
Repetitive	Stereotypies, echolalia, lining objects, sameness, narrow intense interests	Distress at a small change of route
Sensory	Over or under reactive; fascination with lights or spinning; pain indifference	Covering ears at ordinary sound

- **Severity and specifiers.** Three levels of support, graded separately for the social and the repetitive domain. Specify with or without intellectual impairment, with or without language impairment, an associated medical or genetic condition, another neurodevelopmental disorder, and catatonia.
- **What travels with it.** Intellectual disability, epilepsy, sleep and feeding problems, attention deficit disorder, anxiety, self injury, and catatonia appearing in adolescence. Indian assessment uses the Indian Scale for Assessment of Autism for certification, and the INCLEN diagnostic tool for case finding.

WHAT EARNS THE MARKS

- **The dyad with its counting rule.** Three of three, and two of four, is what turns a list into criteria.
- **An early sign attached to each strand.** Pointing and showing are the cheapest marks on this page.
- **Severity graded per domain,** not as a single global label.
- **Comorbidity named,** because epilepsy and catatonia change the management answer.

SEE ALSO Slot III - 92 diagnostic criteria enumerated Slot III - 90 high functioning autism SPRINT

Day 21, Intellectual disability & autism spectrum disorder

III - 90

Describe the differential diagnosis, management and prognosis of high functioning autism in a child. [3+4+3]

3 + 4 + 3 | AUTISM SPECTRUM DISORDER | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1 What the term means	Autism without intellectual or language impairment
3 Differential diagnosis	Four look-alikes, each with the line that separates it
4 Management	Teach the skill, treat what hurts, prescribe last
2 Prognosis	The predictors, and the adult risks worth naming

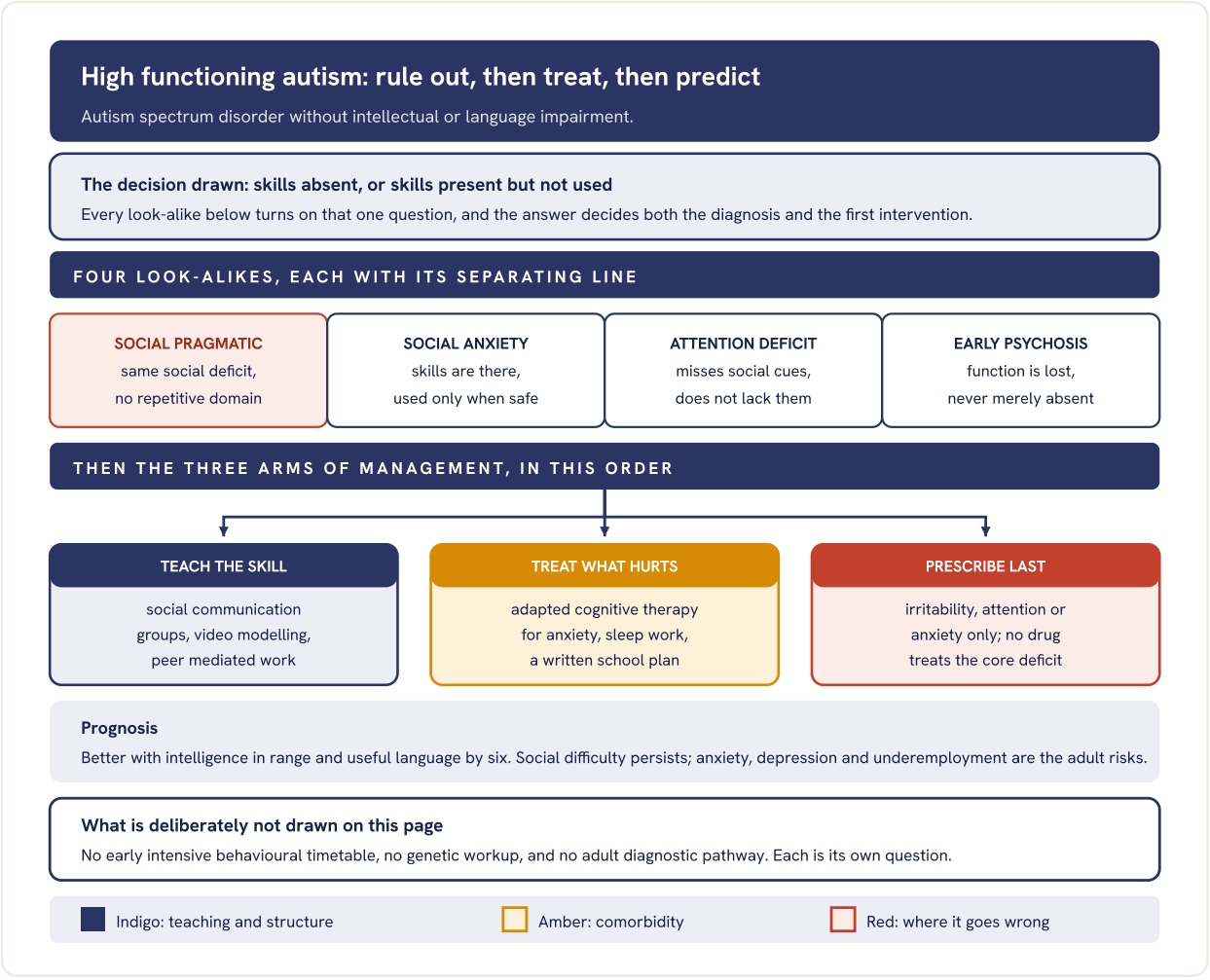


Fig 090.1 Four differentials resolved on one question, then three arms of management in order, with prognosis carried as a strip so the answer ends where the examiner expects it.

- **The rest of the differential.** Developmental language disorder, hearing impairment, obsessive compulsive disorder, reactive attachment disorder, Tourette syndrome, and a gifted child placed badly. Fragile X and the 22q11.2 deletion carry the autism phenotype and are worth testing when dysmorphism or family history points there.
- **Drugs, named honestly.** Risperidone and aripiprazole for irritability and aggression, with metabolic monitoring. Methylphenidate or atomoxetine for attention symptoms, with a lower response rate and more side effects than in attention deficit disorder alone. A serotonin reuptake inhibitor for anxiety, watching for activation.

PITFALL

Adequate speech is not adequate pragmatics. A child with fluent vocabulary who cannot repair a misunderstanding is still in the social communication domain, and calling him shy delays the diagnosis by years.

WHAT EARNS THE MARKS

- **The differential given as separating lines,** not as a list of names.
- **Behavioural and educational work placed before any drug,** with the reason stated.
- **No drug claimed for the core deficit.** Saying so is itself a mark.
- **Prognosis with named predictors,** and the adult mental health risk.

SEE ALSO Slot III - 89 clinical features Slot III - 91 pervasive developmental disorders **SPRINT**

Day 21, Intellectual disability & autism spectrum disorder

III - 91

Describe the clinical features and management of Pervasive developmental disorders. [10]

10 AUTISM SPECTRUM DISORDER NTRUHS, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What the group is	The older family, the triad, and where it sits now
3 The members	Each with the feature that identifies it
3 Management	The pathway: assess, teach, treat, entitle
2 What changes the plan	Epilepsy, regression, and the family's capacity

- **The triad, described.** Social: no shared attention, no reciprocity, indifference to another child's distress. Communication: speech delayed or absent, echolalia, no compensating gesture, no pretend play. Behaviour: stereotypies, rituals, resistance to change, narrow preoccupations.

MEMBER	THE IDENTIFYING FEATURE	WHERE IT SITS NOW
Childhood autism	Full triad, onset before three years	**Autism spectrum disorder**
Atypical autism	Late onset or an incomplete picture	Autism spectrum disorder
Asperger syndrome	No language or cognitive delay	Autism spectrum disorder
Disintegrative disorder	Normal for two years, then marked loss	Regression, workup first
Rett syndrome	Girls, hand wringing, loss of hand use	**Moved out, a genetic condition**

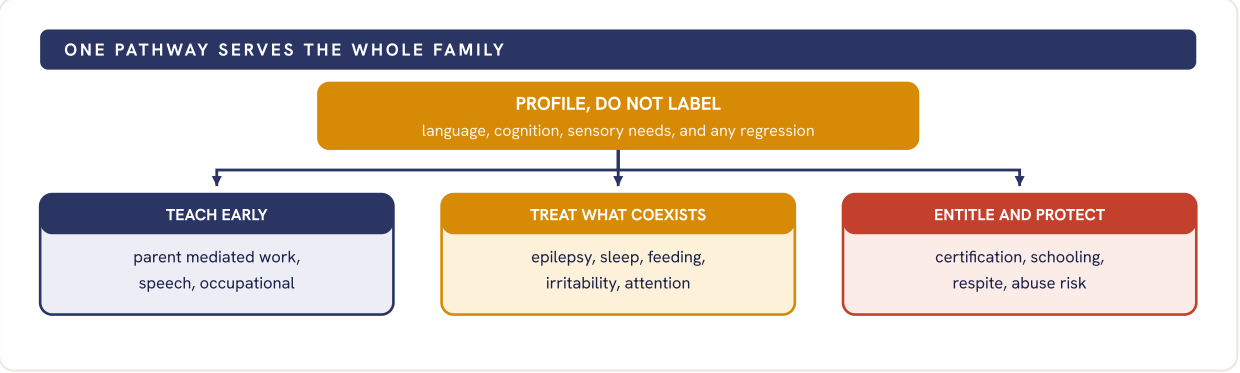


Fig 091.1 The profile decides the plan, and the same three arms serve every member of the family. Regression is the one branch that goes to investigation first.

- **What changes the plan.** Any loss of acquired skill needs electroencephalography and neurological review before a developmental label. Epilepsy appears in a fifth to a third and peaks in adolescence. Certification uses the Indian Scale for Assessment of Autism, and the National Trust covers autism for insurance and guardianship.

WHAT EARNS THE MARKS

- **The family named with the triad,** and the fact that current systems have collapsed it into one spectrum.
- **One identifying feature per member,** with Rett taken out of the group.
- **Regression routed to investigation,** not to a developmental label.

SEE ALSO Slot III - 89 clinical features Slot III - 93 Rett syndrome SPRINT

Day 21, Intellectual disability & autism spectrum disorder

III - 92

Mention clinical features common to Autistic Spectrum Disorders. Enumerate diagnostic criteria of Childhood Autism according to DSM. [10]

10 | AUTISM SPECTRUM DISORDER | RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1 The axis	What is common to all of it, before any criterion
3 Common features	Present at every severity, whatever the language level
4 The criteria, enumerated	A to E, with the counting rule on each
2 Severity and specifiers	Graded per domain; and how the older system differed

COMMON TO THE WHOLE SPECTRUM

Whatever the intelligence or the language level: reduced social reciprocity, impoverished nonverbal communication, difficulty forming and keeping relationships, insistence on sameness, narrow intense interests, and atypical sensory reactivity. Severity changes how much support is needed, not which domains are affected.

CRITERION	WHAT MUST BE PRESENT	COUNTING RULE
A. Social	Reciprocity; nonverbal communication; developing and keeping relationships	**All three, across contexts**
B. Repetitive	Stereotyped movement or speech; insistence on sameness; fixated interests; sensory reactivity	**Two of the four**
C. Onset	Present in the early developmental period, possibly masked until demand rises	Required
D. Impairment	Clinically significant, in social, occupational or other important areas	Required
E. Not better explained	By intellectual disability or global developmental delay alone	Required

- **Severity and specifiers.** Three levels of support, graded separately for the social and the repetitive domain. Specify with or without intellectual impairment, with or without language impairment, an associated medical or genetic condition, another neurodevelopmental disorder, and catatonia.
- **How the older system differed.** The earlier manuals used a triad, counted language as a separate strand, and named separate disorders. The current system folds language into a specifier, merges the disorders into one spectrum, and requires the counting rule above.

WHAT EARNS THE MARKS

- **Common features stated before the criteria.** The question asks for both, in that order.
- **All five criteria lettered,** with the counting rule written on A and B.
- **Severity graded per domain,** not as one global label.
- **The triad to dyad change named.** It shows which system the answer is written from.

SEE ALSO Slot III - 89 clinical features described Slot III - 91 pervasive developmental disorders SPRINT
Day 21, Intellectual disability & autism spectrum disorder

III - 93

Rett's syndrome. [10]

10 AUTISM SPECTRUM DISORDER KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The genetics	Gene, inheritance, and why boys are rarely seen
4	The four stages	Age and the change that defines each one
2	Diagnostic criteria	The four main criteria, and the supportive signs
2	Management	What is treatable, and what is only supported

THE DEFINITION

An X linked dominant neurodevelopmental disorder caused by mutation in the MECP2 gene, almost always arising new rather than inherited, seen almost exclusively in girls because affected boys usually do not survive infancy. Development is normal for the first six months or so, then stalls and regresses.

STAGE	AGE	WHAT DEFINES IT
Stagnation	6 to 18 months	Development slows, head growth decelerates, interest fades
Regression	1 to 4 years	**Purposeful hand use and spoken language are lost; hand wringing appears**
Plateau	2 to 10 years	Some social contact returns; seizures, breathing dysrhythmia, bruxism
Late motor decline	After 10 years	**Scoliosis, rigidity, loss of walking; eye gaze communication survives**

- **The four main criteria.** Partial or complete loss of acquired purposeful hand skills; loss of acquired spoken language; gait abnormality; and stereotyped hand movements such as wringing, washing or mouthing. Supportive signs include awake breathing disturbance, bruxism, cold small hands and feet, scoliosis, and intense eye contact.
- **Differential and variants.** Separate it from autism spectrum disorder, cerebral palsy, Angelman syndrome and a mitochondrial disorder. Atypical forms exist: a preserved speech variant, an early seizure variant, and a congenital variant, each with its own gene.
- **Management, honestly.** No treatment reverses the disorder. Antiepileptic drugs for seizures, screening for prolonged QT, physiotherapy and bracing for scoliosis, nutrition support, sleep and behaviour work, and communication through eye gaze devices. Genetic counselling matters even though most mutations are new.

WHAT EARNS THE MARKS

- **MECP2 named, with the reason boys are rarely seen.**
- **Four stages with ages,** and the regression stage identified as the diagnostic one.
- **Hand stereotypy described,** not merely called stereotypy.
- **The cardiac screen.** Prolonged QT is the line most answers never reach.

SEE ALSO Slot III - 91 pervasive developmental disorders Slot III - 12 aetiology of intellectual disability

AREA 12 OF 13 · P3-A06

Child assessment, pharmacotherapy and interventions

4	3.2%	III-94	III-97
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 4

REVISION ORDER

The two internet addiction slots share nearly all their content, so take them together. Gender dysphoria and the scales slot are independent and short.

WHAT IS IN THIS AREA

Internet addiction in two slots, gender dysphoria in an adolescent, and the rating scales used in child psychiatry.

III - 94

Describe behavioral addiction and how to manage a adolescent with internet addiction. [5+5]

5 + 5 | CHILD ASSESSMENT, PHARMACOTHERAPY AND INTERVENTIONS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Definition**	A reward behaviour carrying the dependence criteria
3	**What is recognised**	ICD-11 admits two entities, the rest stay proposed
2	**Assessment first**	Quantify the use, screen for what drives it
3	**Management**	The plate: engage, sort, treat, prevent relapse

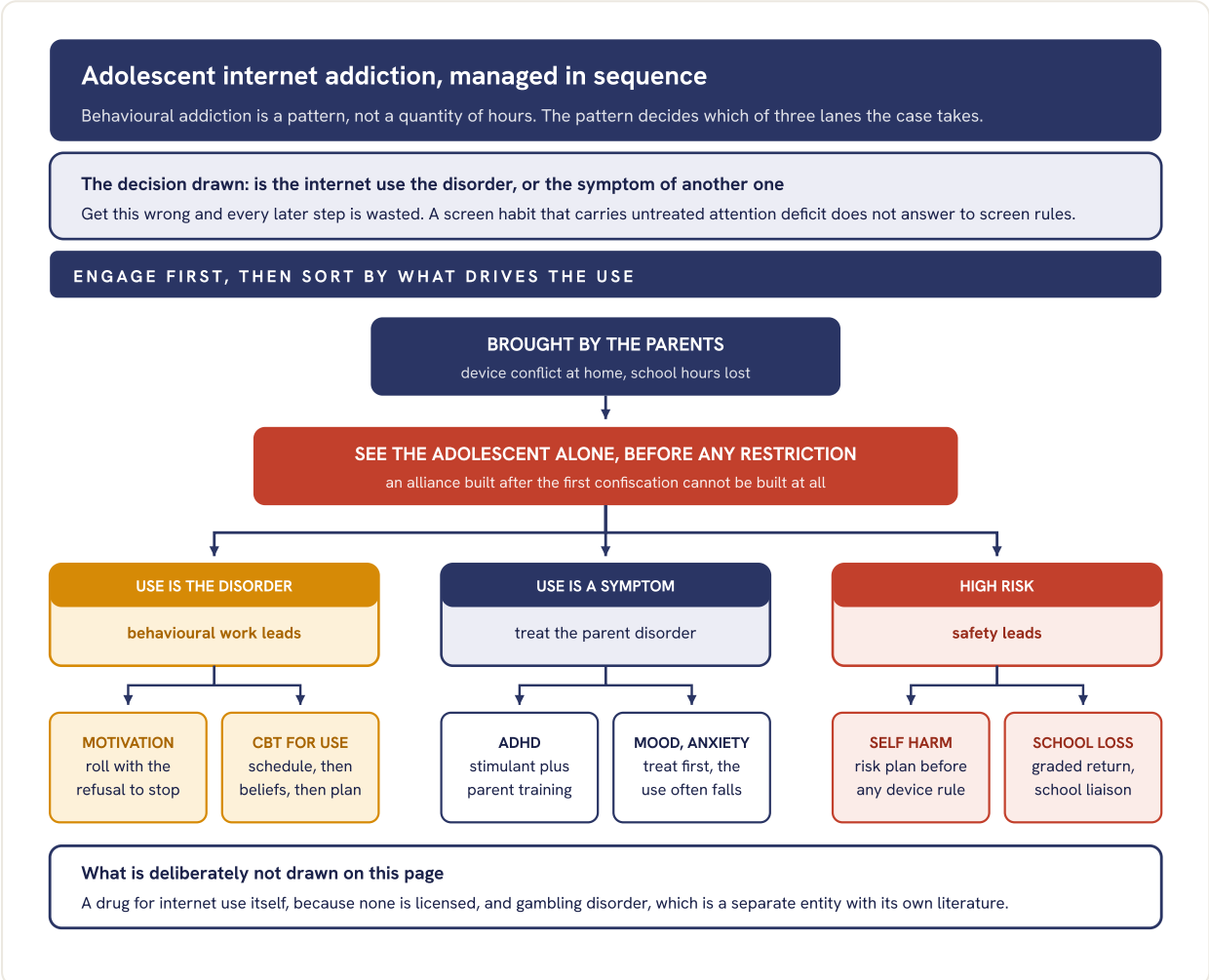


Fig 094.1 The management sequence, with the sorting decision placed above the three lanes so that treating a comorbid disorder is never mistaken for a failure of the behavioural plan.

COLOUR CODE steps and structure · where the marks sit · danger and escalation

- **Behavioural addiction.** A repeated rewarding behaviour that acquires the features of dependence: impaired control, priority over other interests, and continuation despite harm, for at least twelve months.
- **What is recognised.** The ICD-11 grouping of disorders due to addictive behaviours admits exactly two, gambling disorder and gaming disorder. Shopping, exercise and food remain proposed, not classified.

PITFALL

Hours online are not the diagnosis. Six holiday hours without impairment is no disorder; two hours in an adolescent who has stopped eating with the family and stopped attending school may be. Function, not duration.

WHAT EARNS THE MARKS

- **The dependence criteria applied to a behaviour,** not a description of heavy use.
- **Two recognised entities named, and the rest named as proposed.** That distinction is the whole of part one.
- **The sorting decision drawn before treatment,** as the plate does.
- **An engagement step placed ahead of any restriction.** Most answers begin with rules and lose the adolescent.

SEE ALSO Slot III - 96 internet addiction as a disorder Slot III - 97 rating scales in children **SPRINT** Day 24, Child psychiatry

III - 95

What is gender dysphoria? Discuss its clinical presentation and management in a young adolescent. [2+3+5]

2 + 3 + 5 | CHILD ASSESSMENT, PHARMACOTHERAPY AND INTERVENTIONS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	Incongruence plus distress, and where each system puts it
3	Presentation	Onset, the adolescent picture, what travels with it
3	The staged pathway	Assess, social, suppression, hormones, in that order
2	Law and ethics	Self-identification, and the prohibition on conversion

- **Gender dysphoria.** Distress from a marked incongruence between experienced gender and assigned sex, for at least six months. ICD-11 moved the construct out of mental disorders as gender incongruence; DSM-5 keeps gender dysphoria.
- **Presentation at this age.** Insistence rather than preference, distress at pubertal change, binding or tucking, avoidance of changing rooms, and a request for a new name and pronoun.
- **What travels with it.** Depression, self-harm, anxiety, autism co-occurrence, and family rejection, the strongest predictor of poor outcome.

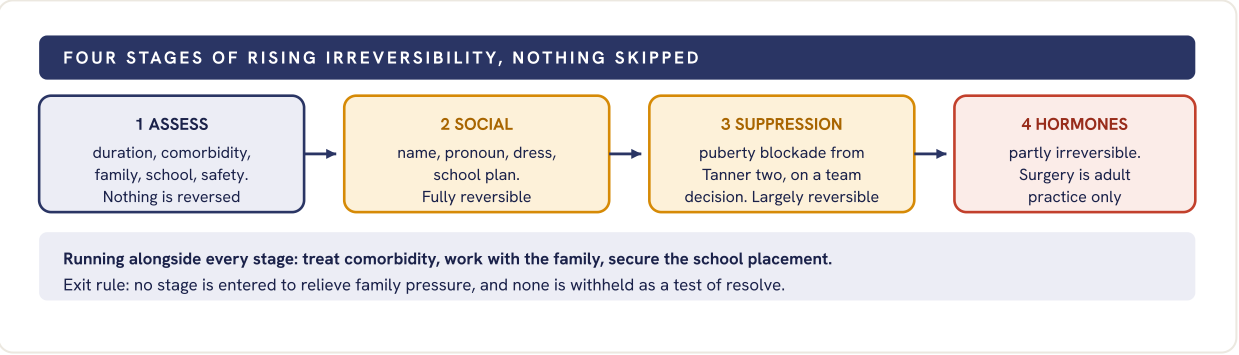


Fig 095.1 The pathway drawn as four stages of rising irreversibility, with the supports that run through all four kept on the foot strip so they are not read as a later phase.

PITFALL

Any attempt to change the young person's gender identity is prohibited. Conversion practice was held unlawful by the Madras High Court in 2021 and notified as professional misconduct by the National Medical Commission in 2022. Self-identification is protected by NALSA, 2014.

WHAT EARNS THE MARKS

- **Incongruence and distress named as two separate requirements,** with the ICD-11 relocation stated.
- **The four stages in order of irreversibility,** as the figure sets them out.
- **Comorbidity and family work placed alongside, not after.** Suicidality is the exam-critical line.
- **Self-identification named to NALSA, 2014,** and the conversion prohibition to an Indian authority.

SEE ALSO Slot III - 97 assessment instruments in children **SPRINT** Day 24, Child psychiatry

III - 96

a) Is internet addiction a mental disorder? b) What are the psychological and social problem faced by them? c) How to treat an adolescent patient of internet addiction? [2+2+2+4]

2 + 2 + 2 + 4 | CHILD ASSESSMENT, PHARMACOTHERAPY AND INTERVENTIONS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The status	Answer yes and no, and say who says which
2 Psychological harm	Sleep, mood, attention, self-worth
2 Social harm	School, family, peers, money, safety online
4 Treatment	Three tiers, none of them a device ban

- **The honest answer is qualified.** Gaming disorder is a recognised ICD-11 diagnosis, so a specified form of internet use is a mental disorder. Generalised internet addiction is not: DSM-5 lists internet gaming disorder only as a condition for further study.

DOMAIN	WHAT IS SEEN	WHAT IT IS MISTAKEN FOR
Sleep and mood	Delayed sleep phase, morning unrousability, irritability on interruption, low mood offline	Laziness, or an independent depressive episode
Attention	Poor sustained attention offline, falling grades	**Attention deficit disorder, which may equally be the cause**
Family and school	Escalating conflict, deceit about hours, aggression on device removal, absence, offline friendships dropped	Conduct disorder, or social anxiety, which is often genuinely present
Money and safety	In-game spending, loot box gambling, grooming and cyberbullying exposure	Ordinary adolescent spending

The amber cell is the discriminator: attention deficit predating the heavy use is treated first, and the use often settles behind it.

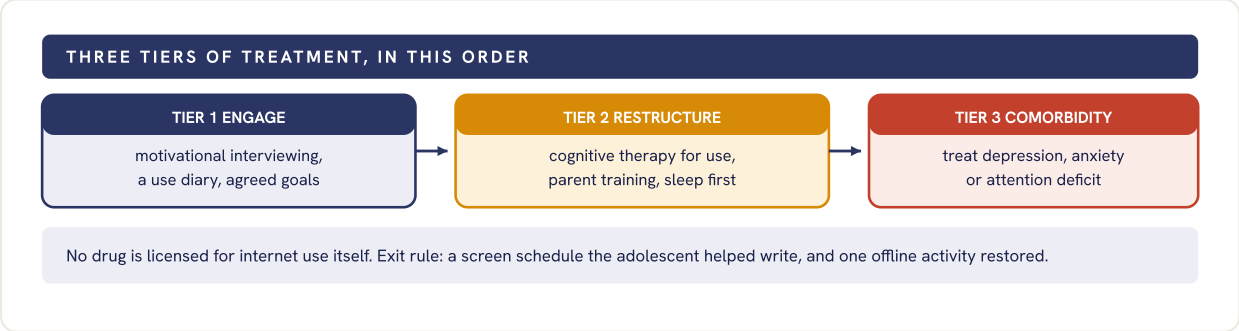


Fig 096.1 The treatment tiers, with the absence of any licensed pharmacotherapy stated on the figure so that medication is not read as a fourth tier.

WHAT EARNS THE MARKS

- **Part a answered both ways, with the classification named.** A flat yes or a flat no both lose marks.
- **Psychological and social harms kept apart,** because the question asks for two things.
- **Treatment tiered rather than listed,** with engagement placed before restriction.
- **The line that no drug is licensed for the behaviour itself.**

SEE ALSO Slot III - 94 behavioural addiction and its management **SPRINT** Day 24, Child psychiatry

III - 97

a) What is Behavioural and Emotional Rating Scale? b) Enumerate the rating scale for ADHD? Describe any one such. c) How to assess autism in a 4 year old child? [2+2+2+4]

2 + 2 + 2 + 4 | CHILD ASSESSMENT, PHARMACOTHERAPY AND INTERVENTIONS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The strength scale	What BERS measures, and why that is unusual
2	The ADHD set	Grouped by informant, not listed at random
2	One described	Items, informants, scoring, what it cannot do
4	Autism at four	History, observation, instrument, certification

THE DEFINITION

A standardised, strength-based measure of a child's emotional and behavioural competence, against a field of problem-based instruments. Five domains: interpersonal strength, family involvement, intrapersonal strength, school functioning and affective strength, on parent, teacher and youth forms.

INSTRUMENT	INFORMANT	SHAPE	BEST USED FOR
Conners scales	Parent, teacher, self	Broad, normed, long and short forms	Diagnosis and severity together
Vanderbilt scales	Parent, teacher	Symptom items plus performance and comorbidity screens	Free clinic and school use
ADHD Rating Scale	Parent, teacher	Eighteen criterion items, nought to three	Direct criterion mapping
Child Behaviour Checklist	Parent, teacher, self	Broad-band, with an attention problems scale	Screening across disorders

The axis is the informant, because ADHD requires symptoms in more than one setting and no single rater can supply that.

- **One described.** The Vanderbilt parent form carries the eighteen criterion items rated nought to three, plus screens for oppositional, conduct and anxious-depressive symptoms and separate performance items. A domain counts only when six of nine items score two or three and performance is impaired. It supports a diagnosis; it never makes one.
- **Autism at four, in sequence.** History with regression enquiry, hearing and vision testing, examination for dysmorphology; then the autism diagnostic observation schedule at the child's language level with the parent interview; then severity, adaptive and cognitive measures, since intellectual disability is the commonest co-occurrence.
- **Keep certification separate.** The Indian Scale for Assessment of Autism is the instrument notified for disability certification, and it is scored for that purpose, not for diagnosis.

WHAT EARNS THE MARKS

- **The strength-based framing of BERS, named as its distinguishing feature.**
- **ADHD scales grouped by informant,** which is the axis the table divides on.
- **One instrument described with items, scoring and its limit.**
- **Autism given as a sequence,** certification instrument kept apart from diagnostic ones.

SEE ALSO Slot III - 48 domains and scoring of IDEAS **SPRINT** Day 24, Child psychiatry

AREA 13 OF 13 · P3-A01

Normal child development

3	0.7%	III-98	III-100
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 3

REVISION ORDER

Three unrelated slots, three unrelated answers. None of them is long. This is the area to read on the night before, not the week before.

WHAT IS IN THIS AREA

Temperament and its implications, Piaget, and adverse childhood experiences. Three slots, the smallest area in the book.

III - 98

What is temperament and its implication in the development of behavioral and emotional problems in child? [10]

10 | ASKED AT 15 MARKS | NORMAL CHILD DEVELOPMENT

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	Style of behaviour, constitutional, early, measurable
2 The nine dimensions	Named, then clustered into the three constellations
4 Implications	Fit first, then the externalising and internalising routes
2 Qualifiers	Stability, cultural loading, and what it cannot predict

THE DEFINITION

Temperament is the constitutionally based, early appearing style of behaviour: the how of behaviour rather than the what or the why. Thomas and Chess described nine dimensions in the New York Longitudinal Study. Moderately heritable, moderately stable from the second year, rated by parent report.

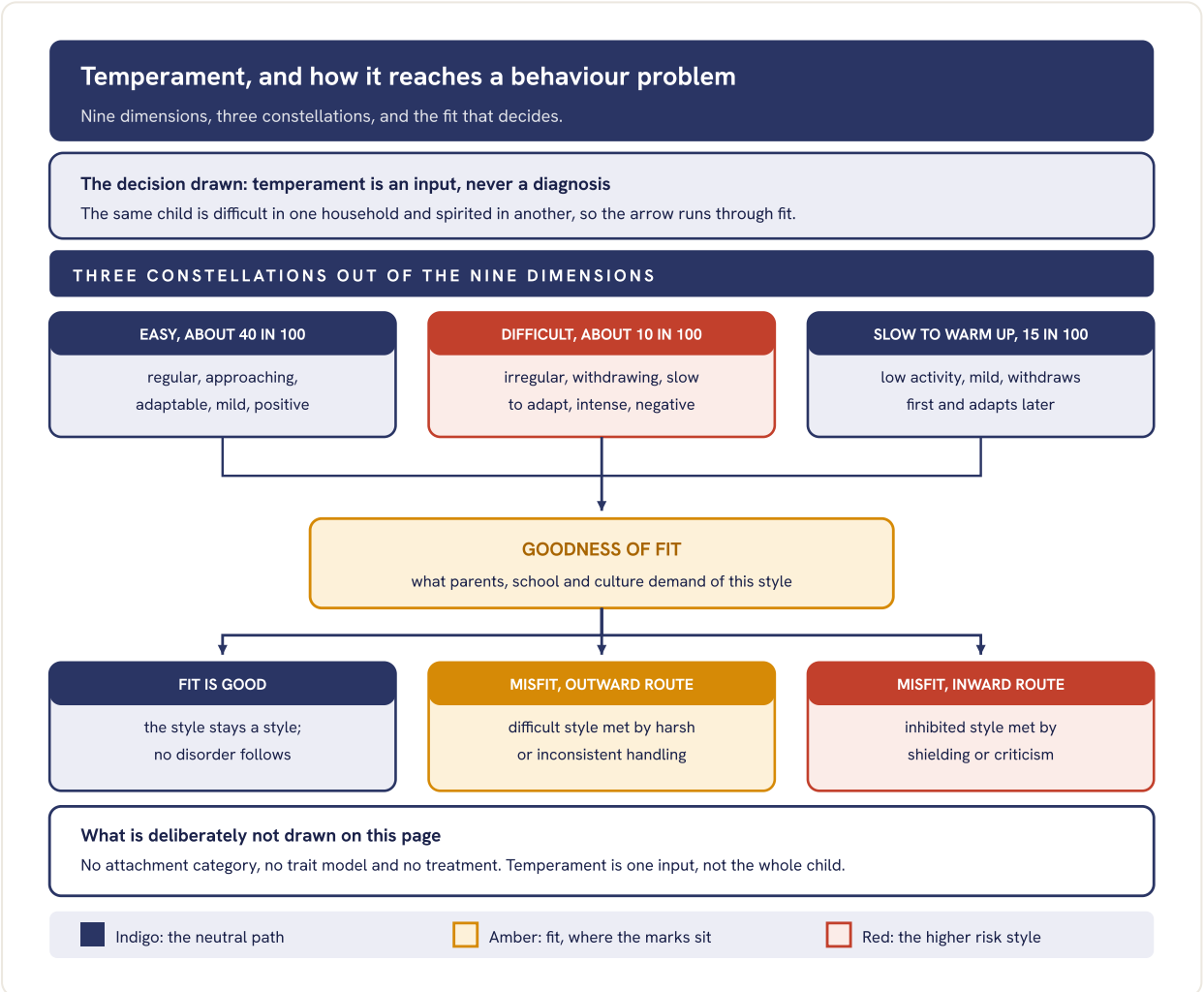


Fig 098.1 The nine dimensions clustered into three constellations, routed through goodness of fit, and the three outcomes that follow.

- **The nine dimensions.** Activity, rhythmicity, approach or withdrawal, adaptability, intensity, threshold, mood, distractibility, persistence. About a third of the original cohort fitted none of the three clusters.
- **Why fit carries the marks.** Difficult temperament predicts oppositional and conduct problems mainly where handling is harsh or inconsistent. Behavioural inhibition, described by Kagan, predicts social anxiety mainly where novelty is repeatedly avoided for the child.
- **The honest limit.** Effects are small and probabilistic. Indian ratings load differently on rhythmicity and approach, so use an Indian instrument such as the Malhotra Temperament Measurement Schedule.

WHAT EARNS THE MARKS

- **Definition as a style, then the nine dimensions named.** Naming three clusters without the dimensions is half the definition.
- **The fit arrow.** An answer that runs temperament straight to disorder cannot earn the four implication marks.
- **The outward and inward routes kept apart.** Different temperaments, different handling, different outcomes.
- **One honest limit.** Small effect, culture-bound ratings, no prediction for an individual child.

SEE ALSO Slot III - 100 adverse childhood experiences Slot III - 51 oppositional defiant disorder **SPRINT** Day 20, Normal child development & milestones

III - 99

Jean Piaget. [10]

10 NORMAL CHILD DEVELOPMENT KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The framework	Constructivism, and the four process words
4	The four stages	Age, what is gained, one bedside test each
2	Clinical use	Explaining illness, consent, choosing a therapy
2	Criticism	What later work overturned, and who answered him

THE FRAMEWORK IN ONE BLOCK

A constructivist stage theory: the child builds knowledge by acting on the world. Four process words carry the theory. A schema is a unit of knowledge; assimilation fits new experience into an existing schema; accommodation alters the schema to fit the experience; equilibration is the drive that moves the child from one stage to the next. Stages are invariant in order and qualitative, not a matter of knowing more.

STAGE	AGE	WHAT IS GAINED	TEST AT THE BEDSIDE
Sensorimotor	0 to 2	Circular reactions, means and ends, object permanence, deferred imitation	Hide a toy under a cloth and watch for the search
Preoperational	2 to 7	Symbols, language, pretend play; still egocentric, animistic, centred, irreversible	Pour water into a taller glass and ask which has more
Concrete operational	7 to 11	Conservation, reversibility, seriation, classification, transitivity	Conservation now passes, but only for present objects
Formal operational	11 upward	Abstract and hypothetical reasoning, systematic testing of possibilities	Give a pendulum problem and watch for one change at a time

Amber cells are the two tests an examiner expects by name: object permanence and conservation.

- **Why a psychiatrist uses it.** It sets the level at which illness is explained, the age at which assent becomes meaningful, and the readiness for a therapy: thought records need concrete operations, so younger children get play, drawing and story instead.
- **Where he was wrong.** Infants show numerical and physical expectations far earlier than the theory allows, transitions are gradual rather than stage-like, and formal operations are neither universal nor reached by every adult. Vygotsky placed the same development in the social zone between child and helper.

WHAT EARNS THE MARKS

- **The four process words, defined.** Assimilation and accommodation as a pair is the commonest place marks are lost.
- **All four stages with ages and one gain each.** A stage list with no age band reads as recall.
- **Object permanence and conservation named as the markers,** with the test each is shown by.
- **One clinical use and one criticism.** They are what separate a top answer from a textbook summary.

SEE ALSO Slot III - 98 temperament Slot III - 100 adverse childhood experiences SPRINT

Day 20, Normal child development & milestones

III - 100

Adverse childhood experiences. [10]

10 NORMAL CHILD DEVELOPMENT KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition and origin	The construct, the study it came from, the score
3 The three domains	Ten categories, grouped, with the dose relation
3 Mechanism	Toxic stress to biology to behaviour, in that order
2 What follows clinically	Enquiry, the Indian legal duty, trauma-informed care

THE DEFINITION

Adverse childhood experiences are potentially traumatic events and chronic stressors occurring before the age of eighteen, counted as a simple score of exposure categories rather than weighted by severity. The construct comes from the Kaiser study reported by Felitti and Anda, which found a graded dose relation between the number of categories endorsed and adult disease and death.

DOMAIN	CATEGORIES COUNTED	WHAT RISES WITH THE SCORE
Abuse	Physical, emotional, sexual	Depression, suicide attempt, post-traumatic stress
Neglect	Physical, emotional	Attachment difficulty, poor emotion regulation
Household adversity	Parental mental illness, substance use, incarceration, violence between adults, separation or divorce	Substance use disorder, psychosis, personality difficulty
Dose relation	Score of four or more against a score of zero	Risk rises across most adult health outcomes, not one

Amber cells are the three findings the question is testing. The score counts categories, so it treats one episode and years of exposure alike.

- **Mechanism, in order.** Prolonged activation without a buffering adult is toxic stress: altered hypothalamic pituitary adrenal reactivity, low grade inflammation, epigenetic change at stress-response genes, and altered amygdala and prefrontal development. Health risk behaviour follows as coping, not as cause.
- **Indian frame.** Sexual abuse of a minor carries a mandatory reporting duty under the Protection of Children from Sexual Offences Act 2012. A neglected or abused child is a child in need of care and protection under the Juvenile Justice Act 2015, referred to the child welfare committee.
- **The trap.** The score is a population risk marker, not a diagnosis and not a prediction for the child in front of you. Enquire routinely, never in a first interview with the parent present, and never repeat the history for teaching.

WHAT EARNS THE MARKS

- **The definition with the age boundary and the counting rule.** A list of adversities with no score is half the construct.
- **Three domains, then the dose relation stated as graded.**
- **Mechanism written as a chain,** from buffering failure to biology to behaviour.
- **The two Indian statutes named.** Mandatory reporting is the line most answers never reach.

SEE ALSO Slot III - 98 temperament Slot III - 37 mother infant relationship disorders

PAPER III APPENDIX, PAGE ONE OF TWO

Ten sets, ten sittings: Paper III

The same hundred Paper III questions, dealt into ten papers of ten. Each cell gives the slot and the page it is answered on.

Read down a row to sit a paper. The deal is not random: the hundred are ranked by score and dealt one question from each rank decile into each set, so every set holds one of the ten strongest, one of the ten weakest, and one from each band between. That is why the sets are comparable to each other and why none of them is an easy paper.

SET	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10
III-S1	III-50 p 337	III-33 p 317	III-54 p 342	III-06 p 283	III-43 p 329	III-96 p 396	III-30 p 312	III-85 p 380	III-86 p 382	III-98 p 399
III-S2	III-89 p 386	III-68 p 359	III-13 p 293	III-05 p 282	III-64 p 354	III-16 p 296	III-20 p 300	III-84 p 379	III-80 p 374	III-93 p 391
III-S3	III-31 p 314	III-52 p 340	III-35 p 319	III-04 p 281	III-69 p 360	III-29 p 311	III-45 p 331	III-37 p 321	III-81 p 375	III-92 p 390
III-S4	III-11 p 290	III-22 p 303	III-94 p 393	III-03 p 280	III-65 p 355	III-36 p 320	III-10 p 288	III-70 p 361	III-57 p 345	III-91 p 389
III-S5	III-01 p 278	III-41 p 326	III-12 p 291	III-62 p 352	III-66 p 356	III-28 p 310	III-17 p 297	III-38 p 322	III-72 p 364	III-74 p 366
III-S6	III-51 p 339	III-02 p 279	III-76 p 369	III-61 p 351	III-55 p 343	III-15 p 295	III-97 p 397	III-71 p 363	III-87 p 383	III-88 p 384
III-S7	III-75 p 368	III-90 p 387	III-53 p 341	III-26 p 308	III-44 p 330	III-83 p 378	III-18 p 298	III-56 p 344	III-78 p 372	III-58 p 346
III-S8	III-67 p 358	III-34 p 318	III-23 p 304	III-25 p 306	III-07 p 285	III-09 p 287	III-77 p 371	III-49 p 335	III-39 p 323	III-73 p 365
III-S9	III-59 p 348	III-21 p 302	III-24 p 305	III-42 p 328	III-14 p 294	III-27 p 309	III-46 p 332	III-48 p 334	III-79 p 373	III-100 p 402
III-S10	III-32 p 316	III-82 p 377	III-60 p 350	III-63 p 353	III-95 p 395	III-08 p 286	III-19 p 299	III-47 p 333	III-40 p 324	III-99 p 401

No set repeats a question, a merged variant of a question, or a near-duplicate, and no set takes more than three questions from any one area.

PAPER III APPENDIX, PAGE TWO OF TWO

How to sit one of these

SET	MARKS	MEAN SCORE	AREAS	ANCHOR OR HIGH	HEAVIEST AREAS IN THE SET
III-S1	100	0.387	8	2	ADHD 2; Perinatal 2; Childhood emotional 1
III-S2	100	0.379	7	2	Intellectual disability 3; Autism spectrum disorder 2; ECT 1
III-S3	100	0.374	8	2	Childhood emotional 3; ADHD 1; Criminal 1
III-S4	100	0.363	9	4	Criminal 2; Intellectual disability 1; Ethics 1
III-S5	100	0.361	7	2	Intellectual disability 2; Geriatric 2; ECT 2
III-S6	100	0.358	8	0	ADHD 2; Perinatal 2; Criminal 1
III-S7	100	0.353	7	3	ADHD 3; Emergency psychiatry 2; Autism spectrum disorder 1
III-S8	100	0.353	6	3	ECT 2; Childhood emotional 2; Ethics 2
III-S9	100	0.346	6	0	Ethics 3; Mental health legislation 3; Geriatric 1
III-S10	100	0.348	8	1	Childhood emotional 2; Geriatric 2; Perinatal 1

Mean score is the selection score of the ten questions in that set, on the same scale used across all four books. The spread between the strongest and the weakest set is small by construction.

Three hours, ten questions, one hundred marks. That is the shape of the real paper, so it is the shape of these. Eighteen minutes a question leaves you twenty minutes at the end, which is roughly what you will have and roughly what you will need.

Write the skeleton first, for all ten. Spend the first twenty minutes writing nothing but mark splits and section headings across all ten answers. The map on each page shows what that skeleton should look like, so you can mark your own against it before you have written a line of content. Most marks lost in this examination are lost to allocation, not to ignorance.

Then mark yourself against the anchors. Every map ends with three or four things an examiner ticks. Those are the marking scheme, as close as this book gets to one. Count how many you hit before you read the page, because reading it first will convince you that you knew it.

Sit them in order, or sit them backwards. Set one holds the strongest question in the book and set ten the weakest, but the decile deal keeps the mean scores close, so any set is a fair paper. In order simply means the first one you sit is the one most like the paper you will get.

What a set is not. It is not a mock paper from any board, it is not a leak, and it is not a prediction. It is ten questions a board set, arranged so that ten of them look like a paper. Sit five and you will have written a hundred answers in the shape the examination wants, which is the only preparation that has ever reliably worked.

PART FOUR OF FOUR

PAPER IV

Brain, Drugs and the Community

The organic paper: epilepsy, the dementias, delirium, stroke and head injury, the movement disorders, the endocrine and infective presentations, psychopharmacology and neurostimulation, the medical wards, and community and public mental health.

15	100	78	8	13.3%
AREAS, IN WEIGHT ORDER	ANSWER MAPS	SITTINGS COUNTED	EXAMINING BOARDS	HEAVIEST AREA SHARE

ANCHOR 18 · HIGH 15 · RECURRENT 2 · SINGLE 65

Heaviest area in this paper: Community and public mental health. The weightage chapter overleaf shows how every area in Paper IV was weighted, and what the weight could not promise.

THE WEIGHTAGE CHAPTER, PAGE ONE OF THREE

Where the marks have gone

Nothing in this book is a prediction. It is a count of 78 sittings from 8 examining boards, and a statement of where those boards have put their marks in Paper IV.

Fifteen areas, weighted. The university boards are pooled by how much each is trusted, DNB is held out and given a fixed thirty percent, and the resulting weight becomes a share of one hundred and then a whole number of slots. The ledger of which boards were counted, how their papers reached us, and how far each one is trusted, is on the page opposite.

AREA, IN WEIGHT ORDER	UNIV POOL	DNB	WEIGHT	SHARE	SLOTS
01 Community and public mental health	0.363	0.562	0.423	13.34%	11
02 Dementias: types and differential	0.388	0.304	0.362	11.44%	10
03 Consultation-liaison and systemic medicine psychiatry	0.364	0.214	0.319	10.07%	9
04 Clinical neurology, headache and sleep neurology	0.240	0.287	0.254	8.01%	8
05 Stroke, TBI and focal brain syndromes	0.267	0.215	0.251	7.93%	8
06 Movement and degenerative disorders in psychiatry	0.241	0.140	0.211	6.66%	7
07 Endocrine and metabolic psychiatry	0.262	0.048	0.198	6.25%	6
08 Recent advances and landmark studies	0.091	0.414	0.188	5.92%	6
09 Drug emergencies, interactions, TRD and neurostimulation	0.167	0.200	0.177	5.59%	6
10 Psychopharmacology: principles and drug classes	0.118	0.252	0.158	5.00%	5
11 Epilepsy and psychiatry	0.197	0.055	0.154	4.87%	5
12 Nutritional, toxic and metabolic encephalopathies	0.160	0.067	0.133	4.19%	5
13 Neuro-infective and autoimmune neuropsychiatry	0.181	0.000	0.127	4.00%	5
14 Delirium and amnestic syndromes	0.156	0.048	0.123	3.89%	5
15 Cognitive assessment and dementia management	0.128	0.000	0.090	2.84%	4

Univ pool is the trust-weighted verdict of the seven university boards; DNB is the national paper, held out. Weight = 0.70 times the pool plus 0.30 times DNB. Share is that weight as a fraction of Paper IV. Slots are the share turned into whole questions by largest remainder, with a floor of two and a cap of twenty; Paper IV reached neither.

THE WEIGHTAGE CHAPTER, PAGE TWO OF THREE

How the hundred were picked

BOARD	SITTINGS COUNTED	HOW THE PAPERS REACHED US	TRUST
RGUHS	12	Hand transcription and public mirrors of the printed papers	0.806
KUHS	9	Official university portal PDFs	0.600
TN-MGRMU	35	Public archive of the printed papers, exact sitting dates recovered	0.480
KNRUHS	3	Official university portal PDFs	0.300
MUHS	1	Watermark-confirmed printed papers, read by OCR; one of the two carries a Paper IV sitting. A third-party recurrence compilation was tested against them, matched none, and was removed from the sitting count	0.267
NTRUHS	2	Mirror text of the printed papers	0.160
WBUHS	2	Public archive of the printed papers	0.180
DNB	10	Official NBE material; 32 of 40 files verified by checksum against the board's own published path	fixed 0.30

Trust is how far a board sets the university pool: its standing, its number of sittings, and the quality of its evidence. DNB sits outside the pool. A third-party compilation claiming thirteen further MUHS sittings was tested against the only confirmed MUHS papers inside its own window, matched none of them, and was removed from this ledger; its topic distribution still contributes to area mass at reduced quality.

1 Count the sittings

78 sittings from 8 boards, each weighted so that it halves roughly every six years and is reduced again when the paper reached us as a mirror, an archive or a transcription rather than an official file.

2 Weight the areas

Each board reads each area twice: breadth, how often it examines there, and mark share, how much of its marks land there. The two are averaged, the boards are pooled by trust, and DNB is held out at a fixed thirty percent.

3 Turn weight into slots

The fifteen weights become shares of one hundred, then whole slots by largest remainder, with a floor of two and a cap of twenty so no area disappears and none swallows the book.

4 Score every eligible question

Four components: weighted recurrence (45 per cent), recency (30), how many boards have asked it (15), and how well it is shaped as a ten-mark question (10). Normalised across all four papers, so one score means one thing everywhere.

5 Fill the slots

The highest scorers in each area, up to that area's slot count, near-duplicates merged first. All 100 were filled from questions actually set: 18 anchor, 15 high, 2 recurrent, 65 single-appearance.

THE WEIGHTAGE CHAPTER, PAGE THREE OF THREE

What this book cannot promise

A frequency count is a strong prior, not a prophecy. Here is exactly what that sentence costs, stated before you rely on it.

It does not predict a paper. Nothing here says what your board will set. It says where the marks have gone across 78 sittings, which is a different claim and a smaller one. A board that changes its examiner, its syllabus or its habits can leave every number in this book behind in one sitting.

Most questions were seen once. Of the 394 selectable Paper IV questions in the record, 334 appeared in a single sitting. That is not a defect in the counting, it is what happens when eight boards set different papers: the same TOPIC returns constantly while the same QUESTION rarely does. So the selection is driven by AREA weight, where the signal is dense, and each map prints a band rather than a frequency claim about its own question.

The band on a map is a description, not a forecast. Anchor means this question has come back across boards and years. Single means it was set once in the record we hold. A single-appearance question is here because its AREA is heavy, and it is a fair worked example of that area, which is the only thing it is offered as.

Nothing here is invented. All 100 slots in this book are questions a board actually set. Where a paper in this series cannot fill its slots from observed questions, the shortfall is authored to syllabus and labelled as such on the map. Paper IV has no such slots.

The record is uneven, and openly so. Two of the eight boards set papers in a legacy structure that does not map onto the modern four, so their sittings are counted against all of their papers rather than one. Two more contribute only two sittings each. The trust column on the first page of this chapter is where that unevenness is priced in, and it is the number to argue with if you want to argue with the weighting.

No board has endorsed this. Board names appear on every page because a count that hides its sources is not a count. None of them was asked, and none of them is responsible for anything in this book.

AREA 01 OF 15 · P4-A14

Community and public mental health

11	13.3%	IV-01	IV-11
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 3 SINGLE 8

REVISION ORDER

The two Act slots together, they are one answer split: the critique first, then the modes of admission. The three programme slots next as a block, District, one lakh and school, because they share a planning spine. Rehabilitation and the mental health promotion slot after that. The media, pandemic, adolescent suicide and LGBTQIA+ slots are independent of all of it and each stands alone.

WHAT IS IN THIS AREA

The District Mental Health Programme, a mental health programme planned for a population of one lakh, and the school mental health programmes with two Indian ones named. The Mental Healthcare Act in two slots, one a critique against the 1987 Act and one on the modes of admission. Then the principles and practice of rehabilitation, mental health defined with the strategies for promoting it, electronic media and its status in India, adolescent parasuicide and suicide with the legal and social issues, substance use and behavioural addictions during the pandemic with the theories of gambling, and the myths about LGBTQIA+ identities with the tenets of affirmative counselling.

IV - 01

Write a note on the District Mental Health Programme. [10]

10 COMMUNITY AND PUBLIC MENTAL HEALTH

DNB, KUHS, NTRUHS, RGUHS, TN-MGRMU - 7 APPEARANCES, 7 OF 78 SESSIONS

SKELETON

2	**Where it came from**	The national programme, the district pilot, the years
2	**What it is meant to do**	Objectives, stated as objectives
4	**The components**	The original core, then what was added
2	**The honest critique**	Manpower, funds, and where the service actually sits

THE LINEAGE, IN ONE BLOCK

The National Mental Health Programme was launched in 1982. Its district arm, the District Mental Health Programme, began in 1996 and was modelled on the district pilot run at Bellary in Karnataka from 1985. The idea both rest on is the same: mental health care delivered through the general health system, by people who are already there, rather than through a separate specialist service.

- **Objectives.** Minimum mental health care available and accessible to all, especially the underserved; mental health knowledge applied within general health care and social development; and community participation in the service rather than passive receipt of it.
- **The original components.** Early detection and treatment through the general health system; training of medical officers and health workers; public awareness through information, education and communication; monitoring and evaluation; and community participation.
- **What was added later.** A district mental health team with a psychiatrist, clinical psychologist, psychiatric social worker and psychiatric nurse; a small inpatient unit at the district hospital; life skills education and counselling in schools; college counselling; workplace stress programmes; and suicide prevention services.
- **The critique.** The manpower the design assumes does not exist in most districts, so the team is rarely complete. Allocated funds are frequently unspent. The service concentrates at the district hospital rather than at the primary health centre, which is the level the programme was written for. And the programme runs vertically, so it competes with the general health system it was meant to enter.

WHAT EARNS THE MARKS

- **Both years, and the district pilot named,** which almost no answer supplies.
- **Objectives kept separate from components.** Merging them is the commonest structural error here.
- **The later additions listed as additions,** which shows the programme is known as it stands rather than as first written.
- **A critique naming manpower and unspent funds,** not a closing line about the need for more awareness.

SEE ALSO Slot IV - 07 planning a programme for a population Slot IV - 08 promotion of mental health SPRINT Day 39, Community psychiatry and public health

IV - 02

Write about the principles and the practice of rehabilitation of psychiatric patients. [5+5]

5 + 5 | COMMUNITY AND PUBLIC MENTAL HEALTH

DNB, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	Definition and target	Impairment, disability, handicap, and which is treated
3	The principles	Early, functional, negotiated, in the real setting
3	The practice	Assessment, skills, family, work, and where it happens
2	The Indian frame	Family as the service, and the disability certificate

DEFINITION, AND WHAT IT IS AIMED AT

Rehabilitation helps a person with a psychiatric disability reach and hold the best level of independent functioning their illness allows. Impairment is the symptom, disability is the lost ability, handicap is the social disadvantage that follows. Treatment aims at the first; rehabilitation aims at the second and third. Saying so is the definition mark.

- **The principles.** Begin alongside treatment, not after it. Assess residual ability rather than diagnosis. Set the goal with the person, not for them. Train in the setting where the skill will be used. Work in the least restrictive place that will hold the person. Measure function, not symptoms. Expect years.
- **The practice.** Functional assessment and disability rating; skills training for daily living and for social situations; cognitive remediation where thinking is the barrier; family psychoeducation, which is the highest yield intervention available in most Indian settings; and vocational work running from sheltered activity to a real job with support.
- **Where it happens.** Day care centres, halfway homes, long stay homes for the small number who need them, self-help and peer groups, and, for almost everyone, the family home.
- **The Indian frame.** The family is the rehabilitation service, because institutional capacity is small, and the plan must be written for a family that has its own work to do. The Rights of Persons with Disabilities Act 2016 recognises mental illness as a disability, so certification, benefits and reservation are part of the rehabilitation plan and not an afterthought.

WHAT EARNS THE MARKS

- **The three level distinction stated before anything else,** because it defines what rehabilitation targets.
- **Principles written as principles,** not as a second list of activities.
- **The family named as the service,** which is what makes the answer Indian rather than translated.
- **Disability certification included in the plan,** which most answers omit entirely.

SEE ALSO Slot IV - 60 recent advances in psychological rehabilitation Slot IV - 01 the District Mental Health Programme

SPRINT Day 39, Community psychiatry and public health

IV - 03

Critically evaluate the Mental Health Care Act 2017. What are the major changes from the Mental Health Act 1987? [10]

10 | ASKED AT 15 MARKS | COMMUNITY AND PUBLIC MENTAL HEALTH

KUHS, TN-MGRMU - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2	**The change of principle**	From licensing and custody to capacity and rights
4	**The journey, step by step**	The table, with the 1987 position beside it
2	**The critique**	Machinery, capacity assessment, and money
2	**What it does not cover**	The exclusions, stated plainly

STEP	UNDER THE 2017 ACT	UNDER THE 1987 ACT	WHO ACTS
1	Capacity to decide is presumed, and assessed when doubted	No capacity concept at all	The treating team
2	An advance directive and a nominated representative may be registered	Neither existed	The person
3	Independent admission where capacity is present	Voluntary admission, or a reception order	The person
4	Supported admission where capacity is absent, time limited	Reception order by a magistrate, open ended	Two independent examiners
5	The Mental Health Review Board reviews and hears appeals	The judicial magistrate	The Board

- **The other major changes.** Section 18 opens with a right to access mental healthcare rather than with the licensing of asylums. Section 21(4) requires insurers to cover mental illness on the same basis as physical illness. Section 95 bars electroconvulsive therapy without anaesthesia and muscle relaxants, and in minors without Board permission. Section 115 presumes severe stress in a person who attempts suicide, so that person is not tried or punished.
- **The critique.** The machinery is heavy: in several States the Boards and Authorities were slow to constitute and remain under-resourced. Advance directives are rarely made, and the route to registering and honouring one is thin. Capacity assessment asks for time and training a lone district psychiatrist does not have. A right of access created without matching funds shifts the failure onto the clinician who cannot deliver it.

WHAT THE ACT DOES NOT COVER

It does not fund the services it creates a right to. It says almost nothing about care outside registered establishments, which is where most patients are, and nothing about the family carer, who does most of the work. Disability benefits and certification sit under the Rights of Persons with Disabilities Act 2016, not here. Criminal responsibility remains a separate question entirely.

WHAT EARNS THE MARKS

- **Capacity named as the organising principle,** which is what actually changed.
- **Sections cited for the four headline provisions,** not paraphrased.
- **A critique with named weaknesses,** since critically evaluate is not satisfied by praise.
- **The exclusions strip,** which separates a top answer from a competent summary.

SEE ALSO Slot IV - 04 modes of admission under the Act SPRINT Day 39, Community psychiatry and public health

IV - 04

Discuss the modes of admission under the Mental Health Care Act. [10]

10 | COMMUNITY AND PUBLIC MENTAL HEALTH | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The organising principle	Capacity decides the route, not the diagnosis
4	The modes	Section, actor and limit, in order
2	Safeguards	Notice, review, appeal, and the right to be told
2	Outside the scheme	What no admission mode reaches

STEP	MODE	WHO ACTS	LIMIT OR CONDITION
1	Independent admission, Section 86	The person, who has capacity and asks	Discharge on request
2	Admission of a minor, Section 87	The nominated representative, after two assessments	Separate ward, an attendant stays, Board review
3	Supported admission, Section 89	Two independent examiners, one a psychiatrist	Up to thirty days, the Board notified
4	Supported admission beyond thirty days, Section 90	The Board, on continued need	Up to ninety days, then further reviewed periods
5	Emergency treatment, Section 94	Any registered medical practitioner	Up to seventy-two hours, to prevent harm

Steps 1 and 2 turn on who asks. Steps 3 and 4 turn on capacity being absent. Step 5 turns on time, and ends whether or not anything has been resolved.

- **The principle, and the safeguards.** The route follows capacity, never the diagnosis: a person with schizophrenia who retains capacity is admitted independently. Every supported admission is notified to the Mental Health Review Board, is time limited, and can be appealed by the person, the representative or any registered organisation. The person must be told the reason for admission and the right to appeal, in a language they understand.

WHAT THE ADMISSION SCHEME DOES NOT COVER

There is no route to compel treatment in an adult who retains capacity and refuses, however unwise the refusal looks. There is no community treatment order in Indian law. And these provisions govern admission to a registered mental health establishment, not admission to a general ward for a physical illness.

WHAT EARNS THE MARKS

- **Capacity named as the sorting rule** before the modes are listed.
- **Section numbers with the actor and the interval,** which is exactly what this archetype is checked on.
- **The seventy-two hour emergency provision,** which answers most often forget is an admission route at all.
- **The exclusions strip,** and particularly the capacitous refusal.

SEE ALSO Slot IV - 03 the Act critically evaluated SPRINT Day 39, Community psychiatry and public health

IV - 05

Discuss the role of electronic media in the management of psychiatric illness. Describe its status in India. [7+3]

7 + 3 | COMMUNITY AND PUBLIC MENTAL HEALTH | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What the term covers	Five distinct things, not one
3	What the evidence supports	Guided against unguided, and video consultation
3	Status in India	The 2020 guidelines and the national helpline
2	The limits	Prescribing, connectivity, privacy, examination

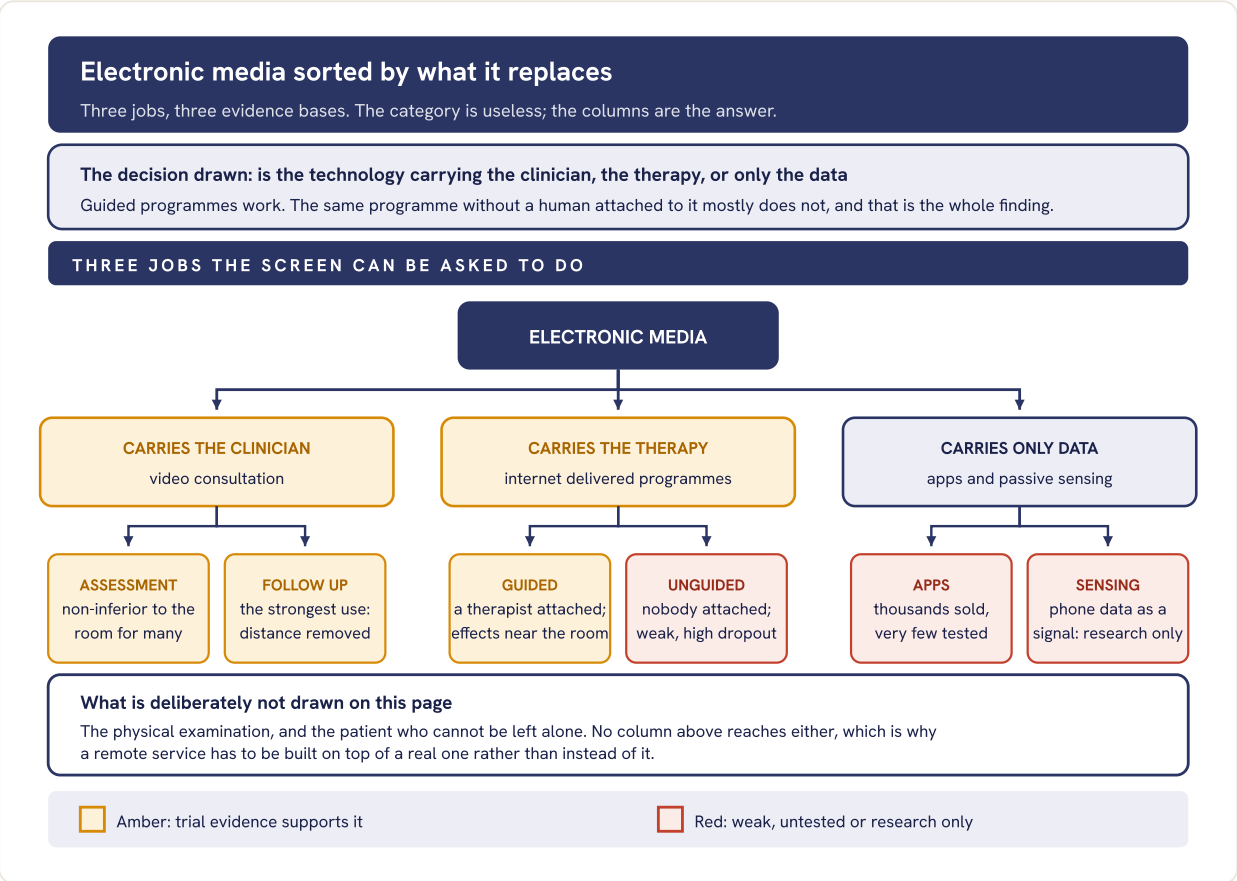


Fig 005.1 Guided and unguided sit in adjacent boxes and in different colours, because that single distinction carries more of the evidence than any other line on the page.

- **Status in India.** The Telemedicine Practice Guidelines of 2020 made remote consultation formally permissible and set out how a registered practitioner may consult and prescribe; telepsychiatry operational guidelines were issued the same year. Drugs on the prohibited list, which includes Schedule X, cannot be prescribed by teleconsultation at all. Tele MANAS, launched in 2022, runs a round the clock national tele mental health helpline with State cells.
- **The limits.** Connectivity and shared handsets, so the poorest patients gain least. No privacy in a one room house. Consent and record keeping requirements that a busy clinic will not meet. And no examination, which matters most in the patients who need it most.

WHAT EARNS THE MARKS

- **The category broken into three jobs** in the first sentence.
- **Guided separated from unguided,** which is the single most examinable finding here.
- **The 2020 guidelines and the prescribing restriction named,** which is the Indian status limb.
- **The limit that the poorest gain least,** rather than a closing line about the digital future.

SEE ALSO Slot IV - 07 planning a programme for a population Slot IV - 01 the District Mental Health Programme

SPRINT Day 39, Community psychiatry and public health

IV - 06

Discuss adolescent parasuicide and suicide. Discuss the legal and social issues related to it. [5+2+3]

5 + 2 + 3 | COMMUNITY AND PUBLIC MENTAL HEALTH

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 **The clinical picture**	Terms, who is at risk, and the means used
2 **Assessment and management**	Seen alone, means restricted, treatment named
3 **The legal steps**	Presumption, reporting duty, protection
2 **The social issues**	Stigma, school, family, media contagion

- **The clinical picture.** Self-harm without clear suicidal intent is far commoner than suicide and is the strongest single predictor of it. Attempts are commoner in girls, deaths in boys, and the difference is mostly method. Risk gathers around previous self-harm, depression, substance use, impulsivity, bullying including online, abuse, academic pressure, and easy access to means.

STEP	WHAT HAPPENS	WHO ACTS	CONDITION OR LIMIT
1	Medical stabilisation, and no discharge before a psychosocial assessment	Emergency team	Before any psychiatric decision
2	Psychosocial assessment, the adolescent seen alone and then with family	Psychiatrist or trained clinician	Every episode, not only severe ones
3	Section 115 applies: severe stress is presumed	The law, not the clinician	Not tried, not punished, care to be provided
4	Disclosed sexual abuse triggers mandatory reporting	The clinician, under POCSO 2012	Overrides the usual confidentiality
5	Safety plan, means restriction, treatment, follow up	Family, with the team	Follow up fixed before discharge

- **Treatment and the social side.** Treat the disorder, restrict means with the family in the room, and offer a therapy with adolescent trial evidence rather than general counselling. Socially: shame that stops the family returning, a school with no plan for the return, and reporting that names the method and seeds contagion.

WHAT THE LAW DOES NOT COVER

Section 115 removes punishment; it does not fund a pathway to care. No statute governs how a school responds or how a pupil returns after an attempt. Confidentiality for a mature minor is unsettled in Indian law. Media guidelines are advisory and unenforceable.

WHAT EARNS THE MARKS

- **The adolescent seen alone,** written as a step rather than assumed.
- **Section 115 stated correctly:** a presumption of severe stress, not blanket decriminalisation.
- **The reporting duty named,** because it is the conflict this question is really about.
- **Means restriction with the family present,** the one intervention with the best evidence per minute spent.

SEE ALSO Slot IV - 03 the Mental Healthcare Act critically evaluated Slot IV - 10 school mental health programmes

SPRINT Day 39, Community psychiatry and public health

IV - 07

How would you plan a mental health programme for a population of one lakh? [10]

10 | COMMUNITY AND PUBLIC MENTAL HEALTH | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Know the denominator	Who the lakh are, and what already serves them
2 Estimate the need	Survey data, not a figure from memory
4 Build into what exists	Train, supply, refer, reach out
2 Measure and sustain	Indicators, registers, and the money

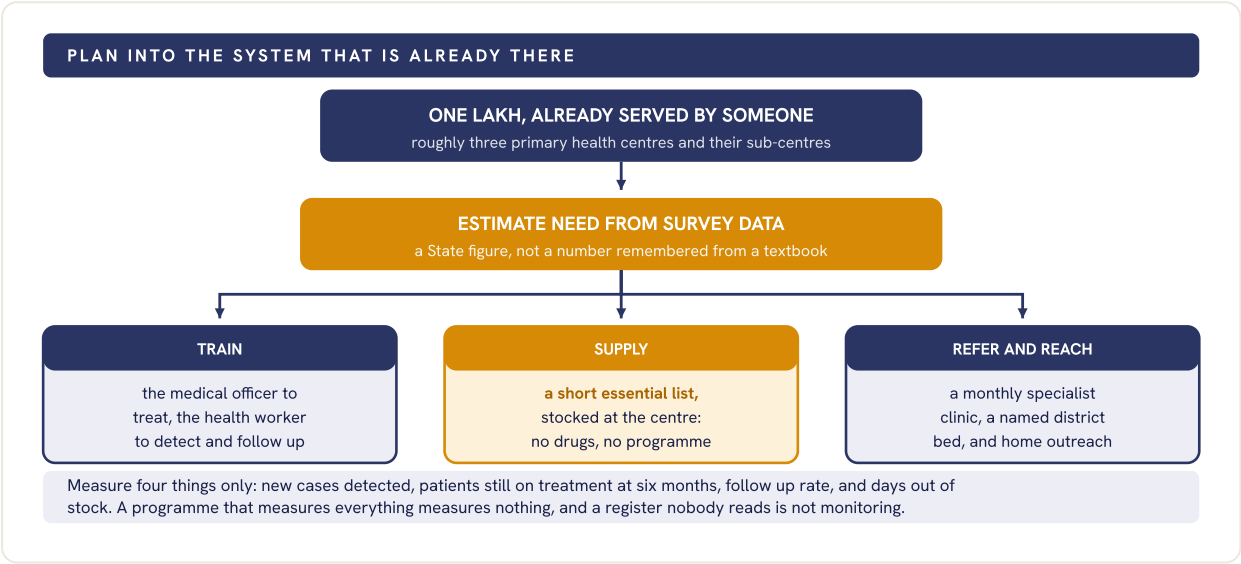


Fig 007.1 Supply sits in amber because a trained officer with an empty shelf produces referrals, not treatment, and that is the commonest way a district plan fails.

- **Know the denominator, then the need.** A lakh in the plains is about three primary health centres, twenty sub-centres, and a community health centre above them. Estimate need from the national survey rather than from memory: it found current mental morbidity in roughly one adult in ten, and a treatment gap above seventy per cent for most disorders. Those two numbers set the target and the reason for it.
- **Build in, not alongside.** Train the existing staff, stock a short essential list of an antipsychotic, an antidepressant, a mood stabiliser, an anticonvulsant, a benzodiazepine and an anticholinergic, run one specialist clinic a month, name the referral bed, and use the health worker for detection and follow up. Add school and workplace components once the clinical spine works, never before.

WHAT EARNS THE MARKS

- **The existing infrastructure counted first.** A plan that invents a new service has answered a different question.
- **Need estimated from a named source,** not asserted.
- **A drug list, by class,** which is the concrete detail examiners look for and answers rarely give.
- **Four indicators, named.** Monitoring described in general terms earns nothing.

SEE ALSO Slot IV - 01 the District Mental Health Programme Slot IV - 05 electronic media in management **SPRINT** Day 39, Community psychiatry and public health

IV - 08

Define mental health. Describe the strategies for promotion of mental health. What role can mental health professionals have in it? [2+5+3]

2 + 5 + 3 | COMMUNITY AND PUBLIC MENTAL HEALTH

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The definition	The wording, then what it is not
2	Promotion against prevention	Different target, different population
3	The strategies	By life stage, by setting, by determinant
3	The professional's role	Advise, train, deliver, evaluate, advocate

THE DEFINITION

Mental health is a state of well-being in which the individual realises his or her own abilities, can cope with the normal stresses of life, can work productively, and is able to contribute to the community. It is not the absence of illness: the two run on separate axes.

- **Promotion is not prevention.** Promotion raises positive mental health and acts on its determinants across a whole population, well or unwell. Prevention targets the occurrence of disorder, and is universal, selective or indicated by whom it reaches. The older primary, secondary and tertiary frame still earns its mark beside the newer one.
- **By life stage and setting.** Perinatal care and parenting programmes, early childhood nutrition and stimulation, life skills teaching in schools, workplace stress programmes, and social contact for older adults. The settings that carry them are the school, the workplace, primary care and the community, because that is where the population already is.
- **By determinant and instrument.** Poverty, education, housing, violence and harmful alcohol use. Then the law: the Mental Healthcare Act 2017 makes promotion and prevention a duty of government at Section 29, and awareness and stigma reduction a duty at Section 30. Anti-stigma work built on contact with people who have recovered outperforms leaflets.
- **The role, in five verbs.** Advise on policy and programme design. Train medical officers, health workers and teachers, so the work is shared rather than held. Deliver the clinical service that promotion cannot replace. Evaluate, so a programme is measured rather than asserted. Advocate with schools, employers, the media and the district administration.

WHAT EARNS THE MARKS

- **The definition written out,** with the line that mental health is not merely the absence of illness.
- **Promotion separated from prevention,** which is the axis this question turns on.
- **Strategies grouped by where they act,** not a loose list of good intentions.
- **An honest line where a figure would go,** since the treatment gap is large and unevenly measured.

SEE ALSO Slot IV - 01 the District Mental Health Programme Slot IV - 10 school mental health programmes SPRINT Day 39, Community psychiatry and public health

IV - 09

Discuss substance use disorder and behavioural addictions during the COVID-19 pandemic and the related movement restriction. What are the theories of gambling behaviour? [7+3]

7 + 3 | COMMUNITY AND PUBLIC MENTAL HEALTH | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **What restriction changed**	Supply, structure and treatment, all at once
3 **Substances**	Alcohol, opioids, tobacco, and the services
2 **Behavioural addictions**	Gaming, screen use, online gambling
3 **Theories of gambling**	The table, three accounts and one that joins them

THE ORGANISING SENTENCE

The pandemic did not create a new disorder. Movement restriction removed three things at once: supply, daily structure, and access to treatment. Which of the three moved first decides what was seen, and that sentence belongs before any list.

- **Alcohol.** Where sale stopped abruptly, restriction acted as an unplanned withdrawal experiment: withdrawal, seizures and delirium in the first days, with recourse to surrogate and illicit alcohol. Where sale continued or resumed, home drinking rose instead. Which pattern applied followed local sale policy, not the virus.
- **Opioids, tobacco, services.** Supply interruption and closed clinics broke opioid substitution therapy, so take-home dosing was widened to hold patients in treatment. Tobacco restriction produced both quit attempts and relapse. Outpatient and harm-reduction services closed or moved to teleconsultation, and relapse rose again as restriction lifted.
- **Behavioural addictions.** Screen use, gaming and online gambling rose with school closure and confinement, and betting platforms filled the space that closed venues left. ICD-11 already recognises gambling disorder and gaming disorder as disorders due to addictive behaviours, so the diagnosis was available where the behaviour disabled.

THEORY	THE MECHANISM IT PROPOSES	WHAT IT EXPLAINS BEST
Learning	Variable ratio reinforcement; a near miss reads as a win	Persistence through steady loss
Cognitive	Gambler's fallacy, illusion of control, recall biased to wins	Why able people keep playing
Neurobiological	Mesolimbic dopamine reward, shared with substance use	Its DSM-5 move beside substance use
Integrative	Pathways model: conditioned, emotionally vulnerable, impulsivist	Matching treatment to the route

WHAT EARNS THE MARKS

- **Supply, structure and treatment named as three losses,** before any substance.
- **The alcohol answer given both ways,** since direction followed local sale policy.
- **Gambling and gaming among the addictive behaviours,** not impulse-control.
- **The integrative model named last,** which lifts the limb above a list of labels.

SEE ALSO Slot IV - 05 electronic media in management Slot IV - 06 adolescent self-harm and suicide

IV - 10

What is a school mental health programme? What are its benefits? Discuss in brief any two Indian school mental health programmes. [2+2+3+3]

2 + 2 + 3 + 3 | COMMUNITY AND PUBLIC MENTAL HEALTH

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Delivered through the school, not relocated into it
2 The three tiers	Universal, selective, indicated
3 The benefits	Reach, timing, access, stigma, transfer
3 Two Indian programmes	The table: who runs it, and what it does

THE DEFINITION

A school mental health programme is a planned set of activities delivered in and through the school, by teachers and school staff with specialist backup, to promote well-being, build life skills, identify difficulty early, and link the pupils who need care to a service. The delivery agent is the definition: a clinic relocated into a school building is not one.

- **The three tiers.** Universal: whole-school life skills teaching, a policy on bullying, and a staff room trained to notice. Selective: groups at raised risk, such as bereavement, learning difficulty or examination pressure. Indicated: named pupils identified, referred and followed up. Naming the tiers separates a programme from a talk.
- **The benefits.** Reach, because the age band is already assembled. Timing, because most disorders begin here and the school sees the change before the family names it. Access without a family having to seek a psychiatric service. Less stigma, because it arrives as education. Skills that transfer. And attendance and academic gain, which is what keeps a school engaged.

PROGRAMME	WHO RUNS IT	WHAT IT DOES	THE LIMIT
Rashtriya Kishor Swasthya Karyakram, 2014	Health and Family Welfare	Adolescent health with mental health as a strategic area; peer educators, and adolescent friendly health clinics	Health-system led, so school reach needs local linkage
Manodarpan, 2020	Education	Psychosocial support for students, teachers and families: a helpline, a counsellor directory, advisory handbooks	Helpline led, so thin at the universal tier
School component of the district programme	State health, district team	Life skills education and counselling in schools, with the district team as clinical backup	Needs a district team that is rarely complete

WHAT EARNS THE MARKS

- **The delivery agent named in the definition,** which is the distinction being tested.
- **The three tiers,** since the benefits differ by tier and a flat list cannot show it.
- **Two named programmes with the ministry behind each,** not two descriptions of life skills teaching.
- **A stated limit per programme,** which is what discuss asks for beyond naming.

SEE ALSO Slot IV - 06 adolescent self-harm and suicide Slot IV - 01 the District Mental Health Programme SPRINT Day 39, Community psychiatry and public health

IV - 11

What are the common myths and misconceptions about LGBTQIA+ identities such as intersex and Hijra? What are the basic tenets of LGBTQIA+ affirmative counselling? [4+6]

4 + 6 | COMMUNITY AND PUBLIC MENTAL HEALTH | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Three axes, kept apart**	Orientation, identity, sex characteristics
2 **The myths, corrected**	The table, each against what is settled
3 **The stance**	Not a disorder, minority stress, who learns
3 **The work, and the refusal**	Disclosure, risk, access, conversion

THREE AXES, AND MOST MISCONCEPTIONS ARE ONE READ AS ANOTHER

Sexual orientation is who a person is drawn to. Gender identity is the gender a person knows themselves to be. Sex characteristics are anatomy, chromosomes and hormones, and intersex is variation in those, present from birth. The three vary independently.

THE MISCONCEPTION	WHAT IS SETTLED	WHERE THAT STANDS
These identities are mental disorders	Homosexuality left the classification in 1990; ICD-11 places gender incongruence outside the mental disorders chapter	Classification
Intersex and transgender are the same	Intersex is bodily variation from birth, not a claim about identity	Definition
Hijra is the Indian word for a trans woman	A South Asian community with its own kinship, not a translation	Social, not clinical
Upbringing causes it, so it can be changed	Conversion practice has no evidence of benefit and is associated with harm	Ruled professional misconduct

- **The stance.** The identity is not the presenting problem and is never a target of treatment. Minority stress is the working model: discrimination, rejection and violence from outside, and concealment, expected rejection and internalised stigma from within, together account for the raised rates of depression, anxiety, substance use and suicidality.
- **The conduct.** The therapist does the learning, not the client: know the terms, the local community structures and the law before the session, and use the person's own name and pronouns. Disclosure belongs to the client, so never out a person to family, school or employer, and let coming out be paced by them.
- **The work, and what is refused.** Screen for family coercion and forced marriage, violence, homelessness, unsafe hormone sources and suicide risk. Treat the presenting disorder on its own terms. Support access to gender-affirming care and to legal recognition, since identity is self-perceived in law and not certified by a psychiatrist. Refuse conversion practice plainly, including when a family asks for it.

WHAT EARNS THE MARKS

- **The three axes separated before any myth is answered,** since most of the myths are a category error.
- **Hijra treated as a social identity,** not as a synonym for a clinical term.
- **Minority stress named as the model,** which explains the raised rates without pathologising the identity.
- **Conversion practice refused in the answer,** and the refusal extended to the family who asks.

SEE ALSO Slot IV - 08 promotion of mental health Slot IV - 06 adolescent self-harm and suicide

AREA 02 OF 15 · P4-A03

Dementias: types and differential

10	11.4%	IV-12	IV-21
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 5 HIGH 2 SINGLE 3

REVISION ORDER

The two mild cognitive impairment slots together, they are one answer split and both fragments declare it. The two subcortical slots likewise, and the grid writes most of both. Frontotemporal and Lewy body dementia next as a pair, with pseudodementia directly on top of them because it is the differential they share. Prion disease, the Alzheimer prevention slot and the cholinesterase inhibitor slot are independent and each stands alone.

WHAT IS IN THIS AREA

Mild cognitive impairment in two slots, one asking for the definition, the subtypes and the assessment and one for the presentation, the course and the management. Frontotemporal dementia and Lewy body dementia. The subcortical dementias in two slots, one as a grid against the cortical picture and one as the concept, the circuit anatomy and the causes. Then pseudodementia, prion disease, the risk factors and prevention of Alzheimer disease, and the cholinesterase inhibitors with their indications and contraindications.

IV - 12

Mild cognitive impairment [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

KNRUHS, KUHS, MUHS, NTRUHS, RGUHS - 8 APPEARANCES, 8 OF 78 SESSIONS

SKELETON

2 Definition	Decline beyond age, with independence still intact
2 Subtypes	Amnestic or not, one domain or several
3 Assessment	Informant, testing, and the reversible causes
3 Course and management	Three possible futures, and what is actually done

THE DEFINITION

Cognitive decline greater than expected for age and education, in one or more domains, reported by the patient or an informant and preferably shown on testing, with everyday independence essentially preserved. It is neither normal ageing nor dementia, and the same state is called mild neurocognitive disorder in current classification.

SUBTYPE	WHAT IS AFFECTED	MOST OFTEN PRECEDES	NOTE
Amnestic, single domain	Memory alone	Alzheimer disease	**Highest conversion risk**
Amnestic, multiple domain	Memory plus another domain	Alzheimer or vascular disease	Commonest in clinics
Non-amnestic, single	Language, executive or visuospatial	Frontotemporal or Lewy body disease	**Memory can be normal**
Non-amnestic, multiple	Two or more, memory spared	Lewy body or vascular disease	Screen for parkinsonism

Amber cells are the discriminators. Naming the subtype is what turns a screening result into a prediction the examiner can mark.

- **Assessment.** Informant history is essential. A cognitive screen sensitive to mild deficits is preferred to a basic one, and reversible contributors are sought: depression, thyroid disease, vitamin B12 deficiency, sleep apnoea, alcohol and anticholinergic load.
- **Testing in Indian practice.** Education and literacy shift every cut-off, so use an instrument validated in the local language rather than a translated score read against foreign norms.

WHAT EARNS THE MARKS

- **Preserved independence written into the definition.** It is the single line that separates this from dementia.
- **Subtype named, with what it tends to precede.**
- **Reversible contributors listed,** because they are the part of the assessment that changes the outcome.

SEE ALSO Slot IV - 15 course and management of mild cognitive impairment Slot IV - 16 prevention of Alzheimer disease

SPRINT Day 32, Dementias & neurocognitive disorders

IV - 13

Etiology, Pathology, clinical features and management of Fronto-temporal Dementias. [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

DNB, KNRUHS, KUHS, RGUHS, TN-MGRMU - 8 APPEARANCES, 8 OF 78 SESSIONS

SKELETON

2 **Aetiology**	About a third familial; three genes carry most of it
2 **Pathology**	Lobar degeneration, sorted by the protein inside
3 **Clinical features**	Behaviour and language first, memory relatively spared
3 **Management**	Nothing alters the course; name what genuinely helps

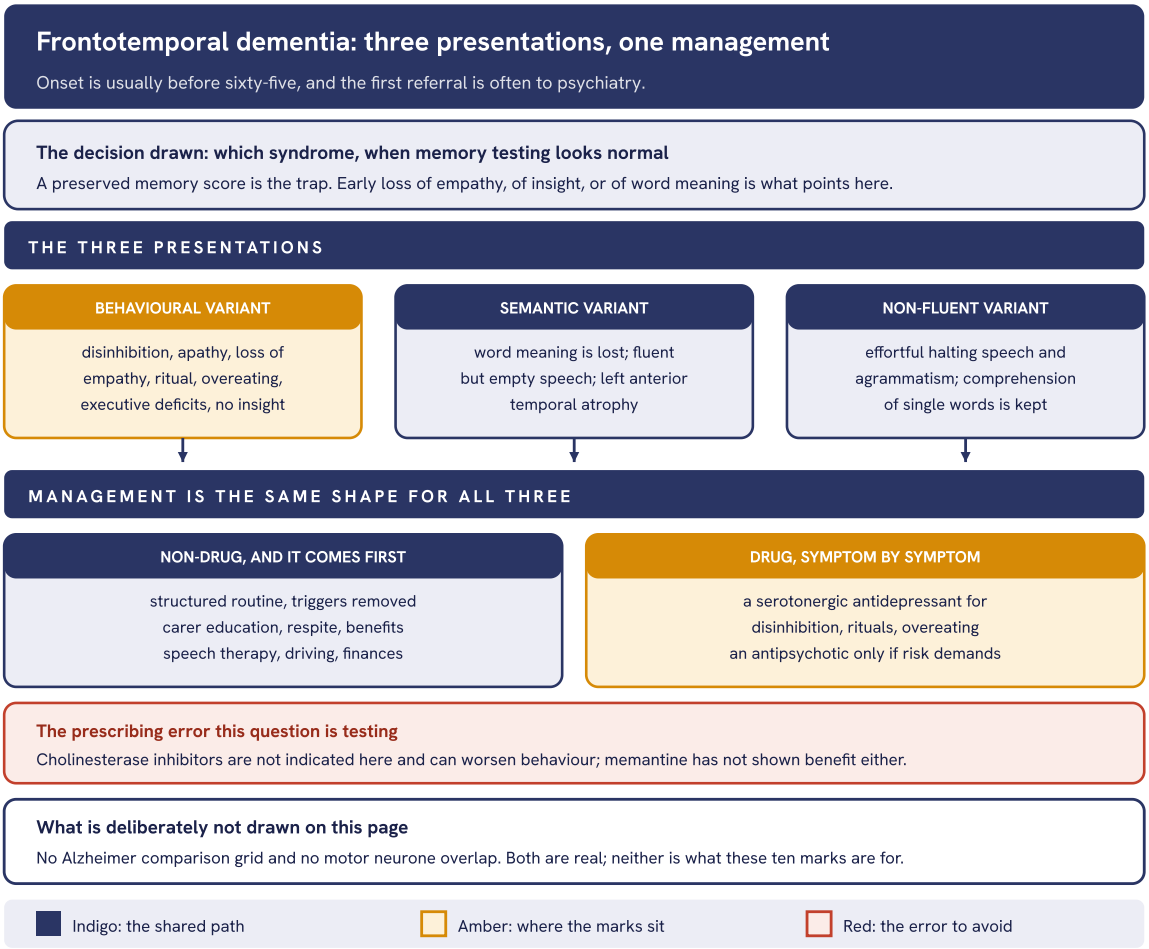


Fig 013.1 The three clinical presentations above, the single management shape below, and the prescribing error carried as a red strip because it is the line that separates this dementia from Alzheimer disease in practice.

- **Aetiology and pathology.** Around a third have a family history, most often through the MAPT, GRN or C9orf72 genes. The pathology is frontotemporal lobar degeneration, grouped by the protein in the inclusions: tau, TDP-43 or FUS.
- **Why it reaches psychiatry first.** Disinhibition, apathy and ritual look like mania, depression or obsessive compulsive disorder, and standard memory testing can be normal for years.

PITFALL

Waiting for a memory complaint. Episodic memory and orientation can test normally for years here, and insight is lost early, so the history that decides this diagnosis comes from the family rather than from the patient or the score.

WHAT EARNS THE MARKS

- **The three variants named separately,** not folded into one description of frontal behaviour.
- **Pathology given by inclusion protein,** which is what the word pathology in the stem is asking for.
- **Cholinesterase inhibitors explicitly excluded.** Most answers transplant Alzheimer treatment onto this page.
- **Carer support written as management,** because for this dementia it is most of it.

SEE ALSO Slot IV - 17 cholinesterase inhibitors Slot IV - 19 cortical and subcortical dementias **SPRINT** Day 32, Dementias & neurocognitive disorders

IV - 14

Clinical features of Lewy body dementia and its management. [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

DNB, KNRUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

3	The core features	Four of them, and the biomarkers that support
2	Placing it	The one-year rule against Parkinson disease dementia
3	Drug management	Cholinesterase inhibitor first, and what is forbidden
2	Everything else	Sleep, falls, blood pressure, and the carer

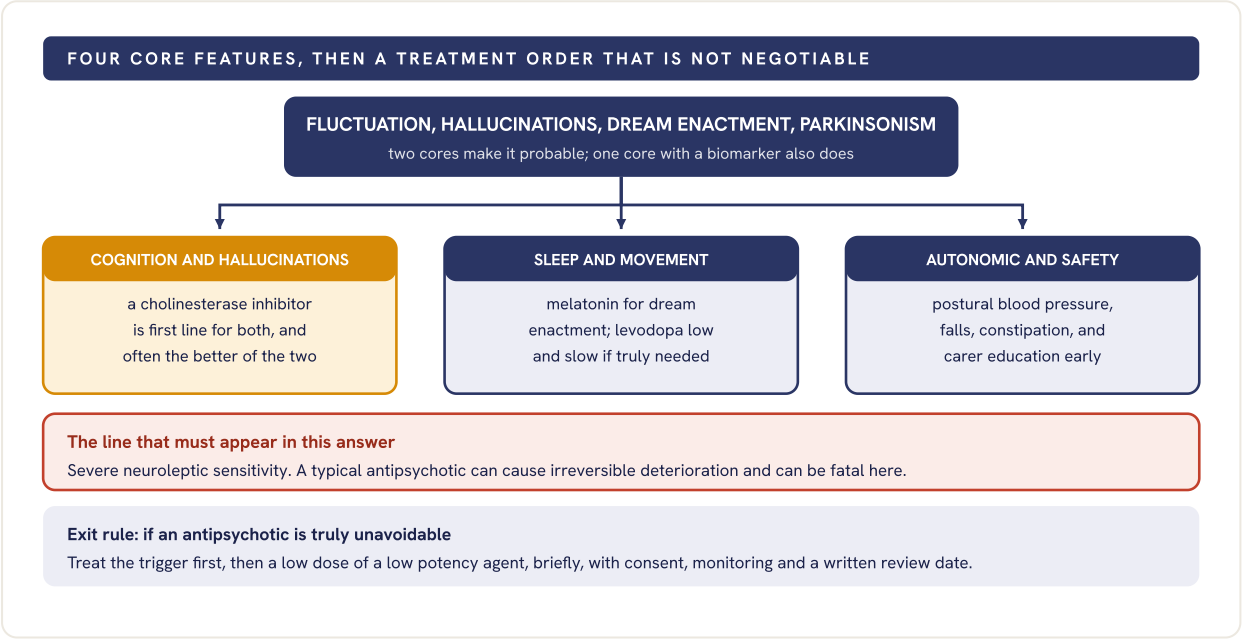


Fig 014.1 The four core features feeding three treatment strands, with neuroleptic sensitivity carried as a red strip because prescribing an antipsychotic here is the error that kills.

- **Supporting the diagnosis.** Reduced dopamine transporter uptake in the basal ganglia, abnormal cardiac MIBG scintigraphy, and sleep study confirmation of REM sleep without atonia.
- **The one-year rule.** Dementia before, or within a year of, parkinsonism is Lewy body dementia. Parkinsonism for longer than a year first is Parkinson disease dementia; the pathology is shared.

PITFALL

Visual hallucinations here are usually well formed and often non-threatening. Reaching for an antipsychotic rather than a cholinesterase inhibitor is the classic error.

WHAT EARNS THE MARKS

- **All four core features,** with dream enactment included; it is the one most often left out.
- **The one-year rule stated,** which shows the boundary is understood rather than memorised.
- **Neuroleptic sensitivity named as a danger,** not as a side effect.

SEE ALSO Slot IV - 17 cholinesterase inhibitors Slot IV - 19 cortical and subcortical dementias **SPRINT** Day 32, Dementias & neurocognitive disorders

IV - 15

Definition, presentation, course and management of Mild Cognitive Impairment. [2+2+3+3]

2 + 2 + 3 + 3 | DEMENTIAS: TYPES AND DIFFERENTIAL

DNB, RGUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	Definition	Decline present, everyday independence intact
2	Presentation	How it actually walks into the clinic
3	Course	Three futures, and what predicts which one
3	Management	Subtract, reduce, review; no drug is licensed

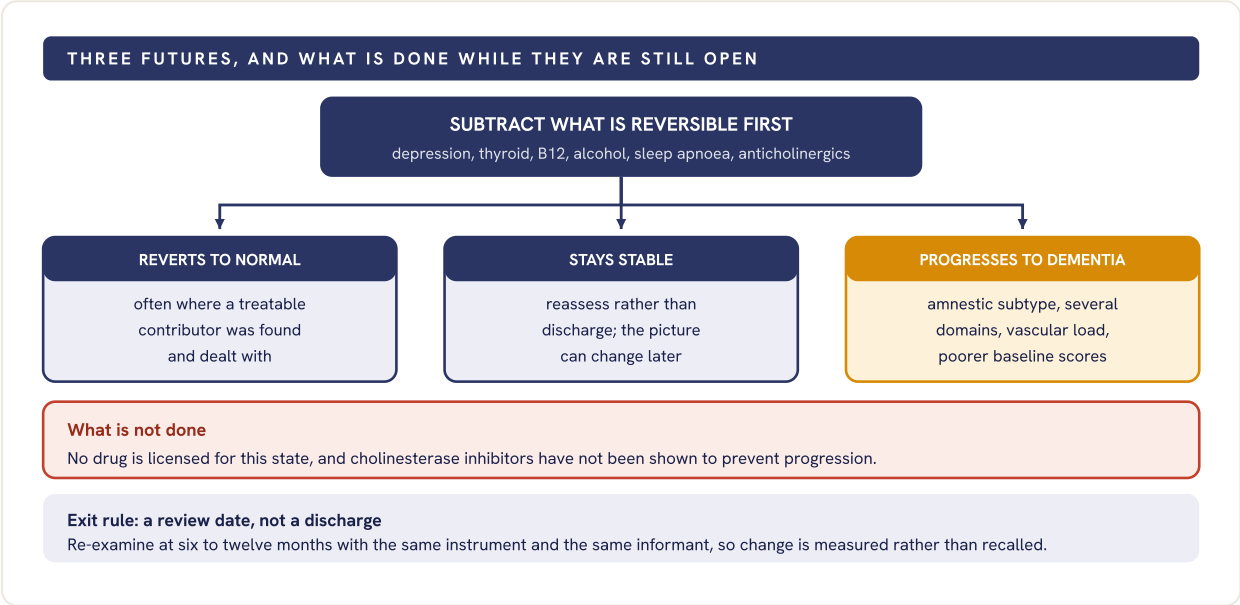


Fig 015.1 Reversible contributors are subtracted before the prognosis is discussed at all, and the three futures are then set against the features that predict each one.

- **Presentation.** A complaint of forgetting names, appointments or where things were put, corroborated by family, with daily tasks still managed, sometimes with lists.
- **Management, in three moves.** Treat what is reversible, reduce vascular risk, raise physical and social activity, then review. Hearing and blood pressure carry the best evidence.

PITFALL

Telling a family that this is early dementia. It is a state with three possible futures, and saying so accurately is both honest and clinically useful.

WHAT EARNS THE MARKS

- **Independence preserved, in the definition line.**
- **Three courses named,** with reversion included; most answers give only progression.
- **No licensed drug stated plainly,** and risk reduction given in its place.

SEE ALSO Slot IV - 12 mild cognitive impairment, subtypes and assessment Slot IV - 16 prevention of Alzheimer disease

SPRINT Day 32, Dementias & neurocognitive disorders

IV - 16

Discuss the risk factors and prevention of Alzheimer's disease. [10]

10 | DEMENTIAS: TYPES AND DIFFERENTIAL | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The framing**	Risk accumulates across a life, so prevention has stages
3 **Not modifiable**	Age and genes, and what a genotype does not tell you
3 **Modifiable**	The named factors, placed at the stage they act
2 **Prevention in practice**	What has trial support, and what is only plausible

THE FRAMING THAT EARNS THE FIRST MARKS

Risk is not one list. It accumulates across early life, midlife and later life, and a prevention answer that mirrors those stages reads as understanding rather than recall. The Lancet Commission (2020) attributed roughly 40 per cent of dementia risk to 12 modifiable factors; the 2024 update raises this to about 45 per cent across 14.

STAGE	FACTORS	WHAT IS ACTUALLY DONE	NOTE
Early life	Less education	Schooling, literacy, cognitive reserve	Acts decades ahead
Midlife	Hearing loss, hypertension, obesity, head injury, excess alcohol	Hearing aids, blood pressure control, helmets, alcohol limits	**Largest single lever**
Later life	Smoking, depression, social isolation, inactivity, diabetes, air pollution	Cessation, treatment, activity, contact, glycaemic control	**Also good for the heart**
Not modifiable	Age, family history, APOE e4, trisomy 21, rare dominant mutations	Counselling, not screening	**Genotype is risk, not fate**

Amber cells are the discriminators. The stage of life a factor acts in is what turns a list into a prevention plan.

- **What has trial support.** Midlife blood pressure control has reduced later cognitive impairment in a randomised trial, and a multidomain programme of diet, exercise, cognitive training and vascular monitoring improved cognitive outcome in an older population at risk.
- **Treatment is not prevention.** The anti-amyloid antibodies slow decline modestly in early disease, carry imaging abnormality risk, and are not part of prevention or of routine Indian practice.

WHAT EARNS THE MARKS

- **Factors sorted by life stage,** which is the structure the marks are written around.
- **Genotype described as risk, not diagnosis,** with predictive testing not recommended.
- **Two named interventions with trial evidence,** separated from what is merely plausible.

SEE ALSO Slot IV - 12 mild cognitive impairment Slot IV - 15 course and management of mild cognitive impairment

SPRINT Day 32, Dementias & neurocognitive disorders

IV - 17

Enumerate cholinesterase inhibitors and their indications and contraindications. [10]

10 | DEMENTIAS: TYPES AND DIFFERENTIAL | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The agents	Three in use, and how each one differs
3 Indications	Where they help, and the dementias where they do not
3 Contraindications	Heart, chest, gut, bladder, and the anaesthetic
2 Practical prescribing	Titrate, review, and know when to stop

AGENT	MECHANISM AND ROUTE	WHERE IT IS USED	ITS OWN NOTE
Donepezil	Reversible, selective for acetyl-cholinesterase; once daily, 5 mg then 10 mg	Alzheimer disease at every severity; Lewy body dementia	Long acting; vivid dreams if taken at night
Rivastigmine	Inhibits acetyl and butyryl cholinesterase; oral twice daily or a patch	Alzheimer disease; Parkinson disease dementia; Lewy body dementia	**Not metabolised through cytochrome P450, so fewer interactions**
Galantamine	Reversible inhibitor plus nicotinic receptor modulation; 8 to 24 mg daily	Alzheimer disease, mild to moderate	Avoid in severe hepatic or renal impairment
Where they do not belong	Not a class effect across all dementias	Frontotemporal dementia, mild cognitive impairment	**Behaviour can worsen in frontotemporal dementia**

Amber cells are the discriminators. The last row is the one that separates a drug list from a prescribing answer.

- **Contraindications and cautions.** Bradycardia, sick sinus syndrome and other conduction disease; syncope; active peptic ulcer or gastrointestinal bleeding; asthma and chronic obstructive disease; bladder outflow obstruction; a seizure history; and prolonged blockade with suxamethonium at anaesthesia.
- **Practical prescribing.** Start low, titrate at intervals of weeks with food, and set a review at about three months. Cholinergic effects are nausea, diarrhoea, appetite and weight loss, cramps and slow pulse.

PITFALL

Prescribing an anticholinergic for the urinary or gastrointestinal effects of the cholinergic. The two cancel each other, the cognition worsens, and the prescription that should have been reviewed is instead defended.

WHAT EARNS THE MARKS

- **Three agents named with a real difference each,** not three names in a row.
- **Indications and non-indications both given.** Frontotemporal dementia is the discriminating entry.
- **Cardiac and respiratory cautions first,** because they are the ones that cause harm.

SEE ALSO Slot IV - 14 Lewy body dementia Slot IV - 13 frontotemporal dementia Slot IV - 100 memantine SPRINT Day 32, Dementias & neurocognitive disorders

IV - 18

Discuss the clinical features and management of Pseudodementia. [10]

10 | DEMENTIAS: TYPES AND DIFFERENTIAL | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **What the term means**	Cognitive impairment belonging to a psychiatric illness
3 **Distinguishing features**	History, answers, effort, and which came first
3 **Management**	Treat the depression properly, then test again
2 **The follow-up rule**	A good number later declare a true dementia

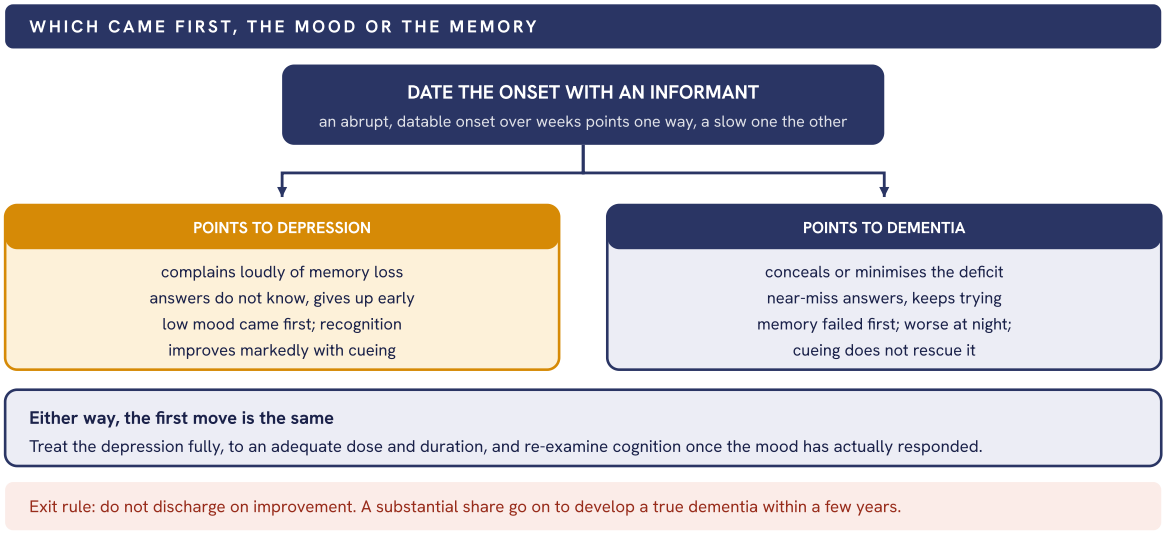


Fig 018.1 The two columns of the classical contrast, converging on one first move, with the follow-up rule carried along the foot because that is the mark most answers never reach.

- **What the term means.** Cognitive impairment produced by a psychiatric illness, most often depression in later life, which improves when that illness is treated. It is a description, not a diagnosis in current classification.
- **Treatment specifics.** An antidepressant at full dose for a full trial, with electroconvulsive therapy where the depression is severe, psychotic, food-refusing or carries high suicide risk. Do not start a cholinesterase inhibitor for the cognitive picture.

PITFALL

Treating the label rather than the patient. The features overlap, both conditions can coexist, and the safest position is to treat the depression well and to keep watching.

WHAT EARNS THE MARKS

- **Onset and sequence stated first.** Which came first, mood or memory, carries more weight than any single test.
- **The answer pattern named.** Do not know against near-miss is the classic discriminator.
- **Follow-up written into management,** not offered as an afterthought.

SEE ALSO Slot IV - 12 mild cognitive impairment Slot IV - 19 cortical and subcortical dementias **SPRINT** Day 32, Dementias & neurocognitive disorders

IV - 19

Enumerate subcortical Dementias. Describe the clinical features differentiating them from Cortical Dementias. [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

DNB, RGUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2	The two lists	Named examples on each side, not a definition each
4	The grid	Domain by domain, with the discriminators marked
2	The three that settle it	Movement, cortical signs, and benefit from cueing
2	The honest limit	A clinical heuristic; most real cases are mixed

DOMAIN	CORTICAL	SUBCORTICAL	WHAT SETTLES IT
Named examples	Alzheimer disease, frontotemporal dementia	Huntington disease, progressive supranuclear palsy, Parkinson disease dementia, HIV dementia, Wilson disease	Enumerate before comparing
Memory	Storage fails; recognition is poor	Retrieval fails; recognition is better	**Cueing rescues the subcortical patient**
Language	Aphasia, and later anomia	Language intact; speech dysarthric and quiet	**Aphasia against dysarthria**
Speed of thought	Normal until late	Slowed throughout, called bradyphrenia	Timed tasks expose it
Higher cortical signs	Apraxia, agnosia, acalculia present	Absent, or explained by slowness	**Their presence is cortical**
Movement and gait	Normal until late in the illness	Abnormal early: rigidity, chorea, tremor, gait	**The strongest single pointer**
Mood and personality	Change later, less prominent early	Apathy and depression early and prominent	Referred to psychiatry first

Amber cells are the discriminators. Everything else in that column supports; saying which is which is itself worth marks.

- **The honest limit.** This is a clinical heuristic about where the pathology sits, not a pathological classification. Vascular disease and Lewy body disease straddle both columns, and mixed pictures are the rule in older patients.

PITFALL

Listing ten differences without ranking them. The examiner is looking for the three that would actually change a bedside opinion: a movement disorder, cortical signs, and whether cueing helps recall.

WHAT EARNS THE MARKS

- **Enumeration answered as enumeration.** The stem asks for named conditions before it asks for features.
- **A grid, not two paragraphs,** so each domain is readable across a row.
- **The discriminators identified as discriminators,** and the limit of the division stated once.

SEE ALSO Slot IV - 20 subcortical dementia Slot IV - 14 Lewy body dementia **SPRINT**

Day 32, Dementias & neurocognitive disorders

IV - 20

Subcortical dementia. [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 The concept	Where the term comes from and what it names
2 The anatomy	Frontal-subcortical circuits, not the cortex itself
3 The syndrome	Slowing, retrieval failure, apathy, and movement
3 Causes and reversibility	Grouped, with the treatable ones written first

THE CONCEPT

A dementia syndrome in which the burden of pathology sits in the basal ganglia, thalamus, brainstem and deep white matter rather than in the cortical mantle. The clinical signature is slowing of thought, retrieval-type forgetfulness, apathy and an early movement disorder, with language and praxis relatively preserved.

GROUP	EXAMPLES	HOW REVERSIBLE	FIRST STEP
Degenerative	Huntington disease, progressive supranuclear palsy, Parkinson disease dementia	Not reversible	Symptomatic and supportive
Vascular	Small vessel disease, lacunar and thalamic infarcts	Progression modifiable	**Control the vascular risk**
Infective	HIV-associated dementia, neurosyphilis	Substantially treatable	**Serology, then treat**
Metabolic and toxic	Wilson disease, chronic alcohol use, heavy metals	Treatable if caught	**Copper studies in the young**
Structural	Normal pressure hydrocephalus	Potentially reversible	Imaging, then referral

Amber cells are the discriminators. Sorting causes by what can be reversed is what makes this a clinical answer rather than a list.

- **The anatomy that explains the picture.** The frontal-subcortical circuits run from prefrontal cortex through striatum, pallidum and thalamus and back. Interrupting the loop below the cortex produces executive and motivational failure without aphasia or apraxia.
- **Management principle.** Treat the underlying disease where it is treatable, then treat depression and apathy on their own terms. Cholinesterase inhibitors are not routine here, the exception being Parkinson disease dementia.

WHAT EARNS THE MARKS

- **The circuit explanation given,** which turns a list of signs into a mechanism.
- **Causes grouped by mechanism,** with the reversible ones identified.
- **Retrieval failure distinguished from storage failure,** because that is the memory finding at the bedside.

SEE ALSO Slot IV - 19 cortical and subcortical dementias compared Slot IV - 14 Lewy body dementia SPRINT Day 32, Dementias & neurocognitive disorders

IV - 21

Prion disease. [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What a prion is	A misfolded protein that templates its own shape
3	The three routes	Sporadic, inherited, acquired, with named examples
3	Clinical picture	Rapid decline, myoclonus, and the psychiatric onset form
2	Diagnosis and care	The tests that support, and honest palliation

THE MECHANISM

The normal cellular prion protein, coded on chromosome 20, misfolds into a protease-resistant form that templates the same change in neighbouring molecules. The result is spongiform vacuolation, neuronal loss and astrocytic gliosis with no inflammatory response, which is why these illnesses are transmissible without being infections in the ordinary sense.

ROUTE	NAMED ENTITIES	DISTINGUISHING FEATURE	NOTE
Sporadic	Sporadic Creutzfeldt-Jakob disease	Around sixty, rapid dementia with myoclonus	**The great majority of cases**
Inherited	Familial Creutzfeldt-Jakob disease, Gerstmann-Straussler-Scheinker syndrome, fatal familial insomnia	Family history; insomnia and dysautonomia in the last	Genetic counselling needed
Acquired, medical	Iatrogenic disease from grafts, growth hormone, instruments	Long latency after the exposure	Instrument decontamination
Acquired, dietary	Variant Creutzfeldt-Jakob disease, kuru	**Young, psychiatric onset, painful sensory symptoms**	Comes to psychiatry first

Amber cells are the discriminators. The last row is the one a psychiatry examiner is testing for.

- **What supports the diagnosis.** Periodic sharp wave complexes on the record in the sporadic form, cortical ribboning and basal ganglia change on diffusion-weighted imaging, and cerebrospinal fluid assays including real-time quaking-induced conversion. Certainty comes only from tissue.
- **The red flag to state.** Rapidly progressive dementia over months rather than years, with myoclonus and ataxia. There is no curative treatment, so care is symptomatic, palliative and directed at the family.

WHAT EARNS THE MARKS

- **Templated misfolding described,** not merely the words slow virus or infectious protein.
- **All three routes with a named entity in each.**
- **The variant form separated from the sporadic form** by age and by psychiatric onset.

SEE ALSO Slot IV - 19 cortical and subcortical dementias Slot IV - 18 pseudodementia SPRINT Day 32, Dementias & neurocognitive disorders

AREA 03 OF 15 · P4-A15

Consultation-liaison and systemic medicine psychiatry

9	10.1%	IV-22	IV-30
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 · HIGH 2 · SINGLE 5

REVISION ORDER

The consultation-liaison slot first, it frames the other eight. The two transplant slots together, they share one timeline and are declared as a deliberate split in both fragments. The cardiac, gastrointestinal and respiratory slots next as a block, because the liaison shape is the same on all three. Fibromyalgia, the infertility and cancer slot and the pre-treatment evaluation slot last, and the last of those is three questions on one page.

WHAT IS IN THIS AREA

Consultation-liaison psychiatry itself, then the organ systems the boards actually set: transplantation in two slots, one across any solid organ and one specific to the kidney, depression with ischaemic heart disease, irritable bowel syndrome, bronchial asthma, and fibromyalgia. The last two are the mental health issues of infertility and of cancer, and a pre-treatment evaluation slot that puts hyperthyroidism, a generalised seizure disorder and intellectual impairment on one page.

IV - 22

Write a note on consultation liaison psychiatry. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

KNRUHS, KUHS, WBUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	What it is	Consultation, liaison, and the difference between them
3	The models of service	The table, and what each one costs
3	Why patients are referred	The commonest reasons, in their real order
2	How it is actually done	Team first, question clarified, note usable

THE DEFINITION, AND THE DISTINCTION INSIDE IT

Consultation liaison psychiatry is the subspecialty that provides psychiatric assessment and treatment to patients in medical and surgical settings. Consultation is the episode of advice about one patient. Liaison is the standing attachment to a unit, with teaching, joint rounds and shared care. The name keeps both because the referral is only half the work.

MODEL	HOW THE SERVICE IS ORGANISED	WHAT IT BUYS AND WHAT IT COSTS
Consultation	Psychiatrist responds to written referrals from any ward	Cheap and wide; misses everyone not referred
Liaison	Psychiatrist attached to a named unit, attends its rounds	Finds cases early; covers few wards
Hybrid	Referral service plus attachment to high-yield units	The usual working compromise
Collaborative care	Case manager in primary care, psychiatrist supervising	Best evidence in chronic illness; needs staff

- **Why patients are referred.** Delirium, which is the commonest and is often referred as depression or as an uncooperative patient. Assessment after self-harm. Depression and adjustment in physical illness. Capacity and consent questions. Substance withdrawal on the ward. Agitation. Psychiatric effects of medical drugs. Medically unexplained symptoms. Pre-transplant and pre-surgical evaluation.
- **How the consultation is done.** Read the notes and speak to the team before seeing the patient, because the written question is often not the real one. Examine, including cognition. Review every drug, including those stopped. Then write a note the team can act on: a formulation in their language, one clear recommendation, and a named person for follow-up.

WHAT EARNS THE MARKS

- **Consultation and liaison defined apart,** which is the whole point of the compound name.
- **Models given as a table with their trade-offs,** not as four synonyms.
- **Delirium named first among referrals,** since it is the commonest and the commonest missed.
- **The note written for the referring team,** which is what makes the advice actually happen.

SEE ALSO Slot IV - 24 the psychiatrist in organ transplant Slot IV - 27 depression with ischaemic heart disease

SPRINT Day 40, Medical and systemic-disease psychiatry

IV - 23

Write a note on fibromyalgia. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

KNRUHS, KUHS, RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 The definition	Widespread pain, with the mechanism named
2 How it is diagnosed	From tender points to a symptom score
3 What travels with it	Sleep, mood, adversity, without reducing it to them
3 Management	The table: what works, what helps, what does not

THE DEFINITION

Fibromyalgia is chronic widespread musculoskeletal pain with tenderness, accompanied by fatigue, unrefreshing sleep and cognitive difficulty, without an inflammatory or structural cause sufficient to explain it. The mechanism is central: altered central pain processing, or nociplastic pain, rather than peripheral tissue damage. Saying that sentence is what stops the answer becoming a debate about whether the condition is real.

- **How it is diagnosed.** The 1990 criteria required widespread pain for at least three months and tenderness at eleven of eighteen defined points. The 2010 and 2016 revisions dropped the tender point count for a widespread pain index and a symptom severity scale, so the diagnosis became clinical and symptom-based, and may now be made alongside another disorder rather than only after excluding one.
- **What travels with it, and what that does not mean.** Sleep disturbance is close to universal. Depression, anxiety, post-traumatic disorder and childhood adversity are over-represented. None of that makes this a psychiatric disorder, and treating it as one is the commonest error. Give the diagnosis positively and early, because a label withheld for years drives further investigation.

TIER	WHAT IT IS	WHERE IT STANDS
First line, non-drug	Graded aerobic exercise, education, cognitive behavioural therapy, sleep treated directly	**The strongest evidence in this condition**
Drugs	Low-dose amitriptyline at night; duloxetine or milnacipran; pregabalin or gabapentin	Useful, modest, and often not tolerated
Not helpful	Opioids; corticosteroids and anti-inflammatories used alone	**Opioids worsen the central mechanism**

WHAT EARNS THE MARKS

- **Central sensitisation named as the mechanism,** not an apology for the absence of pathology.
- **Both criteria sets, and what changed between them.**
- **Exercise and therapy placed above the drugs,** which is the order the evidence gives.
- **Opioids named as harmful,** not merely as unhelpful.

SEE ALSO Slot IV - 30 irritable bowel syndrome Slot IV - 22 consultation liaison psychiatry **SPRINT**

Day 40, Medical and systemic-disease psychiatry

IV - 24

Discuss the role of the psychiatrist in organ transplant. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

KNRUHS, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Four points on one timeline	Not one assessment, a continuing role
3	Evaluating the recipient	Capacity, adherence, substance, support
2	Evaluating the living donor	Voluntariness, and what the law requires
3	After the operation	Delirium, the drugs, and the long adherence

STAGE	WHAT THE PSYCHIATRIST DOES	WHAT THE FINDING DECIDES
Pre-transplant, recipient	Capacity, psychiatric history and its treatment, substance use, cognition, social support, adherence record	**Listing, deferral, or conditions attached**
Pre-transplant, living donor	Voluntariness in a private interview, capacity, absence of coercion or payment, understanding of risk	**Whether the donation may proceed**
Peri-operative	Delirium, steroid-induced mood or psychosis, calcineurin inhibitor neurotoxicity	Treatment on the unit, not a new diagnosis
Long term	Adherence, depression, quality of life, the donor's own outcome	Graft survival and who is followed

- **The recipient assessment.** Adherence history is the single best predictor and should be sought from records, not from the interview alone. Structured instruments exist for this judgement, SIPAT and PACT among them, and using one makes the reasoning auditable. Active dependence, untreated psychosis and severe cognitive impairment are usually grounds for deferral with a treatment plan, not for permanent exclusion, and writing it that way is what distinguishes an assessment from a gatekeeping exercise.
- **The donor, and the law.** The living donor is interviewed alone. Donation in India is governed by the Transplantation of Human Organs and Tissues Act 1994 as amended in 2011, under which an authorisation committee scrutinises donation by anyone who is not a near relative, to exclude commercial dealing. The psychiatrist tests voluntariness; the committee tests the relationship.
- **Prescribing across the graft.** Immunosuppressants are metabolised through CYP3A4, so fluvoxamine, fluoxetine and nefazodone raise calcineurin inhibitor levels. Steroids raise mood, then lower it on withdrawal. Lithium needs particular care where renal function carries the graft.

WHAT EARNS THE MARKS

- **A timeline, not a single pre-operative assessment.**
- **Adherence named as the best predictor,** and sought from the record.
- **The donor interviewed alone, and the Act named,** which is the Indian limb of this question.
- **Deferral written as deferral,** rather than an exclusion list.

SEE ALSO Slot IV - 28 the psychiatrist in kidney transplant Slot IV - 22 consultation liaison psychiatry SPRINT Day 40, Medical and systemic-disease psychiatry

IV - 25

Describe the pre-treatment evaluation of a psychiatry patient suffering from hyperthyroidism, generalised seizure disorder, and intellectual impairment. [3+3+4]

3 + 3 + 4 | CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Hyperthyroidism**	Confirm the thyroid state before naming a disorder
3	**Generalised seizure disorder**	The drug already there, and the threshold
4	**Intellectual impairment**	Baseline, communication, pain, and consent

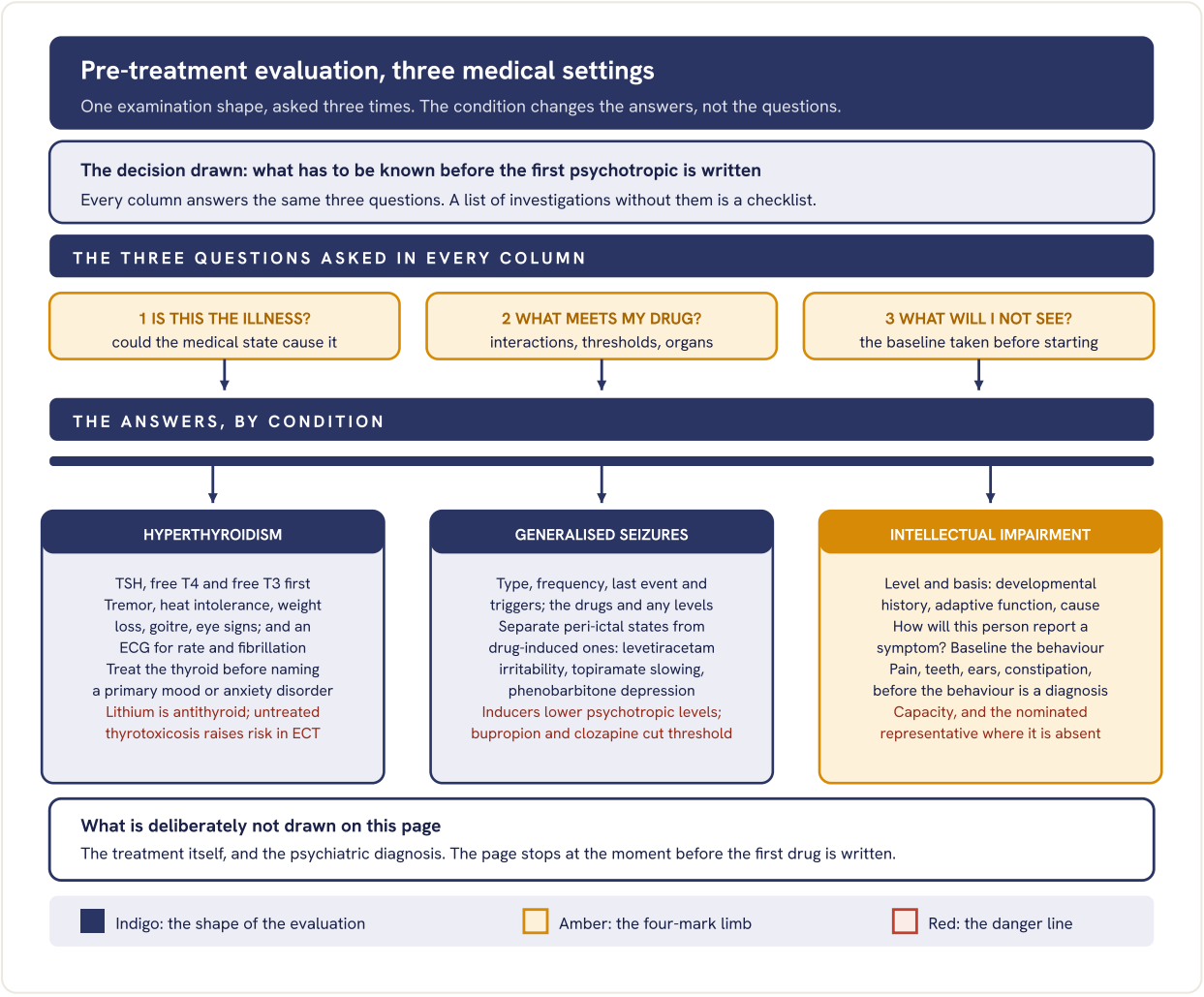


Fig 025.1 The same three questions run along one spine into all three columns, so the answer is one method applied three times, not three checklists.

- **The seizure limb, completed.** Carbamazepine, phenytoin and phenobarbitone induce enzymes and lower many psychotropic levels; valproate inhibits and raises lamotrigine. Then by seizure threshold: bupropion and clozapine lower it most, chlorpromazine more than haloperidol, and the serotonin reuptake inhibitors are safer. Electroconvulsive therapy is not contraindicated in epilepsy.

- **The impairment limb, completed.** Behaviour that challenges is communication or pain until proved otherwise, so the physical search comes first. Record a side-effect baseline the person cannot report, then start low, review on a date, and stop what has not worked.

WHAT EARNS THE MARKS

- **One method stated before the columns,** not three unconnected lists.
- **The thyroid treated as a possible cause,** which is the first limb's whole point.
- **Enzyme induction and seizure threshold both named,** as different problems.
- **A behaviour baseline and a pain search** in the four-mark limb.

SEE ALSO Slot IV - 22 consultation liaison psychiatry **SPRINT** Day 33, Endocrine & metabolic psychiatry Day 30, Epilepsy & psychiatry

IV - 26

What is mental health? Discuss the mental health issues related to infertility and to cancer. [2+4+4]

2 + 4 + 4 | CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Mental health, defined**	Well-being, and not merely the absence of illness
4	**Infertility**	Grief, the couple, and where the blame lands
4	**Cancer**	The sequence, the scoring problem, the interactions

THE DEFINITION

Mental health is a state of well-being in which the individual realises his or her own abilities, can cope with the normal stresses of life, can work productively, and is able to contribute to the community. It is not the absence of illness, which matters here because both these populations are ill and most of them are not mentally ill.

- **Infertility.** The evidence that distress causes infertility is weak; the evidence that infertility causes distress is strong, and saying so in that order protects the patient from being blamed for it. What is seen: grief that has no ritual and recurs every month; anxiety and depression that peak at a failed cycle; sex that becomes scheduled and instrumental, with sexual dysfunction following; and a social burden that in Indian practice falls disproportionately on the woman, with in-law pressure, threat of separation and sometimes violence.
- **Infertility, what the psychiatrist does.** Name the grief. Treat depression and anxiety on their own terms. Work with the couple, not only the woman who was sent. Counsel the decision to stop treatment, which nobody else offers. And know that clomifene and the gonadotropin releasing hormone agonists carry mood effects of their own.
- **Cancer.** The sequence runs from distress at diagnosis, through adjustment disorder, depression and anxiety, to delirium, which is the commonest psychiatric diagnosis in advanced disease, and on to demoralisation and desire for hastened death, with fear of recurrence and body image difficulty in survivors. Somatic items score falsely, so weight hopelessness, worthlessness, guilt and anhedonia. Exclude pain, hypercalcaemia, brain secondaries, steroids and opioid toxicity before diagnosing depression. Avoid strong CYP2D6 inhibitors such as fluoxetine and paroxetine alongside tamoxifen; mirtazapine buys appetite and sleep as well as mood.

WHAT EARNS THE MARKS

- **The direction of causation stated for infertility,** which is the examinable claim in that limb.
- **The social and gendered burden named,** since that is where Indian practice differs.
- **Delirium named in advanced cancer,** not depression alone.
- **The somatic-item problem, and one named drug interaction,** which lift this above a sad list.

SEE ALSO Slot IV - 08 promotion of mental health Slot IV - 22 consultation liaison psychiatry **SPRINT** Day 40, Medical and systemic-disease psychiatry

IV - 27

Write a note on depression with ischaemic heart disease. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	The association	Common, and independently prognostic
3	Why they travel together	Behaviour and biology, in both directions
2	Making the diagnosis	The overlap, and which items to weight
3	Treatment	The table, rehabilitation, and the honest limit

THE CLAIM WORTH WRITING FIRST

Depression is common after myocardial infarction and in stable coronary disease, and it predicts a worse cardiac outcome independently of the severity of the heart disease. That independence is why this is a question at all: a mere reaction to illness would carry no separate prognosis.

- **Why they travel together.** Behaviourally: smoking, inactivity, poor diet, and poor adherence to cardiac drugs and to rehabilitation. Biologically: reduced heart rate variability from autonomic imbalance, platelet activation, inflammation, and activation of the hypothalamic pituitary adrenal axis. The traffic runs both ways, which is why the answer should not be written as one causing the other.
- **Making the diagnosis.** Fatigue, poor sleep and poor appetite belong to both illnesses, so weight the cognitive and affective items: low mood, anhedonia, hopelessness, guilt. Screen rather than wait for the patient to volunteer it. Then exclude the impostors: adjustment disorder, delirium after bypass surgery, hypoxia, and the effects of drugs.

TIER	THE AGENT OR THE MEASURE	WHY IT SITS THERE
First choice	Sertraline, with citalopram and escitalopram acceptable	**Best cardiac safety evidence after infarction**
Non-drug	Cardiac rehabilitation with an exercise component; cognitive behavioural therapy	Improves mood, and the exercise limb helps the heart
Watch	Citalopram at higher dose, and other agents that prolong the QT interval	Get an electrocardiogram before and after
Avoid	Tricyclic antidepressants	**Conduction delay, tachycardia, postural drop**

- **The honest limit, and the interactions.** Treating the depression reliably improves mood, function and adherence; that it reduces cardiac events is not established, and trials of therapy after infarction have shown the mood gain without a survival gain. Serotonergic drugs with antiplatelets raise bleeding risk, and strong inhibitors of CYP2C19 may blunt clopidogrel, which is a prodrug.

WHAT EARNS THE MARKS

- **Independence of the prognosis stated,** not just that the two occur together.
- **Both mechanisms, behavioural and biological.**
- **Sertraline named, and tricyclics named as the thing to avoid.**
- **The limit admitted,** since claiming a survival benefit is the error this question invites.

SEE ALSO Slot IV - 22 consultation liaison psychiatry Slot IV - 29 psychological intervention in bronchial asthma

SPRINT Day 40, Medical and systemic-disease psychiatry

IV - 28

Discuss the role of the psychiatrist in kidney transplant. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Why kidney is different**	A living-donor programme, and a dialysis population
3	**The recipient**	Depression on dialysis, cognition, adherence
3	**The living donor**	Voluntariness, the committee, coercion at home
2	**Prescribing and after**	The table, and what follows graft failure

WHY THIS QUESTION IS NOT THE GENERIC TRANSPLANT QUESTION

Kidney is the transplant with a large living-donor programme, so the donor assessment is a routine part of the work rather than an occasional one. And the recipient has usually spent years on dialysis, which supplies both the commonest psychiatric problem in this group and the best available test of adherence.

- **The recipient.** Depression is the commonest psychiatric disorder in end-stage renal disease, is under-recognised, and is associated with poorer outcome and with withdrawal from dialysis. Score it on the cognitive items, because fatigue, poor sleep and poor appetite belong to uraemia and to dialysis. Assess cognition, which is often impaired. Then read the dialysis record: attendance, fluid restriction and diet are the behavioural rehearsal for post-transplant immunosuppression.
- **The living donor.** Interview the donor alone, without the family in the room. Test voluntariness, capacity, understanding of the risk, and the absence of payment or pressure. Donation is governed by the Transplantation of Human Organs and Tissues Act 1994 as amended in 2011, under which an authorisation committee scrutinises donation by anyone who is not a near relative. Coercion inside a family is the harder case, and in Indian practice it falls most often on wives, daughters-in-law and unmarried sisters.

PRESCRIBING	WHAT TO DO	WHY
Usual first choices	Sertraline or citalopram, started low	Little renal handling, few graft interactions
Reduce or avoid	Renally cleared agents, including amisulpride, sulpiride, paliperidone, gabapentin, pregabalin	**They accumulate as clearance falls**
Special care	Lithium	**The graft is the clearance route now**
Expect from the graft drugs	Steroid mood change; calcineurin inhibitor tremor, insomnia, confusion	Treat the cause, not a new diagnosis

WHAT EARNS THE MARKS

- **Depression on dialysis named as the commonest problem,** and scored on cognitive items.
- **The donor interviewed alone, and the Act and committee named.**
- **Coercion inside the family raised,** which is the part a generic answer never reaches.
- **Renal clearance driving the prescribing,** not a list of favourite antidepressants.

SEE ALSO Slot IV - 24 the psychiatrist in organ transplant Slot IV - 22 consultation liaison psychiatry **SPRINT** Day 40, Medical and systemic-disease psychiatry

IV - 29

Discuss psychological intervention in bronchial asthma. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	How the two interact	Perception, adherence, and the panic overlap
2	Assess before intervening	Objective control set against reported symptoms
4	The interventions	The flowchart: three mismatches, three answers
2	The drugs, both ways	What the asthma drugs do, and what to avoid

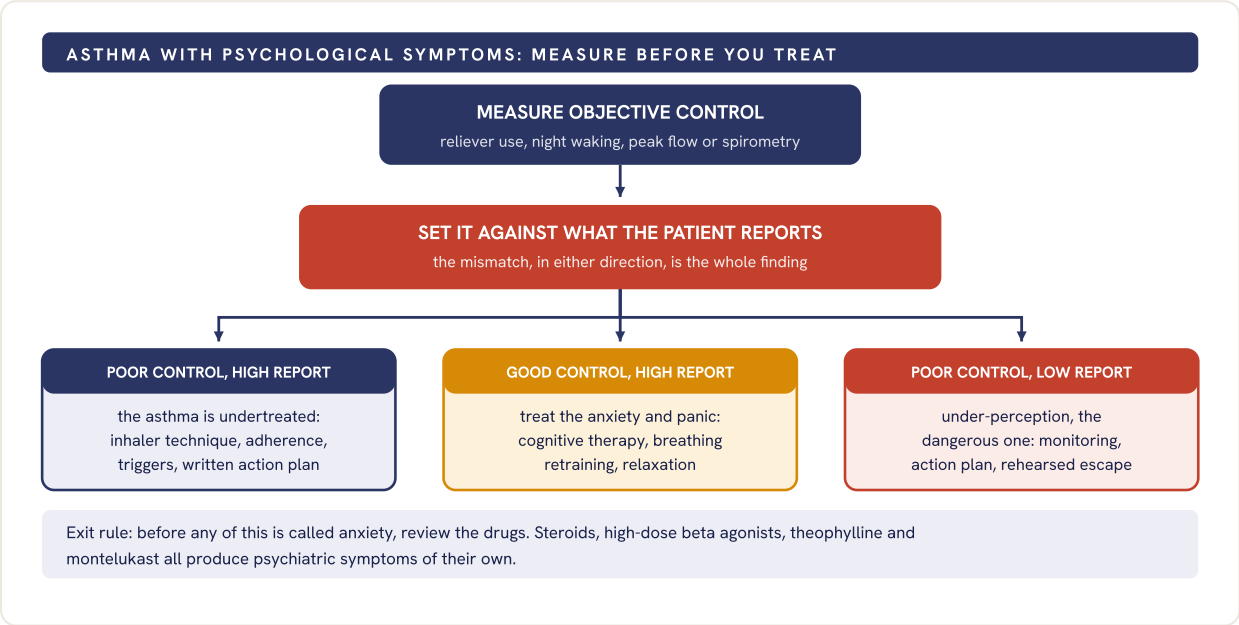


Fig 029.1 The intervention is chosen by the direction of the mismatch between measured control and reported symptoms, which is why nothing is prescribed before the measurement. Indigo optimises the asthma, amber treats the anxiety, red is danger.

- **What the evidence supports.** Self-management education with a written action plan and regular review is the best-evidenced behavioural intervention in asthma. Breathing retraining improves symptoms and quality of life without changing airway measures. Cognitive therapy helps comorbid anxiety and depression; its effect on asthma outcome is inconsistent. Family work matters in children.
- **The overlap, and the prescribing.** Panic and asthma share breathlessness, chest tightness and tachycardia, and each is mistaken for the other; a reliever taken for panic adds tremor and closes the loop. Serotonin reuptake inhibitors are usually safe; use benzodiazepines briefly if at all where there is respiratory compromise.

WHAT EARNS THE MARKS

- **Objective control measured before anything is called psychological.**
- **Under-perception named as the dangerous mismatch,** which almost no answer includes.
- **Breathing retraining described accurately:** symptoms and quality of life, not lung function.
- **The asthma drugs reviewed as a psychiatric cause,** before a psychiatric diagnosis is made.

SEE ALSO Slot IV - 27 depression with ischaemic heart disease Slot IV - 22 consultation liaison psychiatry **SPRINT** Day 40, Medical and systemic-disease psychiatry

IV - 30

What are the clinical features of irritable bowel syndrome? Discuss its pharmacological and non-pharmacological management. [4+6]

4 + 6 | CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	Clinical features	The criteria, the subtypes, and the alarm features
3	Non-pharmacological	A confident diagnosis, diet, and the therapies
3	Pharmacological	The flowchart: by subtype, then the neuromodulator

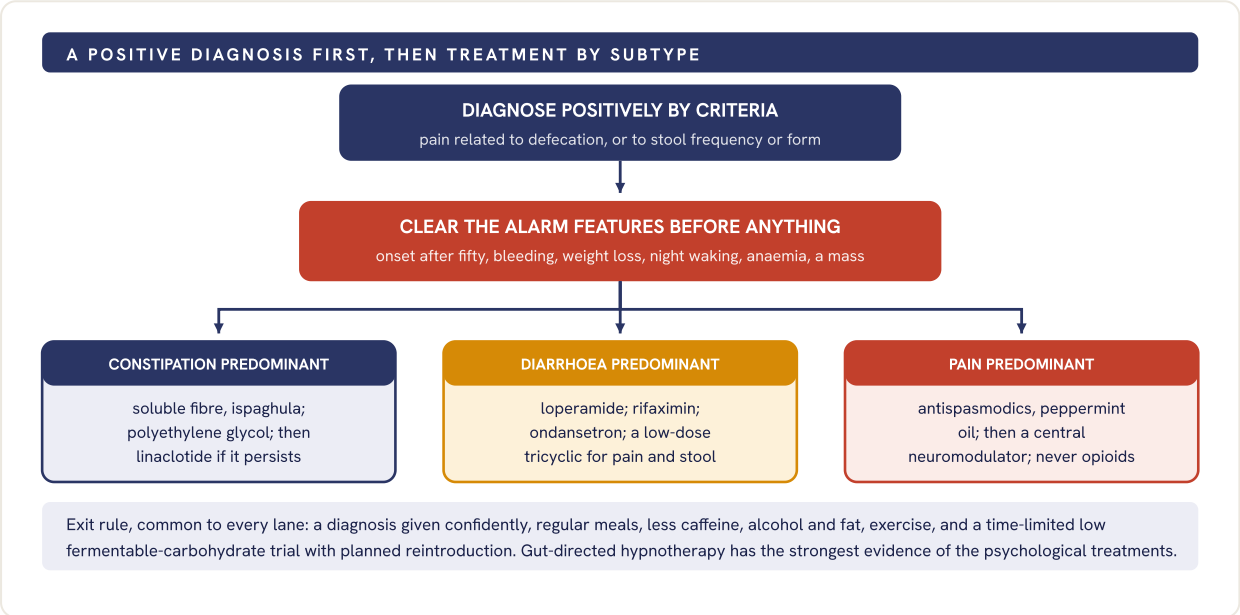


Fig 030.1 The alarm features sit between the diagnosis and every treatment lane, because they are the one thing that removes this diagnosis rather than modifying it. Indigo is structure, amber the fullest lane, red exclusion and pain.

- **The clinical features.** Recurrent abdominal pain, on average at least one day a week over three months, related to defecation or to a change in stool frequency or form, with onset at least six months earlier. Bloating, urgency, mucus and a sense of incomplete evacuation travel with it, symptoms worsen with meals and with stress, and examination is normal. Subtypes follow the predominant stool form: constipation, diarrhoea, mixed, or unclassified.
- **The mechanism, and the sentence patients need.** A disorder of gut-brain interaction: visceral hypersensitivity, altered motility and altered central pain processing, with anxiety, depression and early adversity over-represented. Say that a tricyclic is being used at a low dose for the gut and not as an antidepressant, or the patient reads the prescription as a verdict and stops it.

WHAT EARNS THE MARKS

- **The criteria given with their time frame,** not a vague description of a sensitive bowel.
- **The alarm features listed,** since they are what makes the positive diagnosis safe.
- **Treatment organised by subtype,** rather than one list of every drug ever used.
- **The neuromodulator explained to the patient,** which is the psychiatrist's specific contribution.

SEE ALSO Slot IV - 23 fibromyalgia Slot IV - 22 consultation liaison psychiatry **SPRINT**

Day 40, Medical and systemic-disease psychiatry

AREA 04 OF 15 · P4-A02

Clinical neurology, headache and sleep neurology

8	8.0%	IV-31	IV-38
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 7

REVISION ORDER

The three headache slots first and as one block, because the classification slot opens both of the others. The two sleep slots next, and they share their pharmacology. The imaging and electrophysiology slot after that. Auditory event related potentials and neurological soft signs are independent of everything else here.

WHAT IS IN THIS AREA

Headache in three slots: migraine classified with its epidemiology, course and treatment, tension headache, and a classification slot that runs on to the acute and preventive treatment of the chronic tension type. Sleep in two, insomnia in old age and the orexins with the receptor antagonists. The registered name of this area stops there, and three of its eight slots are investigation questions: radiological and electrophysiological imaging, auditory event related potentials, and neurological soft signs. They are answered on their pages as they were set.

IV - 31

Describe the use of radiological and electrophysiological investigations in psychiatric illnesses. [5+5]

5 + 5 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The principle**	These tests exclude and localise; they do not diagnose
3 **Radiological**	Structural first, functional only where it changes a decision
3 **Electrophysiological**	The record, the sleep study, the evoked potential
2 **Choosing, and limits**	Who to scan, and what a normal result means

THE PRINCIPLE

No psychiatric diagnosis is made by imaging or by a recording. They are ordered to exclude a treatable physical cause, to localise one that is suspected, and in research to test a mechanism. An answer that opens by listing machines rather than indications loses the first two marks.

INVESTIGATION	WHAT IT SHOWS	ORDERED IN PSYCHIATRY WHEN	THE CAVEAT
CT head	Bleed, calcification, gross lesion	Acute, uncooperative, or metal in situ	Poor for temporal lobe
MRI brain	Grey and white matter, hippocampus, small vessels	First episode with atypical features, late onset, focal signs, dementia	**Preferred structural test**
PET and SPECT	Regional metabolism or perfusion	Dementia subtype in doubt after MRI	Tertiary and research use
EEG	Slowing, epileptiform activity, periodic patterns	Delirium, suspected seizure, encephalitis, catatonia	**A normal record excludes little**
Polysomnography	Sleep architecture, apnoea, limb movements	Suspected apnoea, narcolepsy, parasomnia	Not for ordinary insomnia
Evoked potentials	Averaged, time-locked cortical responses	Research, and hearing or nerve conduction questions	**No diagnostic role yet**

Amber cells are the discriminators. Each row is an indication, not a machine, which is the form the question is asking for.

- **The patterns worth naming.** Temporoparietal hypometabolism in Alzheimer disease, frontal in frontotemporal dementia, occipital in Lewy body disease; triphasic waves in hepatic encephalopathy.
- **Indian practice.** The record and computed tomography are widely available; magnetic resonance is district level or above, and metabolic imaging is essentially tertiary, so the sequence has to be defensible with what is at hand.

WHAT EARNS THE MARKS

- **Indications written before modalities.** The examiner is checking clinical reasoning, not equipment knowledge.
- **Both halves answered separately.** Five marks of imaging and five of electrophysiology, not eight and two.
- **At least two named patterns.** A pattern shows the test was understood rather than listed.

SEE ALSO Slot IV - 36 auditory event related potentials Slot IV - 38 neurological soft signs

IV - 32

Classify migraine and discuss its epidemiology, course and treatment. [4+2+2+2]

4 + 2 + 2 + 2 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Classification	The families, with the criteria that define each
2	Epidemiology	Who gets it, and the psychiatric company it keeps
2	Course	Onset, decline, and what turns it chronic
3	Treatment	Two arms, each chosen by the comorbidity present

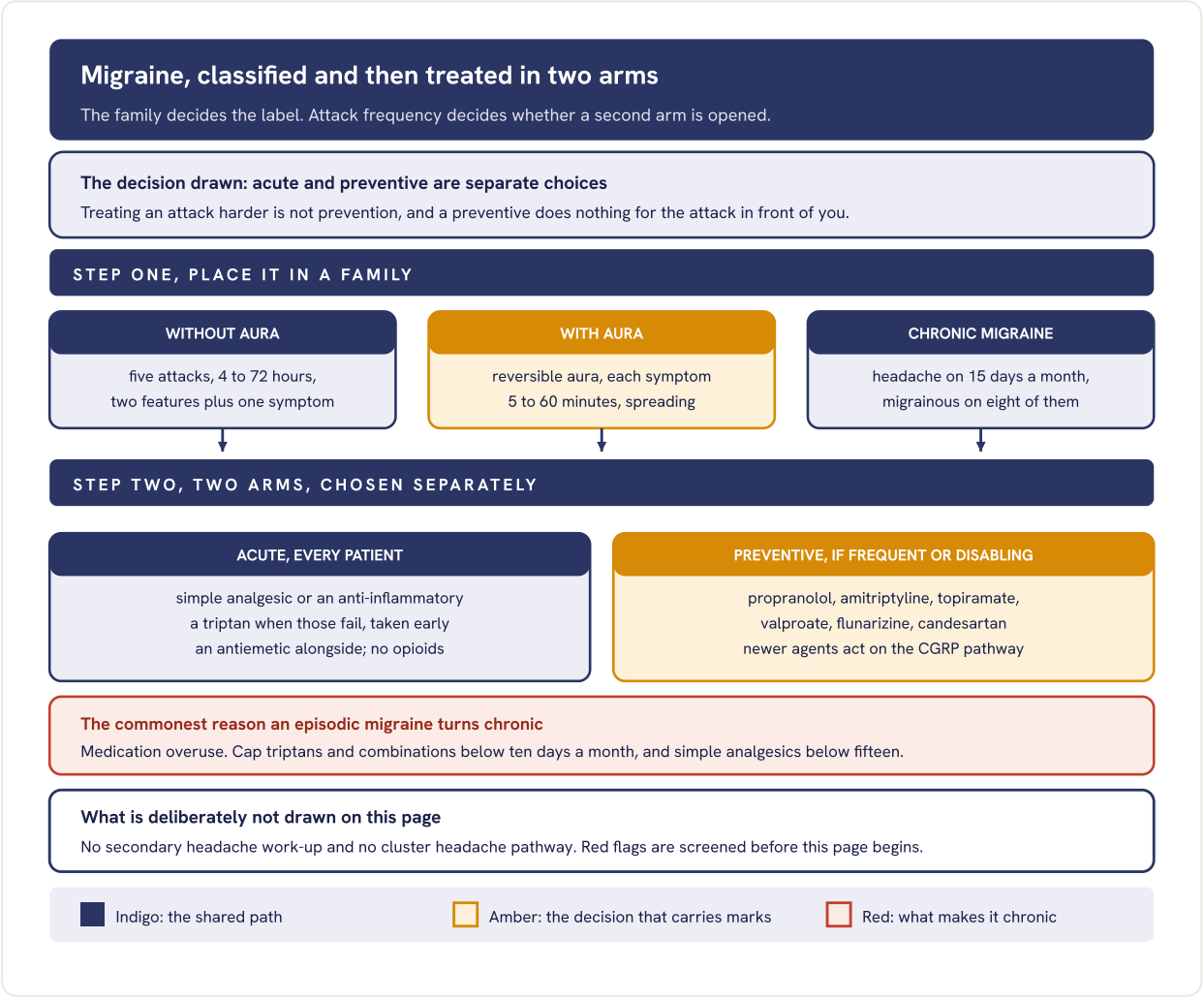


Fig 032.1 The three families most often asked about, then the two treatment arms kept apart, with medication overuse carried as a red strip because it is the modifiable driver of chronic migraine.

- **Epidemiology and course.** Two to three times commoner in women, onset in adolescence or early adult life, peak burden through the working years, and attacks usually easing after menopause.
- **Choosing the preventive by the comorbidity.** Amitriptyline where sleep is poor or tension-type headache overlaps; propranolol avoided in depression and asthma; valproate avoided in women of childbearing potential; flunarizine can cause depression and parkinsonism.

PITFALL

Migraine keeps psychiatric company: depression, anxiety and bipolar disorder are all commoner, and the relationship with depression runs both ways. Treating only the headache in a psychiatry answer misses marks the examiner has already allotted.

WHAT EARNS THE MARKS

- **Named families with their defining numbers,** not a list of headache types.
- **Acute and preventive answered as separate arms,** with the threshold for opening the second one.
- **One preventive chosen by comorbidity,** which is what a psychiatry examiner is looking for.
- **Medication overuse named as the driver of chronicity.**

SEE ALSO Slot IV - 35 classification of headache Slot IV - 33 tension type headache **SPRINT** Day 31, Neurology foundations for psychiatrists

IV - 33

Clinical features and management of Tension head ache. [10]

10 CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 **Clinical features**	Bilateral, pressing, and not worsened by activity
2 **Grade it by frequency**	Infrequent, frequent episodic, chronic
3 **Management**	A capped acute plan, then a preventive if frequent
2 **Non-drug and mood**	Biofeedback, therapy, sleep, and the overuse trap

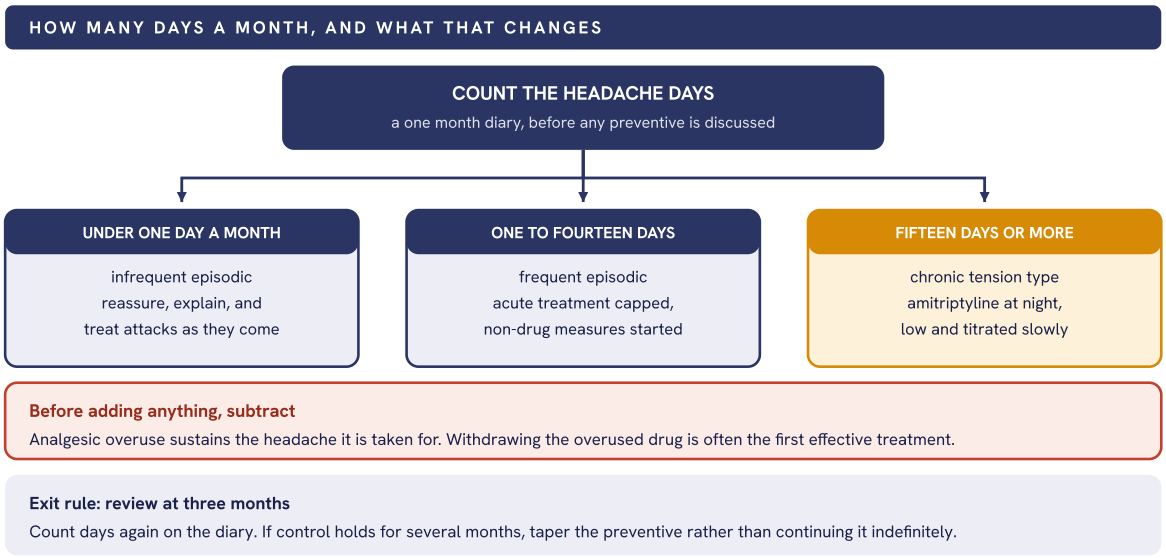


Fig 033.1 Headache days a month set the grade, and the grade decides whether a preventive is opened at all, with analgesic withdrawal placed before any addition.

- **The features that define it.** Bilateral, pressing rather than pulsating, mild to moderate, not worsened by activity, no vomiting, and at most one of light or sound sensitivity.
- **The one positive sign.** Pericranial tenderness on palpation, which also names the subtype and points to the physiotherapy and biofeedback half of treatment.

PITFALL

Amitriptyline is the preventive with the evidence here; the serotonin reuptake inhibitors have not shown benefit for this headache, even when mood is low.

WHAT EARNS THE MARKS

- **Features written as the criteria that exclude migraine,** not as a general description of head pain.
- **The frequency grade stated,** because it is what decides whether a preventive is justified.
- **Withdrawal of overused analgesics placed before any new drug.**

SEE ALSO Slot IV - 35 classification of headache Slot IV - 32 migraine SPRINT
Day 31, Neurology foundations for psychiatrists

IV - 34

There is sudden surge of patients with insomnia in psychiatry OPDs. What is physiological insomnia? What are the causes of insomnia in old age? Discuss its management in detail of such a case. [2+3+5]

2 + 3 + 5 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Physiological sleep change**	What ageing alone does, and where disorder begins
3	**Causes in old age**	Five groups, screened in a single pass
4	**Management**	Behavioural first, drug last, shortest and lowest
1	**What not to prescribe**	The sedatives that buy sleep with falls

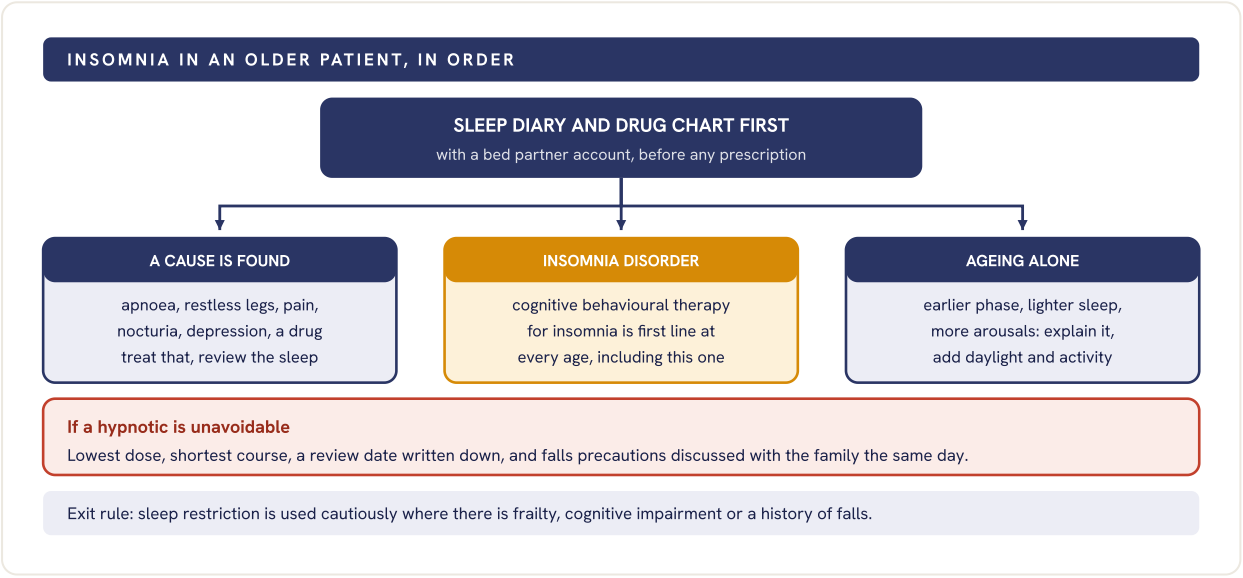


Fig 034.1 The diary and the drug chart decide which of three routes the case takes, with the hypnotic placed after all three rather than inside any of them.

- **Reading the stem.** Physiological insomnia is not a formal category. The defensible reading is normal age-related change: lighter sleep, an advanced phase and more night waking, without daytime impairment.
- **The five groups of cause.** Primary sleep disorders, medical illness, psychiatric illness including bereavement and alcohol, drugs given at the wrong hour, and circadian or behavioural factors.

PITFALL

Benzodiazepines and the Z drugs are potentially inappropriate in older adults: falls, fractures, confusion, dependence. Sedating antihistamines add delirium risk on top.

WHAT EARNS THE MARKS

- **Normal ageing separated from insomnia disorder** in the first two lines, which is what the first part asks.
- **Causes grouped, not listed.** Five headings beat twenty items for the same three marks.
- **Cognitive behavioural therapy named as first line,** with the drug chart reviewed before any drug is added.

SEE ALSO Slot IV - 37 orexin receptor antagonists **SPRINT** Day 17, Eating & sleep-wake disorders

IV - 35

Classify headache and describe acute and preventive treatment of chronic tension type headache. [4+3+3]

4 + 3 + 3 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4 Classification	Three parts, then the primary families inside part one
3 The acute arm	Capped deliberately; the cap is itself the treatment
3 The preventive arm	The drug, the titration, the review, and what is added alongside

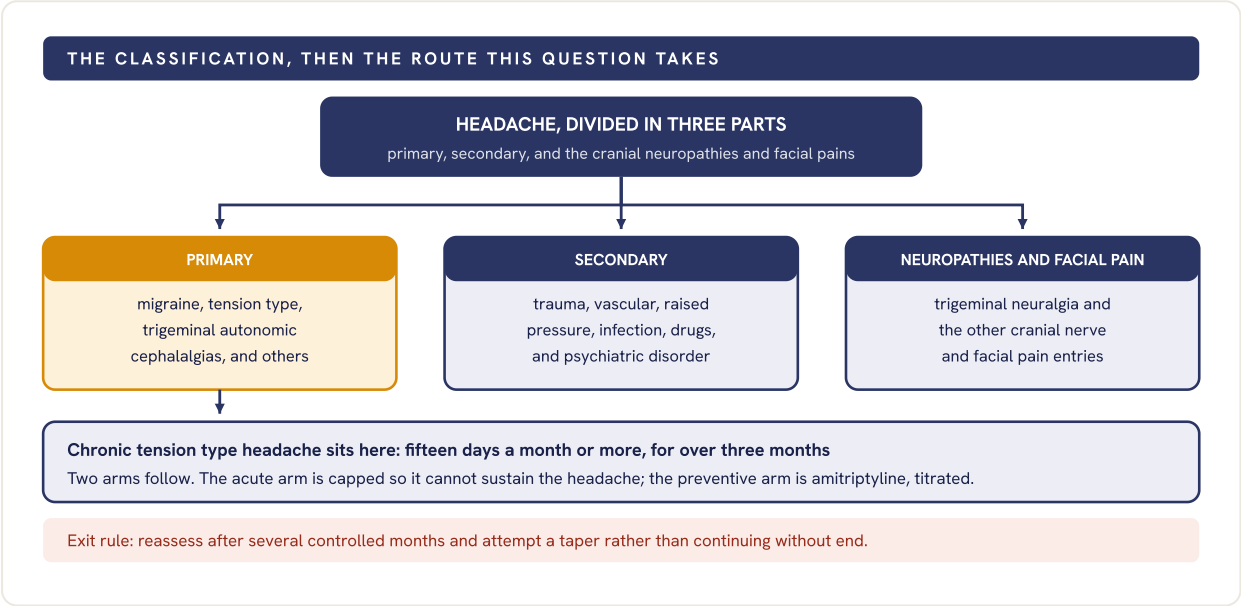


Fig 035.1 The three parts of the classification, with the route this question takes highlighted, so the four classification marks and the six treatment marks sit on one picture.

- **The acute arm, and why it is capped.** Simple analgesics only, kept below fifteen days a month; beyond that they sustain the headache they were taken for.
- **The preventive arm.** Amitriptyline at a low night dose, titrated slowly over weeks to the lowest effective dose, with mirtazapine or venlafaxine as alternatives where it cannot be tolerated.

PITFALL

Biofeedback, relaxation and cognitive therapy are additive to amitriptyline rather than a softer substitute for it, and they are what makes the taper possible later.

WHAT EARNS THE MARKS

- **Three parts named before any headache type.** Listing primary headaches only answers a third of the classification.
- **The chronic threshold stated in days and months.**
- **The cap on acute treatment written as treatment,** which is the line most answers omit.

SEE ALSO Slot IV - 33 tension type headache Slot IV - 32 migraine **SPRINT** Day 31, Neurology foundations for psychiatrists

IV - 36

Discuss about auditory event related potential (ERPs) and its use in psychiatry research. [3+7]

3 + 7 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 What it is	Averaged, time-locked, named by polarity and latency
2 The components	What each paradigm actually probes
3 Use in research	Endophenotype, mechanism, risk and staging
2 Limits	Group level, unspecific, and not diagnostic

THE DEFINITION

An event related potential is a voltage change in the ongoing record that is time locked to a stimulus, and pulled out of background activity by averaging many trials. Each component is named by its polarity and its approximate latency in milliseconds, and reported as amplitude and latency.

COMPONENT	PARADIGM	WHAT IT PROBES	MAIN PSYCHIATRIC FINDING
P50	Paired clicks, ratio of the second to the first	Sensory gating, early and automatic	**Poor suppression in schizophrenia and in relatives**
Mismatch negativity	Oddball with deviant tones, attention not required	Pre-attentive change detection	**Reduced in schizophrenia; among the most replicated**
P300	Oddball, the rare target counted	Attention and working memory updating	**Smaller amplitude, longer latency in schizophrenia**
N400	Sentences ending unexpectedly	Semantic processing	Abnormal where thought disorder is present

Amber cells are the discriminators. Naming the paradigm alongside the component is what separates understanding from a list of labels.

- **Why research uses them.** They are endophenotypes, present in unaffected relatives, so they index liability rather than illness. They probe mechanism, mismatch negativity being tied to glutamate function and P50 to nicotinic receptors, and they are studied for predicting transition in people at clinical high risk.
- **The honest limit.** These are group differences with overlapping distributions, altered by medication, attention, smoking and age. No component diagnoses an individual, and none is used clinically for that purpose.

WHAT EARNS THE MARKS

- **Averaging and time locking in the definition.** Without them this is just an ordinary recording.
- **Each component tied to its paradigm,** which is where the seven discussion marks sit.
- **Endophenotype used precisely,** meaning present in relatives, not merely abnormal in patients.
- **The limit stated.** A research tool described as a diagnostic test reads as unfamiliarity.

SEE ALSO Slot IV - 31 investigations in psychiatric illness Slot IV - 38 neurological soft signs

IV - 37

Discuss the role of orexins in sleep and wakefulness. How orexin receptor antagonists are helpful in sleep issues? [5+5]

5 + 5 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The system	Two peptides, one small nucleus, two receptors
3 What they do	Hold the wake side of the switch steady
3 Blocking them	Removing wakefulness rather than adding sedation
2 Cautions	Narcolepsy, dreaming, next day, interactions

THE SYSTEM, IN ONE PARAGRAPH

Orexin A and B, also called hypocretins, come from a small group of neurons in the lateral hypothalamus and act at two receptors. They project to the noradrenergic, serotonergic, histaminergic and cholinergic arousal nuclei, and hold the wake side of the sleep-wake switch stable, which is why their loss produces narcolepsy with cataplexy rather than simple sleepiness.

DOMAIN	BENZODIAZEPINE RECEPTOR AGONIST	OREXIN RECEPTOR ANTAGONIST	WHAT SETTLES IT
Mechanism	Enhances inhibition across the brain	Removes one wake-promoting signal	**Subtraction, not sedation**
Sleep architecture	Deep sleep reduced, REM suppressed	Architecture better preserved, REM increased	**The main pharmacological claim**
Which insomnia	Mainly onset	Onset and maintenance	Maintenance is the gap it fills
Next day and falls	Psychomotor slowing, falls in the elderly	Less impairment at licensed doses	**Why it is proposed in old age**
Dependence	Tolerance, rebound, withdrawal	Low abuse potential, less rebound	Still prescribed with a review date

Amber cells are the discriminators. Every row follows from the first: blocking a wake signal is a different act from deepening inhibition.

- **The agents.** Suvorexant, lemborexant and daridorexant block both receptors. Beyond sleep, the same system links arousal to feeding and reward, which is why orexin is also studied in addiction.
- **Cautions.** Contraindicated in narcolepsy. Vivid dreams, sleep paralysis and next-day somnolence occur, metabolism is through cytochrome P450 3A4, and cost and availability limit use in Indian practice.

WHAT EARNS THE MARKS

- **The switch model named,** with orexin stabilising it rather than simply causing wakefulness.
- **Narcolepsy used as the proof of function,** which shows the physiology was understood.
- **Subtraction contrasted with sedation,** the single line that answers the second half.

SEE ALSO Slot IV - 34 insomnia in old age **SPRINT** Day 17, Eating & sleep-wake disorders

IV - 38

Neurological soft signs. [10]

10 CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	Definition	Minor, non-localising, and no focal lesion behind them
3	Domains and tests	What is examined, and with what at the bedside
3	Why psychiatry cares	Neurodevelopmental evidence and the relatives
2	Limits	Unspecific, confounded, and not diagnostic

THE DEFINITION

Neurological soft signs are minor, non-localising abnormalities of motor coordination, sensory integration, the sequencing of complex acts and the primitive reflexes, found without a focal lesion or a defined neurological disorder.

DOMAIN	BEDSIDE TEST	A POSITIVE LOOKS LIKE	NOTE
Motor coordination	Rapid alternating movements, finger to thumb, tandem walk	Clumsy, slow, breaks down on speed	Easiest to elicit
Sensory integration	Stereognosis, graphaesthesia, two-point discrimination, face-hand test	Cannot name an object or a traced figure by touch	Needs a cooperative patient
Sequencing	Fist-edge-palm, and other three-step motor sequences	Order lost after a few repetitions	The most discriminating domain
Primitive reflexes	Grasp, snout, glabellar tap; mirror movements	Present, or not extinguishing on repetition	Age norms matter
Rating	Neurological Evaluation Scale, Cambridge Neurological Inventory	Scored, so change can be followed	Name one in the answer

Amber cells are the discriminators. A named scale and a named test are what turn a definition into an examination the candidate could actually perform.

- **Why they matter in psychiatry.** Commoner in schizophrenia, including in drug-naive first-episode patients and unaffected relatives, so they are read as an endophenotype and as neurodevelopmental evidence. They track with negative symptoms and poorer outcome.
- **The honest limit.** Not specific: rates rise in bipolar disorder, obsessive compulsive disorder and with ageing, and drug-induced extrapyramidal signs confound the motor domain.

WHAT EARNS THE MARKS

- **Non-localising written into the definition,** with the absence of a focal lesion stated.
- **Domains with an actual test in each.** Naming the fist-edge-palm sequence beats naming three more syndromes.
- **Drug-naive patients and relatives cited,** because that is what makes the endophenotype claim.
- **One named scale,** and one honest statement of non-specificity.

SEE ALSO Slot IV - 31 investigations in psychiatric illness Slot IV - 36 auditory event related potentials SPRINT Day 31, Neurology foundations for psychiatrists

AREA 05 OF 15 · P4-A07

Stroke, TBI and focal brain syndromes

8	7.9%	IV-39	IV-46
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 · HIGH 1 · SINGLE 5

REVISION ORDER

The five head injury slots as one block and in that order, because each adds an axis to the answer before it: manifestations, severity, prognosis, the post-concussional picture, then the lesion site. Cerebral small vessel disease next, it is where the stroke content sits. Subdural haematoma and Kluver-Bucy last, and both are self-contained.

WHAT IS IN THIS AREA

Head injury in five of the eight slots: the neuropsychiatric manifestations with their management, the severity indices with the sequelae, the definition with the prognostic factors, the post-concussional syndrome, and the personality change read against the site of the lesion. Cerebral small vessel disease and subdural haematoma carry the vascular and the surgical limb, and the Kluver-Bucy syndrome stands on its own.

IV - 39

Discuss the neuropsychiatric manifestations of head injuries and their management. [10]

10 STROKE, TBI AND FOCAL BRAIN SYNDROMES

KUHS, NTRUHS, RGUHS, TN-MGRMU - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2	Injury and severity	Primary and secondary damage, and how severity is graded
4	The syndromes	Grouped by time from injury, not listed at random
3	Management	Non-drug first, then drugs at a lowered threshold
1	Prognosis	What predicts outcome, and the medicolegal line

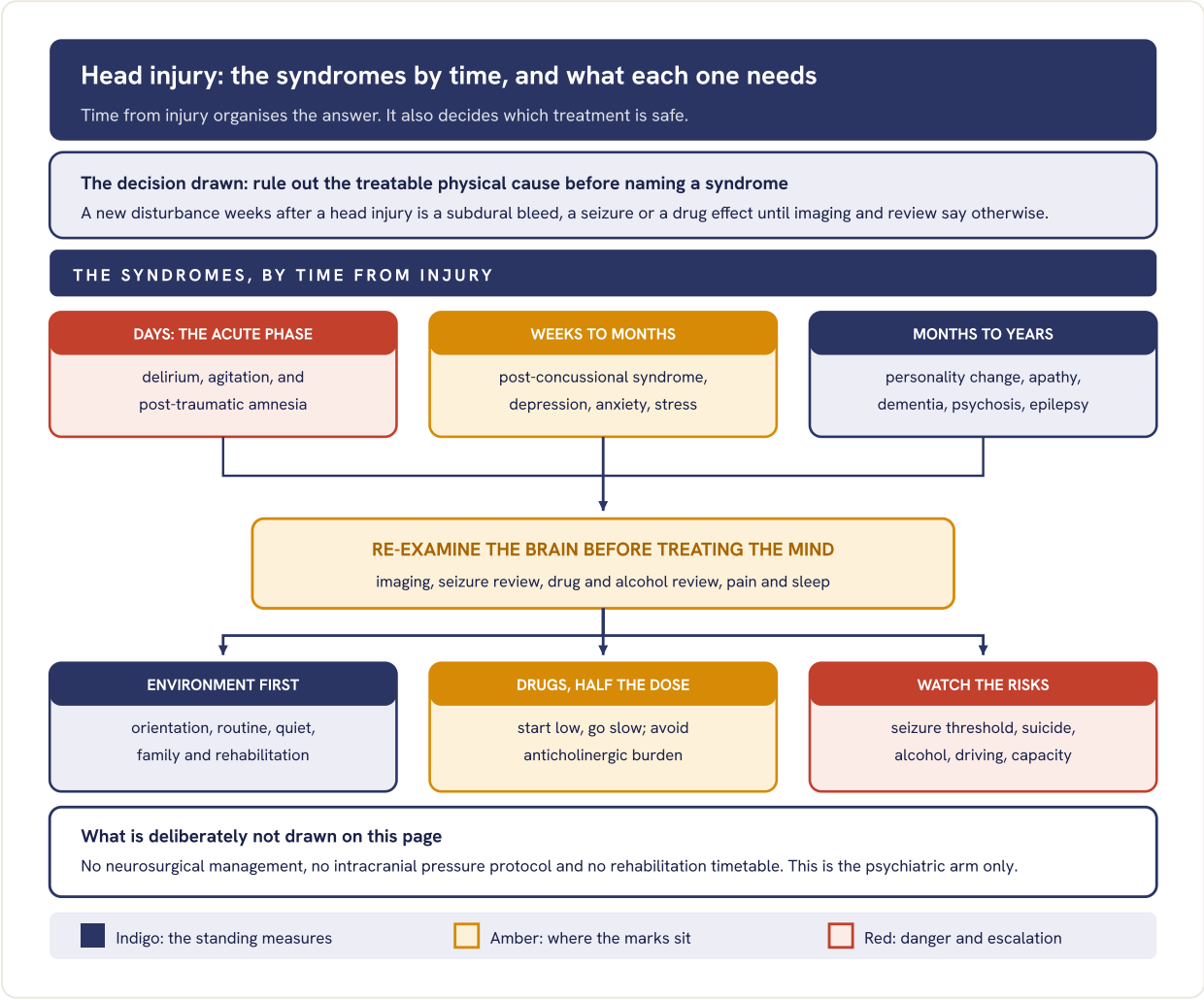


Fig 039.1 The syndromes grouped by time from injury, the physical review that must precede treatment, and the three arms of management that follow.

- **Cognitive and behavioural.** Slowed processing, poor attention and impaired executive function are the commonest and most disabling. Behavioural change follows lesion site: disinhibition and poor judgement from orbitofrontal damage, apathy from medial frontal damage, irritability and aggression from temporal damage.

- **Affective and psychotic.** Depression is the commonest psychiatric diagnosis after head injury and carries a raised suicide risk. Anxiety and post-traumatic stress disorder are common, and can coexist with amnesia for the event itself. Psychosis is uncommon, late, and more likely with temporal injury and post-traumatic epilepsy.
- **The prescribing rule.** The injured brain is more sensitive to every psychotropic. Start at roughly half the usual dose, titrate slowly, and prefer agents with low anticholinergic and low seizure liability. Benzodiazepines worsen cognition and disinhibition, so they are short term at most.

WHAT EARNS THE MARKS

- **Syndromes grouped by time from injury.** An unordered list of diagnoses reads as recall.
- **The physical review written before the psychiatric treatment.**
- **The lowered dosing threshold, stated as a rule** rather than left implied.
- **Depression named as the commonest,** with its suicide risk.

SEE ALSO Slot IV - 42 traumatic brain injury and prognosis Slot IV - 46 personality change by lesion site **SPRINT** Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 40

How will you quantify the severity of head injury? Discuss neuropsychiatric sequelae of head injury. [4+6]

4 + 6 | STROKE, TBI AND FOCAL BRAIN SYNDROMES

DNB, KNRUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

4	Quantifying severity	Three indices, each measuring a different thing
3	Early sequelae	Delirium, amnesia, the post-concussional cluster
2	Late sequelae	Cognitive, affective, behavioural, epileptic
1	The link	Which sequelae severity predicts, and which it does not

INDEX	MILD	MODERATE	SEVERE
Glasgow Coma Scale, at presentation	13 to 15	9 to 12	8 or below
Loss of consciousness	Under 30 minutes	30 minutes to 24 hours	Over 24 hours
Post-traumatic amnesia	Under 24 hours	1 to 7 days	Over 7 days
Structural imaging	Usually normal	Often abnormal	Abnormal

Amber marks post-traumatic amnesia, the single best predictor of outcome. Quoting all three indices, and saying which one predicts best, is what the four marks are for.

- **The three measure different things.** The coma scale is a snapshot at presentation and is degraded by alcohol, sedation and intubation. Post-traumatic amnesia is measured forwards, as the interval until continuous day to day memory returns, and it predicts outcome better than the admission score.
- **The sequelae, by domain.** Cognitive: attention, processing speed and executive function. Affective: depression is the commonest diagnosis, with anxiety and post-traumatic stress disorder. Behavioural: irritability, aggression, disinhibition, apathy. Late and uncommon: psychosis, and post-traumatic epilepsy.
- **The asymmetry.** Severity predicts cognitive impairment and epilepsy well, but post-concussional symptoms and depression follow mild injury just as often as severe. A normal scan and a good coma score never promise a good psychiatric outcome.

WHAT EARNS THE MARKS

- **All three indices, with their bands.** Quoting the coma scale alone answers a quarter of the question.
- **Post-traumatic amnesia defined properly,** as the return of continuous memory rather than as confusion.
- **Sequelae grouped by domain,** and the honest line that mild injury still produces them.

SEE ALSO Slot IV - 42 traumatic brain injury and prognosis Slot IV - 44 post-concussional syndrome SPRINT Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 41

Describe presentation of cerebral small vessel disease in psychiatry. How to manage such a case? [4+6]

4 + 6 | STROKE, TBI AND FOCAL BRAIN SYNDROMES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	The vessel, the lesions, and what imaging shows
3 Presentation	Vascular depression, slowing, gait, and the cognitive profile
4 Management	The vessels first, then mood, then safety
1 Course	Stepwise decline, and what the family is told

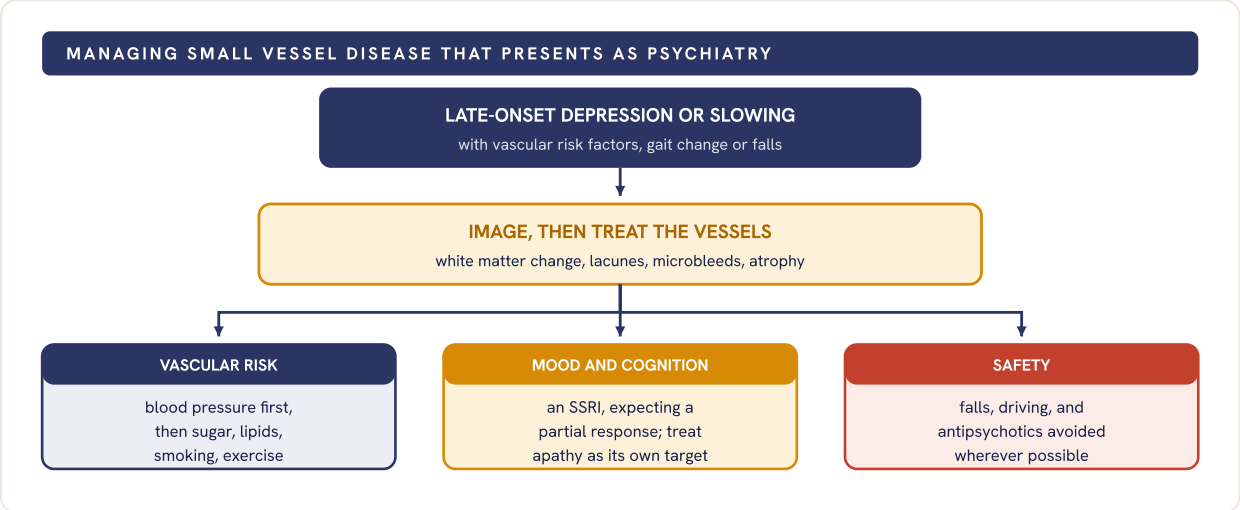


Fig 041.1 One presentation, one confirmatory step, and three arms of management with the vessels treated before the mood.

- **The presentation.** Late-onset depression with prominent apathy and psychomotor slowing, executive and processing-speed impairment with memory relatively spared, unsteady small-stepped gait, urinary urgency and emotional lability. Psychosis is uncommon and late.
- **Vascular depression.** The concept links late-onset depression, executive dysfunction and white matter disease, and it predicts a slower and less complete antidepressant response. Naming it, and saying what it predicts, is the mark.

PITFALL

Antipsychotics raise the risk of stroke and death in older people with vascular brain disease, so agitation is managed by environment and by treating pain, constipation and infection before any drug. Tricyclics are avoided for anticholinergic burden and postural drop, which turns into falls.

WHAT EARNS THE MARKS

- **Blood pressure named as the first treatment,** ahead of any psychotropic.
- **The cognitive profile stated as executive and speed,** not as amnesia.
- **The partial antidepressant response predicted in advance,** so it is not read as treatment failure.

SEE ALSO Slot IV - 52 depression after stroke Slot IV - 19 subcortical dementias SPRINT
Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 42

What is traumatic brain injury? What are the prognostic factors that determine outcome of traumatic brain injury? Discuss the psychiatric sequelae of head injury. [2+3+5]

2 + 3 + 5 | STROKE, TBI AND FOCAL BRAIN SYNDROMES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	What counts as traumatic brain injury, and the two damage phases
3 Prognostic factors	Before, during and after the injury
4 Psychiatric sequelae	By domain, with the commonest named as commonest
1 What follows	Rehabilitation, review, and the capacity question

THE DEFINITION

An alteration in brain function, or other evidence of brain pathology, caused by an external mechanical force. Primary damage is done at impact by contusion, laceration and diffuse axonal injury; secondary damage follows over hours from hypoxia, hypotension, oedema, haematoma and raised intracranial pressure, and it is the part that treatment can still prevent.

GROUP	FACTORS THAT WORSEN OUTCOME	WHY IT MATTERS
Before the injury	Older age, previous head injury, prior psychiatric illness, alcohol and substance use, low education and cognitive reserve	Fixed, but identifies who to follow
The injury itself	Low coma score, long post-traumatic amnesia, hypoxia and hypotension, haematoma, focal frontal or temporal lesion, seizures	**The strongest single group**
After the injury	Unemployment, poor family support, chronic pain, litigation, delayed or absent rehabilitation	**The group that is modifiable**

Amber marks the two rows worth arguing about: injury factors predict most strongly, but post-injury factors are the only ones treatment can change.

- **The sequelae.** Cognitive impairment of attention, speed and executive function; depression as the commonest diagnosis, with raised suicide risk; anxiety and post-traumatic stress disorder; personality change by lesion site; aggression and irritability; and late, uncommon, psychosis and post-traumatic epilepsy.
- **The honest qualifier.** Outcome is determined as much by what surrounds the patient as by what happened to the brain. Saying so, and naming rehabilitation and family support as treatment, is what lifts this answer.

WHAT EARNS THE MARKS

- **Primary and secondary damage separated in the definition.** Two marks are cheaply lost here.
- **Prognostic factors grouped into before, during and after.** An unsorted list scores lower than the same facts sorted.
- **Depression named as the commonest sequela,** with its suicide risk stated.

SEE ALSO Slot IV - 40 quantifying severity of head injury Slot IV - 39 management of head injury **SPRINT** Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 43

Subdural hematoma. [10]

10 STROKE, TBI AND FOCAL BRAIN SYNDROMES

KUHS, NTRUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	What it is	Bridging veins, the subdural space, acute and chronic
2	Who gets it	The psychiatric populations that are already at risk
3	How it presents	The masquerade: dementia, depression, delirium
3	Diagnosis and management	Image early, refer early, and what happens after

THE DEFINITION

Bleeding into the space between dura and arachnoid, classically from torn bridging veins. Because the source is venous and low pressure, the collection can enlarge slowly, and the interval between the injury and the presentation may be long enough that neither patient nor family connects the two.

FEATURE	ACUTE	CHRONIC
Time from injury	Within three days	Three weeks or more
Typical patient	Any age, significant trauma	Older, alcohol dependent, on anticoagulants, epileptic
The injury	Always remembered	Often trivial, and frequently not recalled at all
Appearance on scan	Crescentic, bright, crossing suture lines	Crescentic, dark or mixed, crossing suture lines
Presentation	Falling consciousness, focal signs	Fluctuating confusion, headache, gait change, personality change
Management	Urgent neurosurgical referral	Referral; small collections may be observed

Amber cells hold the two discriminators. A crescentic collection that crosses suture lines is subdural; a lens-shaped one that does not is extradural. The forgotten injury is what makes the chronic form a psychiatric problem.

- **Why psychiatrists must know it.** The chronic form imitates whatever the patient is already thought to have. It presents as new or worsening dementia, as late-onset depression with apathy, or as unexplained delirium, and the populations at highest risk are exactly ours: older patients, alcohol dependence, falls, epilepsy and anticoagulation.
- **The rule that prevents the miss.** Any patient with fluctuating consciousness, a new focal sign, a headache that is new in character, or a cognitive decline that is faster than the diagnosis should allow, gets imaged. Attributing sudden change to a known chronic illness is how this is missed.

WHAT EARNS THE MARKS

- **Acute and chronic separated,** since they differ in patient, presentation and urgency.
- **The forgotten or trivial injury named.** It is the whole reason the diagnosis is delayed.
- **The imaging rule written as a threshold,** not as a general recommendation to investigate.

SEE ALSO Slot IV - 86 neurological complications of alcohol Slot IV - 92 delirium **SPRINT**

Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 44

Post concussional Psychiatric manifestation. [10]

10 STROKE, TBI AND FOCAL BRAIN SYNDROMES

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	What the syndrome is, and what it follows
3	The three clusters	Somatic, cognitive, affective, each named
2	Why it persists	What predicts persistence, and what does not
3	Management	Explain early, grade the return, treat what is treatable

THE DEFINITION

A cluster of symptoms following head injury, usually mild, with brief or no loss of consciousness. The classification asks for a history of head trauma together with three or more of headache, dizziness, fatigue, irritability, insomnia, difficulty with concentration or memory, and reduced tolerance of alcohol, stress or emotion.

- **The three clusters.** Somatic: headache, dizziness, fatigue, sensitivity to noise and light. Cognitive: poor attention, slowed processing, forgetfulness that is worse under pressure. Affective: irritability, anxiety, low mood, emotional lability, and preoccupation with the symptoms themselves.
- **Course and what predicts persistence.** Most patients recover within weeks to about three months. Persistence beyond that is predicted less by the severity of the injury than by prior psychiatric history, the expectation of harm, misattribution of ordinary symptoms to brain damage, ongoing litigation, and unemployment.
- **Management.** Explain early, and give a positive and specific prognosis. Advise graded return to activity rather than prolonged rest, treat headache and sleep directly, treat depression and anxiety on their own merits, and offer cognitive behavioural therapy where symptoms persist. Stop repeating normal investigations.

PITFALL

These symptoms are non-specific. They occur in healthy controls and after orthopaedic injury without any head trauma at all. Attributing every symptom to brain damage and dismissing them all as compensation seeking are the same error made in opposite directions, and both lose the patient.

WHAT EARNS THE MARKS

- **The criterion set, with the number of symptoms required.**
- **Persistence explained by maintaining factors,** not by injury severity.
- **Graded return named instead of rest.** Prolonged rest worsens this syndrome.
- **The even-handed line on litigation.** It is a maintaining factor, not a verdict on the patient.

SEE ALSO Slot IV - 40 quantifying severity of head injury Slot IV - 42 traumatic brain injury and prognosis **SPRINT** Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 45

Kluver-Bucy syndrome. [10]

10 STROKE, TBI AND FOCAL BRAIN SYNDROMES

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition and lesion	Where it was described, and what has to be damaged
4 The features	Named, each with what it looks like at the bedside
2 Causes	What actually produces it in human beings
2 Management	Treat the cause, contain the behaviour, support the family

THE DEFINITION

A behavioural syndrome that follows bilateral damage to the anterior temporal lobes, and to the amygdala in particular. Described by Kluver and Bucy in rhesus monkeys after bilateral temporal lobectomy. In human beings the complete syndrome is rare and the partial form is the rule, which is worth saying.

FEATURE	WHAT IT MEANS	HOW IT APPEARS
Visual agnosia	Failure to recognise objects by sight, with vision intact	Handles a familiar object as if it were new
Hyperorality	Examining everything by mouth	Mouthing objects, eating inedible things
Hypermetamorphosis	Compulsion to attend to every visual stimulus	Touches whatever enters the field of view
Placidity	Loss of fear and of anger	Flat, unreactive to threat or to rebuke
Altered sexual behaviour	Increased and indiscriminate	Public masturbation, inappropriate approaches
Dietary change	Hyperphagia and altered preference	Eats continuously, loses food preferences

Amber marks the two features candidates most often omit. Listing hyperorality and hypersexuality alone is the half answer that this question is set to catch.

- **Causes in practice.** Herpes simplex encephalitis is the commonest, because it targets the temporal lobes bilaterally. Also head injury, bilateral temporal lobectomy for epilepsy, frontotemporal dementia, anoxic brain injury and late Alzheimer disease.
- **Management.** Treat the underlying cause where it is treatable, and treat encephalitis urgently. Beyond that the work is behavioural: supervision, environmental control, protecting dignity, and family support. Selective serotonin reuptake inhibitors or a mood stabiliser may reduce disinhibition and sexual behaviour.

WHAT EARNS THE MARKS

- **The lesion named as bilateral and anterior temporal,** with the amygdala specified.
- **All the features, including visual agnosia and hypermetamorphosis.**
- **Herpes encephalitis named as the commonest human cause.**
- **The line that the human form is usually partial.** It shows the syndrome is understood, not recited.

SEE ALSO Slot IV - 46 personality change by lesion site Slot IV - 13 frontotemporal dementia SPRINT

Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 46

Discuss the personality changes in head injury in relation to the location in the brain. [5+5]

5 + 5 | STROKE, TBI AND FOCAL BRAIN SYNDROMES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Why location matters	The frontal-subcortical circuits, named before the list
5	The syndromes by site	Four sites, four different people
2	Assessment	How the change is established, and against what
1	Management	What actually helps, and what does not

SITE	SYNDROME	THE PERSONALITY CHANGE	HOW THE FAMILY DESCRIBES IT
Orbitofrontal	Disinhibition	**Impulsive, tactless, socially inappropriate, facile jocularity, poor judgement**	Not the same person, but sharp as ever
Dorsolateral prefrontal	Dysexecutive	Poor planning and set shifting, perseveration, concrete thinking, reduced fluency	Cannot organise anything any more
Medial frontal and anterior cingulate	Apathy	**Loss of initiation and drive, flat affect, in the extreme akinetic mutism**	Sits all day, has to be started
Anterior temporal	Irritable and aggressive	Short fuse, emotional lability, aggression; bilateral damage gives the Kluver-Bucy picture	Explodes over nothing
Diffuse axonal	Slowed	Fatigue, slowness, irritability without a focal signature	Tired and short tempered

Amber marks the two poles that make the point of the question: the same injury produces a disinhibited patient from one site and an inert one from another.

- **Assessment.** Personality change is established by informant history set against premorbid personality, not by a cross-sectional interview. Neuropsychological testing and bedside frontal tasks such as verbal fluency support it, and serial review separates change from a bad week.

PITFALL

Formal cognitive testing is often entirely normal in orbitofrontal damage. A normal mini mental state examination therefore never excludes personality change after head injury, and the patient who tests well while his life falls apart is the classic presentation, not a contradiction.

WHAT EARNS THE MARKS

- **The circuits named before the syndromes are listed.** It converts a list into an explanation.
- **Disinhibition and apathy contrasted as different sites,** which is the whole point of the question.
- **Informant history named as the method of assessment.**

SEE ALSO Slot IV - 45 Kluver-Bucy syndrome Slot IV - 39 management of head injury SPRINT Day 34, Stroke, TBI, delirium & focal brain syndromes

AREA 06 OF 15 · P4-A10

Movement and degenerative disorders in psychiatry

7	6.7%	IV-47	IV-53
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 3 HIGH 2 SINGLE 2

REVISION ORDER

The four Parkinson slots together and hardest: they cross-link each other and the safe-agent rule is identical on all of them. Take the whole-disease slot first, it half writes the other three. The two multiple sclerosis slots next, they are one answer at two widths and they cross-link each other. Huntington last, and separately.

WHAT IS IN THIS AREA

Parkinson disease in four slots: the psychological and psychiatric features, the drug induced problems with their management, the relationship with psychosis, and one that asks for the whole disease from aetiology to prognosis. Multiple sclerosis in two, the neuropsychiatric aspects and the aetiology, features and management. Huntington disease takes the seventh. One of the Parkinson slots carries a second limb on the assessment and management of depression after stroke, which the board set there and which is answered on that page as it was set.

IV - 47

Describe the psychological and psychiatric features of Parkinson's disease. [10]

10 MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

KUHS, NTRUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2	Why they occur	Part of the disease, not merely a reaction to it
4	The syndromes	Six, each with what makes it look different here
2	Sleep and the prodrome	The disorder that arrives years before the tremor
2	Assessment	Why the usual diagnostic cues mislead here

SYNDROME	HOW IT APPEARS IN PARKINSON DISEASE	WHY IT IS MISSED
Depression	Common; less guilt and self-reproach than primary depression, more dysphoria and worry	Slowness and flat face are read as mood
Anxiety	Frequent, often locked to the wearing-off periods between doses	Attributed to the situation, not to the dose cycle
Apathy	**Loss of motivation and initiation without sadness or self-criticism**	**Recorded as depression, then treated as depression**
Psychosis	Visual hallucinations first, insight often retained early; delusions later	Patients conceal them unless asked directly
Impulse control disorders	**Gambling, hypersexuality, compulsive shopping and eating, punding**	**Never volunteered, and rarely asked about**
Dementia	Late, subcortical and executive, with prominent visuospatial difficulty	Confused with dementia with Lewy bodies

Amber marks the two that are missed most often and cost most. Apathy is treated as depression that will not respond, and impulse control disorders are found only by a direct question to the patient and the family.

- **The prodrome.** Rapid eye movement sleep behaviour disorder, in which the normal atonia of dreaming sleep is lost and the patient acts out dreams, can precede motor Parkinson disease by years. Depression, anxiety, constipation and reduced smell belong to the same premotor phase.
- **Why assessment is hard.** Masked facies, bradykinesia, stooped posture, soft monotonous voice, fatigue and disturbed sleep all imitate depression, so the diagnosis has to rest on reported mood, anhedonia, hopelessness and guilt rather than on what is observed across the desk.

WHAT EARNS THE MARKS

- **Apathy separated from depression.** It is the discrimination this question is set to test.
- **Impulse control disorders named,** with the fact that they must be asked about.
- **Sleep behaviour disorder given as premotor,** not as a late complication.
- **The overlap problem in assessment, stated explicitly.**

SEE ALSO Slot IV - 49 drug induced problems and their management Slot IV - 53 aetiology, course and management

SPRINT Day 35, Neuropsychiatry of systemic & neurological disease

IV - 48

Huntington's disease. [10]

10 MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **Genetics and pathology**	The repeat, the anticipation, the structure that atrophies
2 **The triad**	Movement, cognition, psychiatry, and which arrives first
4 **The psychiatric picture**	What it looks like, and where the risk sits
2 **Management**	Symptomatic only, plus the family and the testing question

GENETICS AND PATHOLOGY

Autosomal dominant, caused by an expanded CAG trinucleotide repeat in the huntingtin gene on chromosome 4. Forty repeats or more is fully penetrant, 36 to 39 carries reduced penetrance, and the intermediate range does not cause disease but is unstable when transmitted. Anticipation is greater through the father. The striatum atrophies, the caudate first, with loss of medium spiny neurons.

- **The triad.** Movement: chorea early, then dystonia, rigidity and bradykinesia as the disease advances. Cognition: a subcortical decline in speed, executive function and judgement, with insight often preserved painfully long. Psychiatry: frequently the first of the three to appear, sometimes by many years.
- **The psychiatric picture.** Depression is common and may precede the movement disorder. Suicide risk is markedly raised, with two peaks: around the time the diagnosis is made, and at the point independence is lost. Irritability and aggression cause most of the family burden. Apathy, obsessive and perseverative behaviour are common; psychosis is less so.
- **Management.** There is no treatment that changes the course. Chorea is treated with tetrabenazine or an antipsychotic when it is disabling. Depression, irritability and obsessive symptoms are treated conventionally and respond. Beyond drugs the work is genetic counselling, capacity, carer support and planning while the patient can still take part.

PREDICTIVE TESTING

Never a routine blood test. It follows a protocol with counselling before and after, is not performed on children or on anyone who cannot consent for themselves, and is never done at the request of a relative, an employer or an insurer. The result changes a life whether it is positive or negative.

WHAT EARNS THE MARKS

- **The repeat, the gene and the chromosome,** with the penetrance threshold.
- **Psychiatric onset placed before the movement disorder.**
- **The two suicide peaks.** Naming when the risk rises is worth more than saying risk is raised.
- **Predictive testing handled as an ethical procedure,** not as an investigation.

SEE ALSO Slot IV - 82 Wilson disease Slot IV - 47 psychiatric features of Parkinson disease **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease

IV - 49

Enumerate the neuropsychiatric manifestations of Parkinson's disease. Enumerate the drug induced psychological issues in Parkinson's disease. How will you manage them? [3+2+5]

3 + 2 + 5 | MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The manifestations	Enumerated by domain, not as one long list
2	Drug induced problems	Which drug produces which problem
4	Management	Subtract before adding, then the two safe agents
1	The standing rule	What must never be stopped abruptly

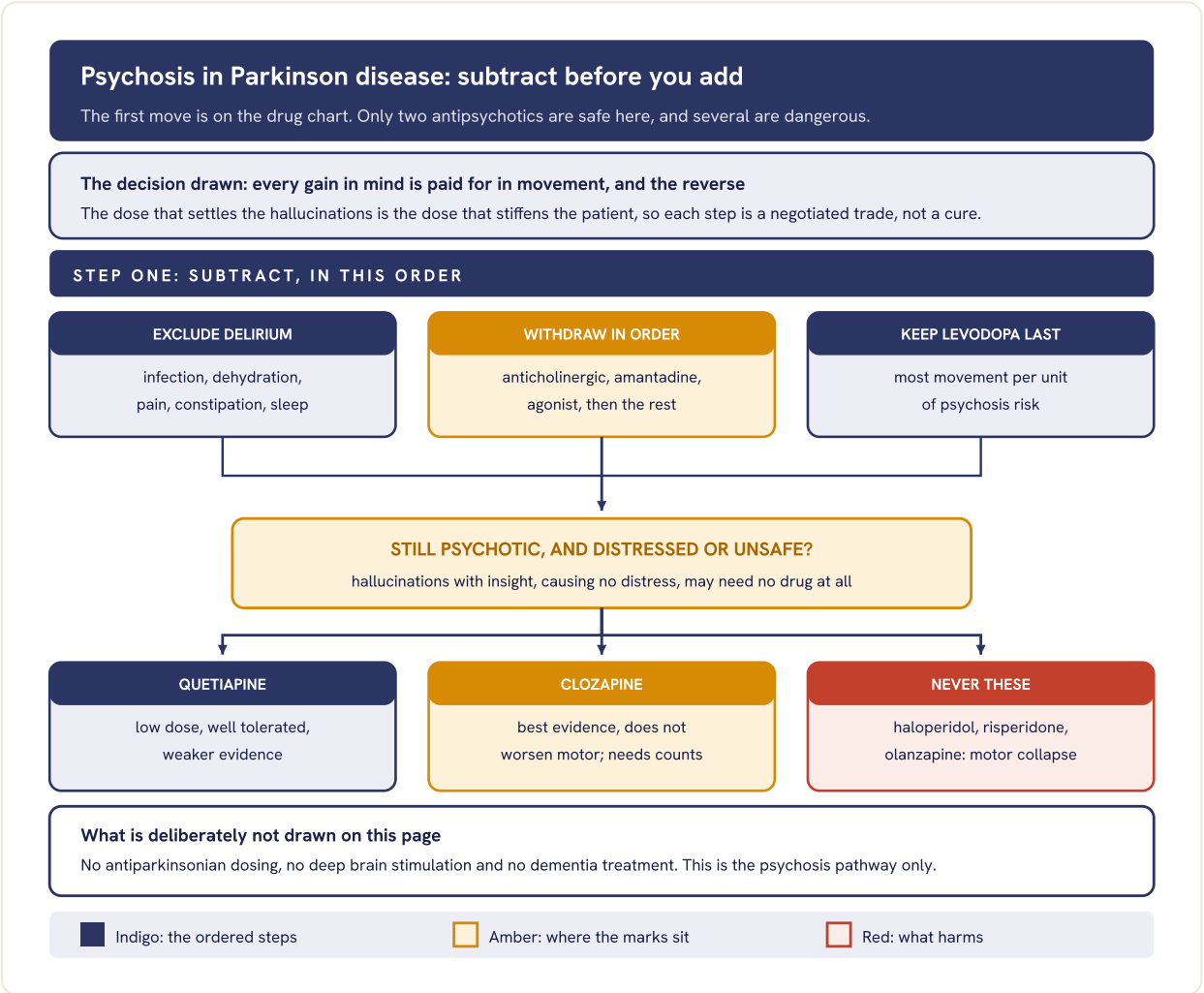


Fig 049.1 The withdrawal order that treats psychosis before any antipsychotic is added, the threshold for treating at all, and the two agents that are safe when it is crossed.

- **The manifestations, by domain.** Mood: depression, anxiety tied to wearing-off, apathy. Cognitive: executive and visuospatial decline, and dementia late. Perceptual: visual hallucinations and delusions. Sleep: rapid eye movement sleep behaviour disorder, insomnia, daytime somnolence. Behavioural: impulse control disorders and punding.

- **Which drug does what.** Dopamine agonists carry the highest risk of impulse control disorders, hallucinations and sudden sleep attacks. Levodopa causes psychosis, dyskinesia and, in excess use, dopamine dysregulation syndrome. Anticholinergics cause confusion and memory impairment, badly in the old. Amantadine causes hallucinations and confusion.
- **The standing rule.** Dopaminergic drugs are never stopped abruptly. Sudden withdrawal can precipitate a parkinsonism-hyperpyrexia syndrome that resembles neuroleptic malignant syndrome and can kill. Reduce stepwise, and watch the motor state at every step.

WHAT EARNS THE MARKS

- **The withdrawal order, written out.** It is the highest-value line on the page.
- **Delirium excluded first,** before any drug is blamed or added.
- **Clozapine and quetiapine named, and the dangerous agents named too.**
- **The rule against abrupt withdrawal,** with the syndrome it prevents.

SEE ALSO Slot IV - 47 psychiatric features of Parkinson disease Slot IV - 52 Parkinson disease and psychosis **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease

IV - 50

Write in detail about the Neuropsychiatric aspect of multiple sclerosis. [10]

10 | ASKED AT 15 MARKS | MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

RGUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 **Why they occur**	Lesion burden and disease context, both
4 **The syndromes**	Five, with the two that are specific to this disease
2 **Iatrogenic**	Steroids and the disease-modifying drugs
2 **Management**	What is treated, and how it is measured

SYNDROME	WHAT IT LOOKS LIKE	THE POINT TO MAKE
Depression	The commonest psychiatric diagnosis in multiple sclerosis	**Suicide risk is raised well above the general population**
Euphoria	Settled cheerful unconcern about real disability	Advanced disease, heavy frontal lesion load; not mania
Pseudobulbar affect	**Involuntary laughing or crying, detached from felt mood**	Distressing, often mistaken for lability of mood
Cognitive impairment	Processing speed first, then memory and executive function	**Occurs early, and tracks physical disability poorly**
Fatigue	The single commonest symptom of the disease	A symptom in its own right, not a depressive one

Amber marks the three that carry most of the marks: the suicide risk, the affect that is not a mood, and cognitive impairment that appears early and does not follow the walking stick.

- **Iatrogenic, and the rarer syndromes.** Psychosis and bipolar disorder are uncommon but more frequent than in the general population, and steroids and lesion site are considered first. High dose corticosteroids for relapse cause insomnia, elevated or irritable mood and occasionally psychosis, and they are the first thing to ask about in a patient with sudden mood change. Interferon beta has historically been linked with depression, though the association is debated; either way, depression here is treated rather than the treatment abandoned.
- **Management.** Depression responds to antidepressants and to psychological therapy. Pseudobulbar affect responds to antidepressants at doses lower than depression needs. Fatigue is treated as fatigue: exercise, sleep and heat avoidance.

PITFALL

Fatigue, slowed thinking, poor concentration and disturbed sleep all belong to multiple sclerosis itself. A depression scale weighted towards somatic items will therefore overdiagnose, so the diagnosis is made on mood, anhedonia, hopelessness and guilt instead.

WHAT EARNS THE MARKS

- **Euphoria and pseudobulbar affect distinguished from mania and from lability.**
- **Cognitive impairment placed early,** and uncoupled from physical disability.
- **Steroids named as a cause of acute mood change.**

SEE ALSO Slot IV - 51 aetiology, features and management of multiple sclerosis

Slot IV - 47 psychiatric features of Parkinson disease SPRINT Day 35, Neuropsychiatry of systemic & neurological disease

IV - 51

Etiology, clinical features and management of Multiple Sclerosis. [10]

10 MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

KUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Aetiology	Immune, genetic, environmental, in one line each
3 Clinical features	The patterns, the presentations, and the diagnostic rule
4 Management	Relapse, disease course, symptoms: three separate jobs
1 The psychiatric arm	Where a psychiatrist actually joins the team

AETIOLOGY AND THE DIAGNOSTIC RULE

An immune mediated demyelinating disease of the central nervous system. Susceptibility is genetic, with the HLA-DRB1 region carrying most of it, and environmental: low vitamin D, Epstein-Barr virus, smoking. Diagnosis rests on events disseminated in space and in time, with imaging and oligoclonal bands.

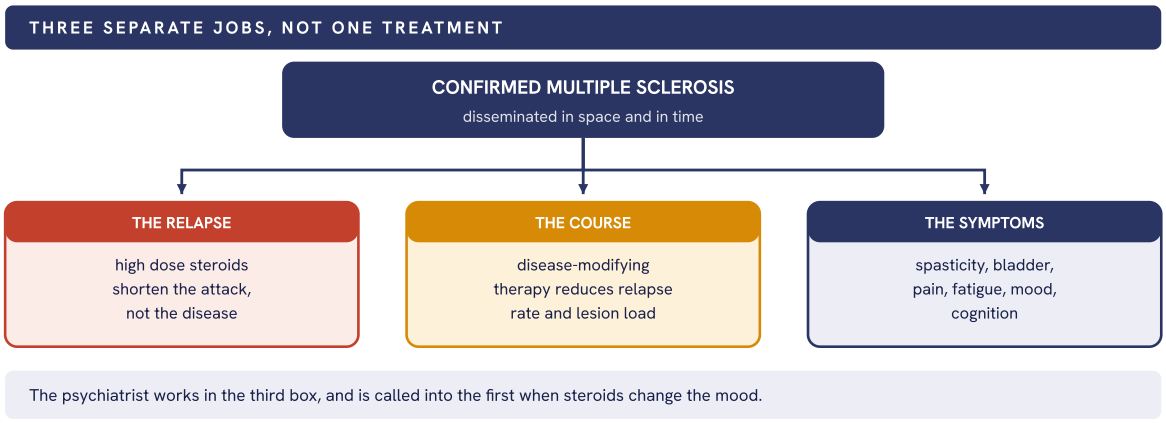


Fig 051.1 Management separated into the three jobs it actually is.

- **Patterns and presentations.** Relapsing-remitting is commonest at onset; many later become secondary progressive, and a minority are primary progressive. Presentations include optic neuritis, internuclear ophthalmoplegia, ataxia and bladder urgency.
- **Where the psychiatrist joins.** Depression, cognitive impairment, pseudobulbar affect, fatigue, and steroid induced mood change. Adjustment to a relapsing illness in a young adult, with its effect on work and family, is treatment rather than a side conversation.

WHAT EARNS THE MARKS

- **Dissemination in space and time,** stated as the diagnostic rule rather than implied.
- **The three jobs kept separate.** Steroids treat the relapse and not the disease, and saying so is the point.
- **The psychiatric contribution named,** which is what makes this a psychiatry answer.

SEE ALSO Slot IV - 50 neuropsychiatric aspects of multiple sclerosis Slot IV - 87 neuropsychiatric manifestations of HIV

SPRINT Day 35, Neuropsychiatry of systemic & neurological disease

IV - 52

a) Relationship between Parkinsons and psychosis. b) Write factors which decide assessment and management of depression in a patient with stroke. [5+5]

5 + 5 | MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Parkinson and psychosis**	A relationship that runs in three directions
2	**What follows from it**	The two safe agents, and the ones that harm
3	**Depression after stroke**	The factors that decide the assessment
2	**Managing it**	What is treated, when, and alongside what

DIRECTION	WHAT HAPPENS	WHAT IT MEANS IN PRACTICE
Disease causes psychosis	Visual hallucinations and delusions arise from the illness itself, more with dementia and long duration	**Not simply a drug side effect**
Treatment causes psychosis	All dopaminergic drugs can, agonists most of all	Review the chart before adding anything
Antipsychotics cause parkinsonism	Dopamine blockade produces the motor syndrome, and unmasks or worsens the disease	**The relationship runs both ways**

Amber marks the two halves that make this a relationship, not a side effect. The safe agents are quetiapine and clozapine; haloperidol, risperidone and olanzapine harm.

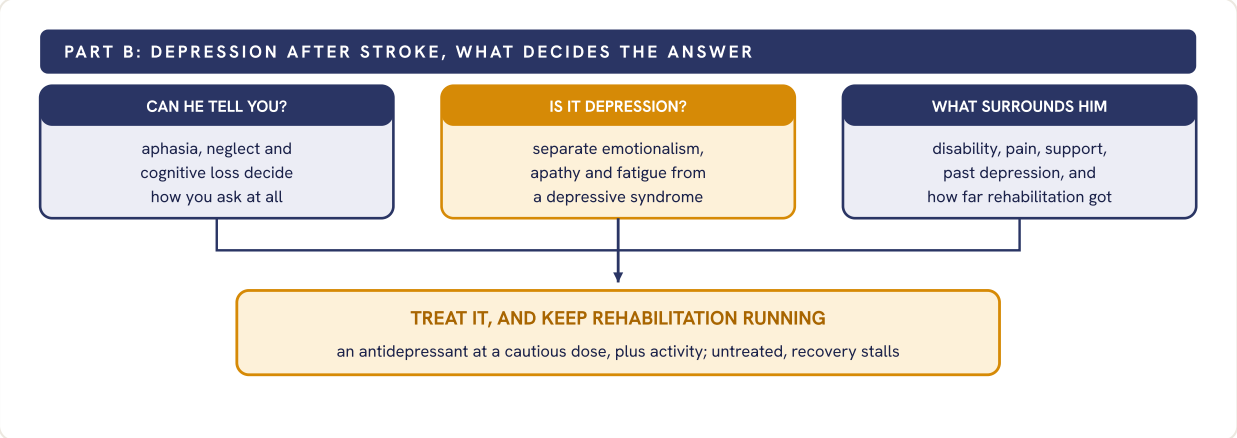


Fig 052.1 What decides the assessment, and treatment that runs alongside rehabilitation.

- **The two traps.** Emotionalism after stroke, sudden crying with little felt sadness, is not depression. And bleak realism about disability is not automatically illness: the diagnosis rests on anhedonia and hopelessness.

WHAT EARNS THE MARKS

- **Part (a) answered as a two-way relationship,** not as a list of drug side effects.
- **Communication named as the first assessment factor.** Aphasia changes the method.
- **Depression tied to rehabilitation outcome,** which is why it is treated promptly.

SEE ALSO Slot IV - 49 drug induced problems in Parkinson disease Slot IV - 41 cerebral small vessel disease **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 53

Describe the aetiology, neuropathology, clinical manifestations, course, prognosis and management of Parkinson's disease. [10]

10 | ASKED AT 20 MARKS | MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

NTRUHS, RGUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2	**Aetiology and pathology**	What is lost, what accumulates, and what is inherited
3	**Clinical manifestations**	Motor and non-motor, both named
2	**Course and prognosis**	The staging, and what predicts a faster decline
3	**Management**	Motor, non-motor and surgical, kept apart

AETIOLOGY AND NEUROPATHOLOGY

Degeneration of dopaminergic neurons in the substantia nigra pars compacta, with Lewy bodies containing aggregated alpha-synuclein. Motor signs appear once much of the nigrostriatal projection is already lost. Mostly sporadic, with age the dominant risk factor; a minority are familial.

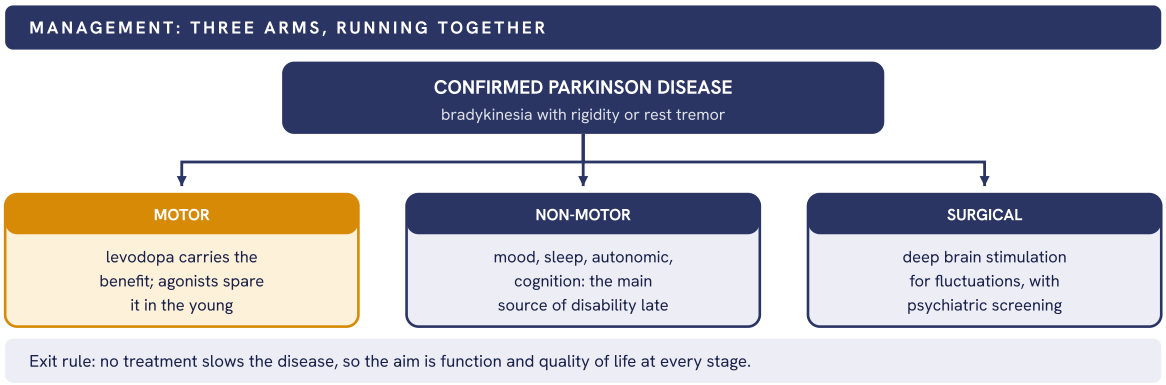


Fig 053.1 The three arms of management, with the non-motor arm marked as the main source of disability once the disease is established.

- **Manifestations.** Motor: bradykinesia is required, with rest tremor, rigidity and later postural instability; onset is asymmetrical. Non-motor: hyposmia, constipation, sleep behaviour disorder, depression and anxiety, often years earlier.
- **Course and prognosis.** Progressive, staged by the Hoehn and Yahr scale from unilateral disease to wheelchair dependence. Poorer with older onset, a rigid-akinetic picture, early falls, early cognitive impairment and poor levodopa response. The last two also warn the diagnosis may not be Parkinson disease.

WHAT EARNS THE MARKS

- **Alpha-synuclein and the Lewy body named,** with the site of degeneration.
- **Non-motor features given equal weight,** and placed before onset as well as after.
- **Prognostic markers listed,** with the two that should raise doubt about the diagnosis.

SEE ALSO Slot IV - 47 psychiatric features of Parkinson disease Slot IV - 49 drug induced problems **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease

AREA 07 OF 15 · P4-A05

Endocrine and metabolic psychiatry

6	6.2%	IV-54	IV-59
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 HIGH 2 SINGLE 3

REVISION ORDER

The three thyroid slots together, they are one answer at three widths and the lithium rule is stated identically on all three. Metabolic syndrome next, it is the one that carries the thresholds and they have to be exact. Diabetes and obesity last, and they share a behavioural spine.

WHAT IS IN THIS AREA

The thyroid in three slots: the psychiatric manifestations of thyroid disorders, hypothyroidism on its own, and a slot that adds the parathyroid axis. Then the metabolic limb, metabolic syndrome set against the second-generation antipsychotics with the first-generation comparison, obesity with the strategies for managing it, and the psychological and psychiatric aspects of diabetes mellitus.

IV - 54

Psychiatric manifestations of Thyroid disorders. [10]

10 ENDOCRINE AND METABOLIC PSYCHIATRY

KUHS, NTRUHS, RGUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 The principle	Thyroid state mimics, worsens and modifies psychiatric illness
3 Underactive	Depression, slowing, cognition, and the psychotic extreme
3 Overactive	Anxiety and mania, and the misleading elderly form
2 What to do	Who to test, what to correct, and the lithium rule

THYROID STATE	PSYCHIATRIC PICTURE	THE PRESENTATION THAT MISLEADS	TEST
Underactive	Depression, apathy, slowing, poor concentration, hypersomnia	A reversible dementia-like picture in an older patient	**TSH raised, free T4 low**
Severely underactive	Paranoid and hallucinatory psychosis, the picture called myxoedema madness	Read as late-onset schizophrenia	Same, with a long history
Overactive	Anxiety, irritability, insomnia, lability, distractibility	Diagnosed as panic disorder or as mania	**TSH suppressed, free T4 raised**
Overactive in old age	Apathy, low mood, weight loss, withdrawal	**Apathetic thyrotoxicosis, read as depression**	Check the pulse rhythm
Subclinical underactive	Low mood, and a poorer antidepressant response	Dismissed because free T4 is normal	TSH raised, free T4 normal

Amber cells are the discriminators. Each row is a presentation that reaches psychiatry before it reaches endocrinology.

- **Who to test.** First-episode psychosis, treatment-resistant depression, rapid cycling, new cognitive impairment, anxiety with prominent somatic features, and any postpartum presentation.
- **The lithium rule.** Lithium causes hypothyroidism and goitre, so check thyroid function at baseline and periodically. Finding it is a reason to add thyroxine, not a reason to stop the lithium.

PITFALL

Correcting the thyroid state and stopping there. Symptoms improve with replacement but often lag behind the biochemistry, so the psychiatric illness still needs treating and reviewing on its own timetable.

WHAT EARNS THE MARKS

- **Both directions answered,** with equal weight; most answers give hypothyroid depression and stop.
- **Apathetic thyrotoxicosis named,** which is the discriminating fact on this page.
- **A testing rule, not a testing list.** Naming who to screen shows clinical judgment.

SEE ALSO Slot IV - 56 hypothyroidism Slot IV - 59 thyroid and parathyroid dysfunction SPRINT Day 33, Endocrine & metabolic psychiatry

IV - 55

Mental health strategies for managing Obesity. [10]

10 ENDOCRINE AND METABOLIC PSYCHIATRY KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Frame the problem	The link runs both ways, and is often iatrogenic
3	Assess three things	The drug chart, the eating pattern, the measurements
3	Psychological strategies	Matched to what the assessment actually found
2	Prescribing and beyond	Switch, augment, refer, and monitor on a schedule

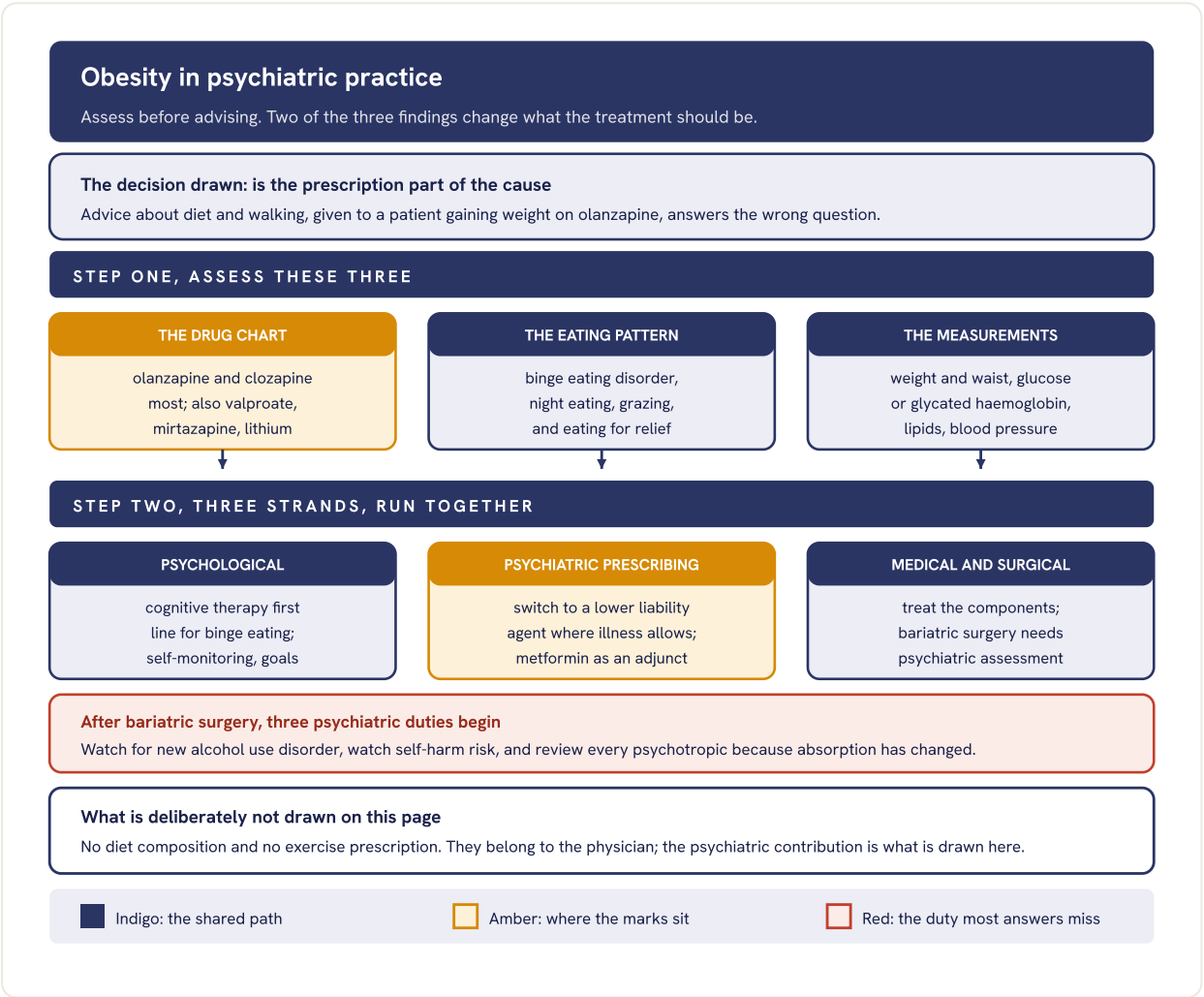


Fig 055.1 Three things assessed before anything is advised, then three strands of management run together, with the post-surgical duties carried as a red strip because they fall to psychiatry specifically.

- **Frame it first.** The link runs both ways: depression raises the risk of obesity and obesity raises the risk of depression, and in psychiatric patients the treatment is frequently part of the cause.
- **Weight stigma is a clinical variable.** Shame-based advice reduces attendance and worsens eating, so language and framing belong in the treatment plan rather than beside it.

WHAT EARNS THE MARKS

- **The drug chart reviewed first,** which is the single most psychiatric move on the page.
- **Binge eating disorder screened for and treated,** with cognitive therapy named as first line.
- **A monitoring schedule, not just a list of tests.**
- **The post-surgical duties,** which almost no answer reaches.

SEE ALSO Slot IV - 57 metabolic syndrome and antipsychotics Slot IV - 58 psychiatric aspects of diabetes **SPRINT** Day 33, Endocrine & metabolic psychiatry

IV - 56

Hypothyroidism. [10]

10 ENDOCRINE AND METABOLIC PSYCHIATRY KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition and levels	Primary, central, and the subclinical band
3	Causes	Autoimmune, iodine, iatrogenic, and the psychiatric drug
3	Features and diagnosis	Body and mind, then the order the tests are read in
2	Treatment	Replace, retest, and the rule about lithium

THE DEFINITION, IN THREE LEVELS

Deficient thyroid hormone action. Primary disease is failure of the gland, with a raised thyroid stimulating hormone and a low free T4. Central disease is pituitary or hypothalamic. Subclinical disease is a raised stimulating hormone with a normal free T4.

CAUSE GROUP	EXAMPLES	THE CLUE	NOTE
Autoimmune	Hashimoto thyroiditis	Goitre, other autoimmune disease	**Commonest where iodine is sufficient**
Iodine deficiency	Endemic goitre	Geography and diet	Still relevant in parts of India
Iatrogenic	Thyroidectomy, radioiodine, amiodarone, interferon	The history gives it away	Ask before testing
Psychotropic	Lithium	Rising stimulating hormone on treatment	**Treat, do not stop the lithium**
Transient	Postpartum thyroiditis	Months after delivery, often after a thyrotoxic phase	**Mimics postnatal depression**

Amber cells are the discriminators. Two of them are the reason this endocrine question sits in a psychiatry paper.

- **Features and testing.** Fatigue, cold intolerance, weight gain, constipation, bradycardia, slow-relaxing reflexes; mentally, depression and slowing. Test the stimulating hormone, then free T4.
- **Treatment.** Levothyroxine once daily on an empty stomach, apart from calcium, iron and antacids, retested after about six weeks and titrated to the stimulating hormone. Start low where the patient is old or has cardiac disease.

PITFALL

Myxoedema coma. Hypothermia, hyponatraemia, hypoventilation and a falling conscious level in a patient whose slowness was being treated as depression.

WHAT EARNS THE MARKS

- **Three levels defined,** subclinical disease included; it is where most psychiatric overlap sits.
- **Lithium named as a cause,** with the rule that replacement is added rather than lithium stopped.
- **Test order given,** stimulating hormone first, which shows the biochemistry is understood.

SEE ALSO Slot IV - 54 psychiatric manifestations of thyroid disorders Slot IV - 59 thyroid and parathyroid dysfunction

SPRINT Day 33, Endocrine & metabolic psychiatry

IV - 57

What is metabolic syndrome? Discuss the emergence of metabolic syndrome due to second-generation antipsychotics as compared with first-generation antipsychotics. [3+7]

3 + 7 | ENDOCRINE AND METABOLIC PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Define it	Five components, three needed, and the Asian waist
3 The comparison	The answer is which drug, not which generation
2 Mechanism	The receptors that drive appetite and insulin
2 What follows	A monitoring schedule, then what to change

THE DEFINITION

Three of five: central obesity by waist, triglycerides 150 or more, high density lipoprotein under 40 in men or 50 in women, blood pressure 130 over 85 or more, fasting glucose 100 or more milligrams per decilitre, or treatment for any. South Asian waist is lower: 90 centimetres in men, 80 in women.

AGENT	METABOLIC LIABILITY	EVIDENCE AND COMMENT	GENERATION
Clozapine, olanzapine	Highest	Largest weight and lipid change in the major pragmatic trial	**Second**
Chlorpromazine, thioridazine	Intermediate to high	Low potency older drugs also cause substantial gain	**First**
Haloperidol, fluphenazine	Lower	The comparison that produced the class story	First
Aripiprazole, ziprasidone, lurasidone	Lowest	The usual switch targets when weight is the problem	**Second**

Amber cells are the discriminators. Rows three and five break the generation story, and saying so is the discussion the seven marks are for.

- **Mechanism.** Histamine H1 and serotonin 5-HT2C blockade drive appetite and weight, muscarinic M3 blockade impairs insulin secretion, and there are direct effects on insulin sensitivity.

STATUS: WHERE THIS SITS TODAY

Settled: liability is drug-specific, clozapine and olanzapine are the worst, and monitoring at baseline, three months and then yearly is standard. Still argued: how much of the excess is illness and lifestyle rather than drug, and where the newer weight-lowering agents belong.

WHAT EARNS THE MARKS

- **Three of five stated,** with the lower South Asian waist threshold named.
- **The generation framing challenged,** using a low potency older drug as the counterexample.
- **Receptor mechanism given,** histamine and serotonin for appetite, muscarinic for insulin.

SEE ALSO Slot IV - 55 mental health strategies for obesity Slot IV - 58 psychiatric aspects of diabetes **SPRINT** Day 33, Endocrine & metabolic psychiatry

IV - 58

Discuss the psychological and psychiatric aspects in the management of diabetes mellitus. [10]

10 ENDOCRINE AND METABOLIC PSYCHIATRY

KUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	**The two-way link**	Each raises the other, and control pays for it
3	**What to screen for**	Four things, and two of them are not depression
3	**Treatment**	Drug choices that respect weight and glucose
2	**The model of care**	Collaborative care, education, and the family

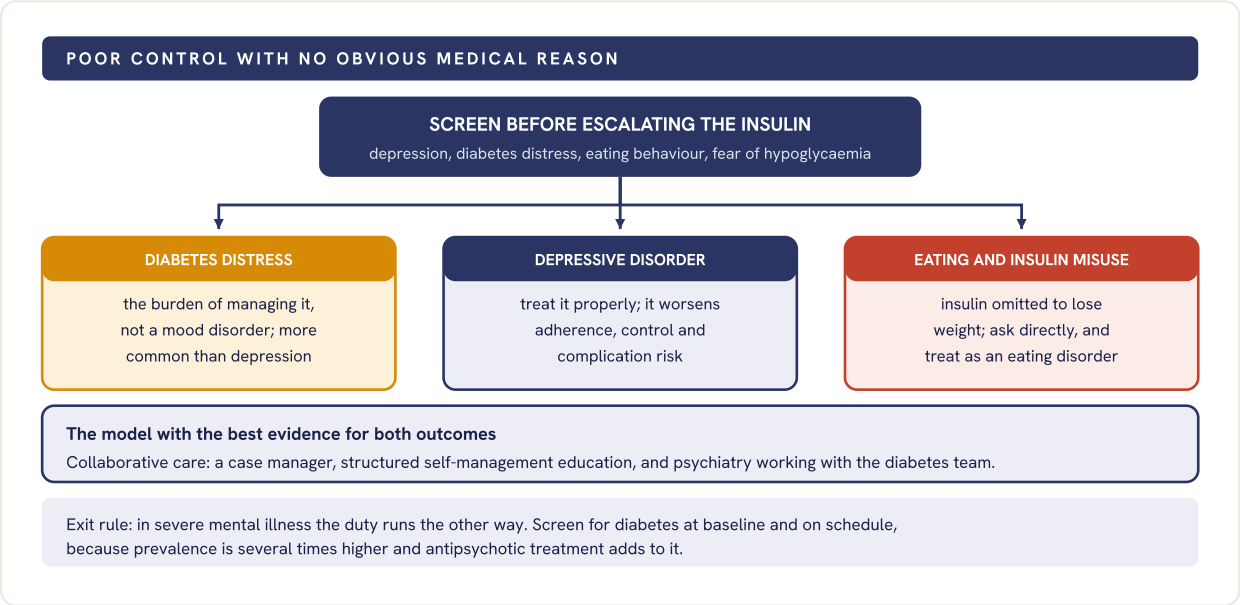


Fig 058.1 Screening placed before any escalation of the diabetic treatment, with distress separated from depression because they need different interventions and only one of them needs a drug.

- **Drug choices.** Selective serotonin reuptake inhibitors are preferred, sertraline commonly. Avoid the weight-promoting options where possible, and consider duloxetine where painful neuropathy coexists.
- **The interactions to know.** Serotonergic antidepressants can lower glucose further, olanzapine and clozapine worsen control, and steroids given for anything else will undo a month of adjustment.

PITFALL

Treating distress as depression. Distress responds to education, problem solving and a change in the regimen, and an antidepressant given instead treats nothing.

WHAT EARNS THE MARKS

- **Distress named and separated from depression.** It is the discriminating fact on this page.
- **Insulin omission asked about directly,** because it is dangerous and rarely volunteered.
- **Collaborative care named as the model,** not simply liaison in general terms.

SEE ALSO Slot IV - 57 metabolic syndrome and antipsychotics Slot IV - 55 mental health strategies for obesity

SPRINT Day 33, Endocrine & metabolic psychiatry

IV - 59

Discuss the psychiatric disorders due to thyroid and parathyroid glands dysfunction. [10]

10 ENDOCRINE AND METABOLIC PSYCHIATRY

KUHS, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	The organising rule	Each gland acts through one measurable number
3	Thyroid, compactly	Both directions, and the form that misleads in old age
3	Parathyroid and calcium	High and low, with the signs that give each away
2	What to do	Who to test, and how symptoms follow correction

THE ORGANISING RULE

Both glands reach the brain through a single number that a routine test reports: the thyroid stimulating hormone, and serum calcium. So the answer is structured as two axes, each with an over and an under state, and each with a psychiatric picture that tracks the level rather than the gland.

STATE	PSYCHIATRIC PICTURE	THE PHYSICAL GIVEAWAY	TEST
Thyroid, under	Depression, slowing, cognitive impairment; psychosis when severe	Cold intolerance, slow reflexes, bradycardia	Stimulating hormone high
Thyroid, over	Anxiety, lability, insomnia; apathy and low mood in the elderly	Tremor, weight loss, irregular pulse	**Stimulating hormone suppressed**
Calcium, high	Depression, apathy, poor concentration; delirium and psychosis as it rises	Thirst, polyuria, constipation, stones, bone pain	**Calcium and parathyroid hormone**
Calcium, low	Anxiety, irritability, lability; psychosis and seizures when severe	Tetany, and the Chvostek and Trousseau signs	**Calcium low, magnesium checked**
Calcium, low and long	Parkinsonism with psychiatric features	Basal ganglia calcification on imaging	Imaging, not biochemistry alone

Amber cells are the discriminators. The calcium rows are what this stem is really testing; the thyroid rows are the setting for them.

- **Two causes to name.** Primary hyperparathyroidism is most often a single adenoma, and lithium raises parathyroid hormone and calcium as well as causing hypothyroidism. Hypoparathyroidism is most often a consequence of thyroid surgery.
- **Who to test.** Unexplained depression with thirst and constipation, any delirium, new psychosis in an older patient, unexplained seizures, and every patient on long-term lithium.

WHAT EARNS THE MARKS

- **Both glands answered,** with the parathyroid given real space; most answers write only about the thyroid.
- **Symptoms tied to the level,** so severity explains why the same gland gives depression in one patient and delirium in another.
- **Lithium named on the calcium axis as well as the thyroid axis.**
- **The lag after correction,** which stops the answer promising instant recovery.

SEE ALSO Slot IV - 54 psychiatric manifestations of thyroid disorders Slot IV - 56 hypothyroidism **SPRINT** Day 33, Endocrine & metabolic psychiatry

AREA 08 OF 15 · P4-A13

Recent advances and landmark studies

6	5.9%	IV-60	IV-65
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 5

REVISION ORDER

The critique slot first: it is the shape the others take and the trials it names carry into three of them. Genetics and the post-traumatic stress slot next, both are advances questions with named studies behind them. The bipolar slot after that, because half of it is classification. Rehabilitation and catatonia are independent, and both cross-link into other areas of this book.

WHAT IS IN THIS AREA

Recent advances asked five different ways: in psychological rehabilitation, in psychiatric genetics with the models of genetic study, in the diagnosis of post-traumatic stress disorder, in the treatment of bipolar affective disorder with the classification first, and as a critique of whether therapeutics have improved prognosis at all. The sixth slot asks for the current status of catatonia in DSM-5 and ICD-11 with its neurobiology and its drug treatment, which is a clinical question the boards set inside this area and is answered on its page as it was set.

IV - 60

Describe the recent advances in psychological rehabilitation in persons with major mental illness. [10]

10 RECENT ADVANCES AND LANDMARK STUDIES

DNB, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What changed	From occupying time to restoring a role
5	The advances	Four, each with the evidence behind it
2	Where they stand	Available here, or still a trial finding
1	What is unsolved	One sentence, honest

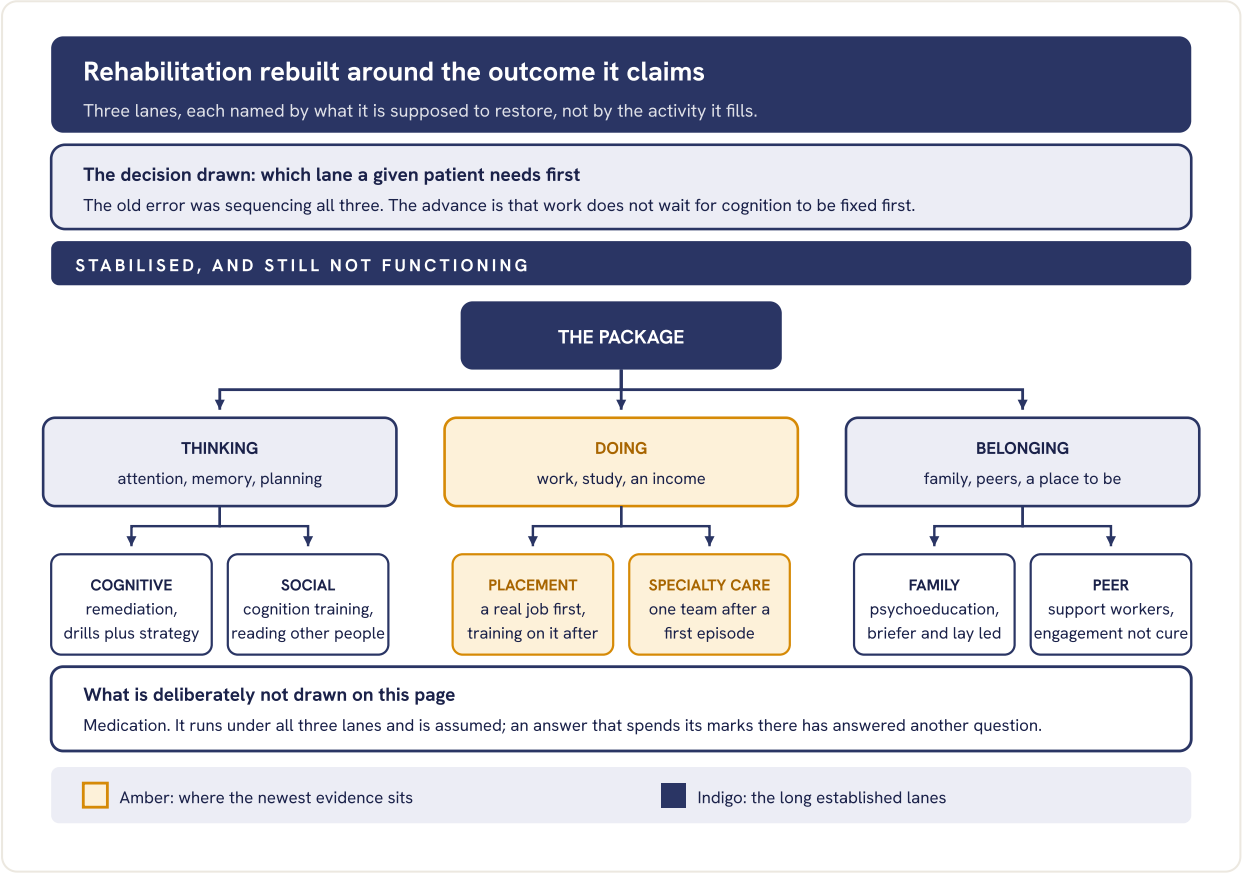


Fig 060.1 The package sorted by the outcome each lane claims, which is what makes place-then-train visible as a change of principle rather than a change of timetable.

ADVANCE	WHAT IT DOES	EVIDENCE SO FAR	STATUS
Cognitive remediation	Drills and strategies for attention, memory and planning	**Meta-analyses show durable cognitive gain, and functional gain when paired with rehabilitation**	In use where staffed
Individual placement and support	A competitive job first, training on the job after	**Randomised trials favour it over train-then-place services**	Established, thinly available
Coordinated specialty care	One team for drugs, family, work and study after a first episode	The RAISE trial improved quality of life and engagement against usual care	Few Indian sites
Task shifted community care	Lay health workers deliver the package at home	The COPSI trial found modest benefit, larger at rural sites	Trial stage here

STATUS: AVAILABLE, AND NOT AVAILABLE

Cognitive remediation and family work need only trained staff, and they travel. Supported employment and coordinated specialty care need an employer network and a funded team, so they stay in a few centres. What is unsolved: none of these has been shown to change long term recovery, only functioning while the programme runs.

WHAT EARNS THE MARKS

- **Place-then-train named as a reversal,** the clearest advance on the page.
- **Each advance tied to its evidence,** not a list of programme names.
- **An Indian trial named,** which almost no answer offers.
- **The unsolved line,** separating functioning from recovery.

SEE ALSO Slot IV - 64 have advances improved prognosis Slot IV - 02 principles of rehabilitation **SPRINT** Day 39, Community psychiatry and public health

IV - 61

Describe the different models of genetic study in psychiatry. Discuss the recent advances in psychiatric genetics. [5+5]

5 + 5 | RECENT ADVANCES AND LANDMARK STUDIES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The classical models	Family, twin, adoption, and what each isolates
3	The molecular models	Linkage, candidate gene, association, sequencing
3	What has advanced	Loci, structural variants, cross-disorder overlap
2	What has not	Prediction for one patient, and why

- **The models, and what each isolates.** Family studies give recurrence risk in relatives but cannot separate genes from a shared home. Twin studies compare identical with fraternal concordance and yield heritability. Adoption studies separate rearing from inheritance. Linkage tracks co-segregation within families and finds large effect loci. Candidate gene association tested single guesses and mostly failed to replicate. Genome-wide association tests millions of common variants and needs enormous samples. Sequencing finds the rare coding variants the others miss.

ADVANCE	WHAT IT DOES	EVIDENCE SO FAR	STATUS
Genome-wide association	Common variants of tiny individual effect	Hundreds of loci in schizophrenia; the strongest signal sits in the histocompatibility region	Established research finding
Rare and structural variants	Large deletions and rare coding variants	**The 22q11.2 deletion carries a large risk; sequencing converges on synaptic and chromatin genes**	Useful in syndromic cases
Cross-disorder overlap	Shared variance across diagnoses	Substantial genetic correlation between schizophrenia, bipolar disorder and depression	Changing how boundaries are read
Pharmacogenomics	Metabolism and hypersensitivity typing	**CYP2D6 and CYP2C19 status guides dose; HLA-B 15:02 typing before carbamazepine**	The one advance in routine use

STATUS: THE GAP BETWEEN DISCOVERY AND THE CLINIC

A polygenic risk score sums thousands of variants and predicts well across a population while telling an individual patient almost nothing, so it stays a research instrument. The findings that have reached practice are not the loci but the pharmacogenomic ones, where a single typed variant changes a dose or prevents a severe drug reaction.

WHAT EARNS THE MARKS

- **Each classical model paired with what it isolates,** not simply named.
- **The candidate gene era described as a failure,** which is the honest history.
- **Population prediction separated from individual prediction,** the line that carries the last two marks.

SEE ALSO Slot IV - 63 bipolar disorder and recent advances SPRINT Day 38, Recent advances and landmark studies

IV - 62

Discuss the recent advances and the future of the diagnosis of post-traumatic stress disorder. [5+5]

5 + 5 | RECENT ADVANCES AND LANDMARK STUDIES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Where the diagnosis moved	Out of anxiety, into trauma and stressor related
3	The two systems now	Four clusters against three, and complex PTSD
3	Measurement	The reference interview, and the new questionnaire
2	What the future rests on	Markers, and why none of them diagnoses yet

- **The two systems have diverged, not converged.** DSM-5 moved the disorder out of the anxiety disorders and widened it: four clusters, being intrusion, avoidance, negative alterations in cognition and mood, and altered arousal, plus a dissociative subtype and a separate preschool form. ICD-11 narrowed it instead to three: re-experiencing in the here and now, avoidance, and a persistent sense of current threat, and added complex PTSD alongside. Applied to the same sample the two produce different, overlapping populations.

ADVANCE	WHAT IT DOES	EVIDENCE SO FAR	STATUS
DSM-5 restructuring	Four clusters, with dissociative and preschool subtypes	Factor analytic work supports more than the original three clusters	In routine use
ICD-11 narrowing, with complex PTSD	Three core clusters, and a separate complex form carrying disturbances of self-organisation	**Latent class studies separate the two forms rather than grading one**	In routine use
The International Trauma Questionnaire	Measures the ICD-11 construct, both forms	Validated across many languages and settings	In routine use
Biological markers	Hippocampal volume, cortisol response, startle, methylation	**Group differences only, and no useful accuracy in an individual**	Research only

STATUS: WHAT THE FUTURE ACTUALLY RESTS ON

There is no validated diagnostic biomarker for this or for any other primary psychiatric disorder, and the candidates listed above have been studied for decades without becoming one. The realistic near future is better structured assessment: a clinician administered interview as the reference standard, dimensional measurement alongside the category, and repeated sampling after a trauma rather than a single cross-sectional judgement.

WHAT EARNS THE MARKS

- **The divergence stated as a divergence.** Most answers describe DSM-5 and assume ICD-11 followed it.
- **Complex PTSD placed in the right system,** with its distinguishing features named.
- **Markers reported as group findings,** which is the difference between a current answer and a hopeful one.

SEE ALSO Slot IV - 65 catatonia in the two systems **SPRINT** Day 38, Recent advances and landmark studies

IV - 63

Classify bipolar affective disorder and discuss the recent advances in its treatment. [5+5]

5 + 5 | RECENT ADVANCES AND LANDMARK STUDIES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The classification	Types and specifiers, in one frame
3	Where the systems differ	The mixed episode, category against specifier
3	Advances in treatment	The agents that changed the depressed phase
2	What the trials removed	The antidepressant question, answered

- **The classification.** Both systems recognise bipolar type I, bipolar type II and cyclothymic disorder, with residual other specified and unspecified categories. Episodes are then described by pole, by the presence of psychotic symptoms, and by course modifiers such as rapid cycling and seasonal pattern.
- **Where they differ.** DSM-5 abolished the mixed episode and replaced it with a mixed features specifier that attaches to a manic, hypomanic or depressive episode. ICD-11 kept the mixed episode as a category. The DSM route makes mixed depression visible in a way the older criteria did not.

ADVANCE	WHAT IT DOES	EVIDENCE SO FAR	STATUS
Cariprazine	D3 preferring partial agonist, licensed across the phases	Randomised trials in acute mania and in bipolar depression	Marketed in India
Lumateperone	Newer agent aimed at the depressed phase	Randomised placebo controlled trials, alone and as an adjunct	Approved abroad, access limited here
Antidepressants reconsidered	An antidepressant added to a mood stabiliser	**The STEP-BD programme found no advantage over placebo added to the stabiliser**	Practice already changed
Psychoeducation and rhythm therapy	Structured relapse prevention, and stabilised daily routine	**Randomised trials show fewer relapses across follow up**	In use where staffed

STATUS: WHERE THE REAL CHANGE HAPPENED

The depressed phase is where nearly all recent movement sits, because it carries most of the disability and had the fewest licensed options. Lithium and the older agents remain the backbone of maintenance. What is unsolved is prevention of the depressive relapse, which every maintenance trial finds harder than mania.

WHAT EARNS THE MARKS

- **The mixed presentation handled as the point of difference,** not as a footnote to the list of types.
- **Each new agent tied to the phase it treats,** since none of them is a general advance.
- **A named trial for the negative finding.** Saying antidepressants are unhelpful without it is an opinion.
- **The unsolved line:** depressive relapse, which the status strip names.

SEE ALSO Slot IV - 74 lithium in youths with bipolar disorder Slot IV - 73 long acting injections in mood disorders

SPRINT Day 38, Recent advances and landmark studies

IV - 64

Have the advances in therapeutics over the last few decades improved the prognosis of mental disorders? Write a critique. [10]

10 | RECENT ADVANCES AND LANDMARK STUDIES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The question restated	Prognosis of what, measured how
4	Domain by domain	Where it improved, and where it did not
2	Why the record looks better than it is	Publication bias and the outcomes chosen
2	The verdict	What actually changed outcome, and it was rarely a molecule

- **Restate before answering.** Prognosis is not one measurement. Symptom response, relapse, social function and death have moved in different directions, and an answer that treats them as one cannot be right about any of them.
- **Why the published record reads better than the clinic.** Analysis of the drug regulator's own trial database showed that positive antidepressant trials were published and negative ones largely were not, so the apparent effect in the literature exceeds the effect in the submitted data.

DOMAIN	WHAT CHANGED	THE EVIDENCE	VERDICT
Acute symptom control	Reliable control of mania, psychosis and catatonia	Decades of trials; ECT and clozapine remain the most effective treatments in psychiatry	Genuinely improved
Newer against older drugs	Second generation antipsychotics	**CATIE and CUtLASS found no advantage on effectiveness or on quality of life**	No gain
Long term function	Employment and social recovery in schizophrenia	Cohort comparisons across decades show little change	Largely unchanged
Physical mortality	Life expectancy in severe mental illness	**One to two decades of life lost, mostly cardiovascular, and the gap has not closed**	The strongest counter-argument

STATUS: WHAT DID MOVE THE PROGNOSIS

Four things, and only one of them is new. Clozapine in resistant schizophrenia, including its effect on suicidal behaviour. Lithium in bipolar disorder, including its anti-suicidal signal. Electroconvulsive therapy in severe and refractory illness. And the reorganisation of services: early intervention after a first episode, and the move out of long stay institutions. The largest gains came from how care was arranged rather than from what was prescribed.

WHAT EARNS THE MARKS

- **Prognosis broken into separate outcomes** in the first two lines.
- **Two named negative trials.** A critique without them is an assertion.
- **The mortality gap raised,** which is the fact that decides the question and is the one most answers omit.
- **A verdict that credits service organisation,** not a closing sentence about the future being bright.

SEE ALSO Slot IV - 60 advances in psychological rehabilitation Slot IV - 75 me too drugs and new agents SPRINT
Day 38, Recent advances and landmark studies

IV - 65

What is the current status of catatonia in DSM-5 and ICD-11? Discuss briefly its neurobiology and the pharmacological options for treatment. [3+3+4]

3 + 3 + 4 | RECENT ADVANCES AND LANDMARK STUDIES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Where it sits now	A DSM-5 specifier against an ICD-11 grouping
3	Neurobiology	GABA, glutamate, dopamine, and the motor circuit
4	Treatment	Lorazepam, then ECT, and what must be stopped

- **The status.** DSM-5 does not make catatonia an independent disorder: it is a specifier on another mental disorder, a catatonic disorder due to a medical condition, or unspecified catatonia, on three or more of twelve features. ICD-11 made it a grouping in its own right, with forms associated with another mental disorder, substance induced, and secondary.
- **Neurobiology.** Reduced GABA-A signalling in orbitofrontal and prefrontal cortex is the best supported account and explains the benzodiazepine response. Glutamatergic overactivity is supported by catatonia in anti-NMDA receptor encephalitis. Dopaminergic hypoactivity explains the resemblance to neuroleptic malignant syndrome.

OPTION	WHAT IT DOES	EVIDENCE SO FAR	STATUS
Lorazepam challenge	1 to 2 mg, response within minutes to hours; both a test and a treatment	**Open trials and large case series; high response rates in the non-malignant form**	First line everywhere
Continued lorazepam	Titration onward, often to 6 to 16 mg a day in divided doses	Observational; sedation is less than the dose suggests while catatonia persists	In routine use
Electroconvulsive therapy	Induced seizure, and the first line in malignant catatonia	**A long observational record and no alternative in the malignant form**	Established
NMDA antagonists	Amantadine or memantine when benzodiazepines fail	Case series only	Second line, weak evidence

WHAT MUST BE STOPPED

Antipsychotics. A dopamine blocker continued in a catatonic patient risks the malignant form or neuroleptic malignant syndrome, and the two are already hard to separate. Treat the cause too: encephalitis, metabolic derangement, withdrawal.

WHAT EARNS THE MARKS

- **The two systems contrasted,** specifier against independent grouping, which is the current status.
- **Each transmitter tied to a clinical observation,** not listed as three hypotheses.
- **The challenge dose given as both test and treatment,** which is the point of it.
- **Stopping the antipsychotic,** which most answers never write down.

SEE ALSO Slot IV - 73 neuroleptic malignant syndrome on a depot

Slot IV - 62 diagnosis of post-traumatic stress disorder **SPRINT** Day 38, Recent advances and landmark studies

AREA 09 OF 15 · P4-A12

Drug emergencies, interactions, TRD and neurostimulation

6	5.6%	IV-66	IV-71
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 6

REVISION ORDER

The four stimulation slots as one block; they declare their own division in the fragment comments and reading them together is how that division becomes visible. Take the mechanism slot first. The two resistance slots next, they are one answer split, and one of them contains the other's whole question as a sub-part.

WHAT IS IN THIS AREA

Brain stimulation in four slots: the neurosurgical options with repetitive transcranial magnetic stimulation, a critique of the stimulation treatments as a group, the physical treatments with the rTMS procedure in detail, and the mechanisms with the other mechanical methods. The other two are the resistance pair, add-on treatment set against augmentation, and the strategies for the patient who has not responded to standard therapy.

IV - 66

In contrast to single drug treatment in psychiatry, there is some inclination towards add-on drug treatment when a patient does not show adequate response to a drug. What is add-on drug treatment, and how does it differ from augmentation treatment? Critically evaluate the add-on approach in the clinical setting. [2+3+5]

2 + 3 + 5 | DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Both terms defined**	One sentence each, and the test that separates them
3	**The grid**	Examples, evidence and risk, side by side
3	**The critique**	Where combining is justified and where it is drift
2	**The rule before either**	An adequate first trial, and a plan to stop

DOMAIN	ADD-ON COMBINATION	AUGMENTATION	WHAT SETTLES IT
What is added	A second drug of the same class, both treating the disorder	A drug that is not itself a treatment for the disorder	**Ask what the added drug would do if given alone**
Worked examples	Mirtazapine or bupropion with a reuptake inhibitor	Lithium, liothyronine, aripiprazole, quetiapine	**Lithium is augmentation, never combination**
Evidence	The CO-MED trial found no gain from starting combined, with more adverse effects	Lithium and liothyronine carry the oldest data; atypicals carry approval	**Augmentation has the stronger trial base**
Main risk	Serotonin toxicity and additive adverse effects	Metabolic, thyroid and renal monitoring	**Same class stacking is the riskier of the two**
When to prefer	Partial response with a residual symptom the second drug covers	Partial response where the first drug is worth keeping	Both need an adequate first trial

Amber cells are the discriminators. The first row is the whole distinction; the rest of the grid only shows what follows from it.

- **The critique, honestly.** Add-on is justified when a specific residual symptom is named and the second drug is chosen to cover it, with a date set to review. It is drift when a drug is added because response was disappointing and nothing was reconsidered.
- **What combining hides.** An undiagnosed bipolarity, an unmentioned substance, a dose never raised, a trial never run long enough. Every one of these looks like resistance and none of them is.

WHAT EARNS THE MARKS

- **The test in the first row of the grid,** not two memorised lists of drugs.
- **A named trial on each side,** which is what turns evaluate into critically evaluate.
- **A stopping plan.** Almost no answer says how a drug that was added comes off again.

SEE ALSO Slot IV - 70 the resistance ladder SPRINT

Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 67

What are the options for neurosurgery in psychiatry? Write in brief about the indications and mechanism of action of rTMS. Critically evaluate the efficacy of rTMS in psychiatry. [2+2+2+4]

2 + 2 + 2 + 4 | DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Neurosurgical options	Ablative procedures, then the stimulation alternatives
2	Indications for rTMS	Who is offered it, and after what has failed
2	Mechanism	Induced current, frequency, and the circuit claim
4	Critical evaluation	Effect size, the ECT comparison, blinding, durability

THE NEUROSURGICAL OPTIONS, IN TWO GROUPS

Ablative: anterior cingulotomy, anterior capsulotomy, subcaudate tractotomy and limbic leucotomy. Non-ablative and reversible: deep brain stimulation and vagus nerve stimulation. In India, Section 96 of the Mental Healthcare Act 2017 permits psychosurgery only with the person's informed consent and the approval of the concerned Board.

- **Indications for rTMS.** A depressive episode that has not responded to antidepressant treatment, where the illness is not severe enough or urgent enough to need ECT. Obsessive compulsive disorder carries a separate approval, and there is trial evidence in auditory hallucinations and negative symptoms.
- **Mechanism.** A rapidly changing magnetic field over the scalp induces a current in the cortex beneath it and depolarises neurons. High frequency over the left dorsolateral prefrontal cortex is excitatory, low frequency over the right is inhibitory. The lasting effect is attributed to plasticity resembling long term potentiation and to downstream change in prefrontal and limbic circuits.
- **The critique.** Sham controlled trials and meta-analyses show a real but moderate antidepressant effect, below that of ECT. Blinding is imperfect because scalp sensation and muscle twitch differ from sham. Benefit needs maintenance, and durability is poorly studied. Theta burst stimulation was non-inferior to standard high frequency treatment in the THREE-D trial, which cut session time rather than raising the effect.
- **And the practical limit.** Four to six weeks of daily attendance at a centre that owns the machine. In Indian practice that is the constraint, not the evidence.

WHAT EARNS THE MARKS

- **Neurosurgery split into ablative and reversible,** with the statutory safeguard named.
- **Frequency tied to direction of effect,** which is the mechanism mark.
- **The comparison with ECT made explicitly.** An answer that praises rTMS without placing it has not evaluated anything.
- **Blinding named as a methodological problem,** not as a criticism of the operators.

SEE ALSO Slot IV - 69 the rTMS procedure and its complications Slot IV - 68 a critique of brain stimulation **SPRINT** Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 68

Write a critique of the use of brain stimulation treatments in psychiatry. [10]

10 DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**What is being critiqued**	Name what the phrase lumps together
4	**The evidence, tier by tier**	The ladder, drawn, with each rung graded
2	**The method problems**	Sham, blinding, durability of effect
2	**Access and ethics**	What is reachable here, and why consent is statutory

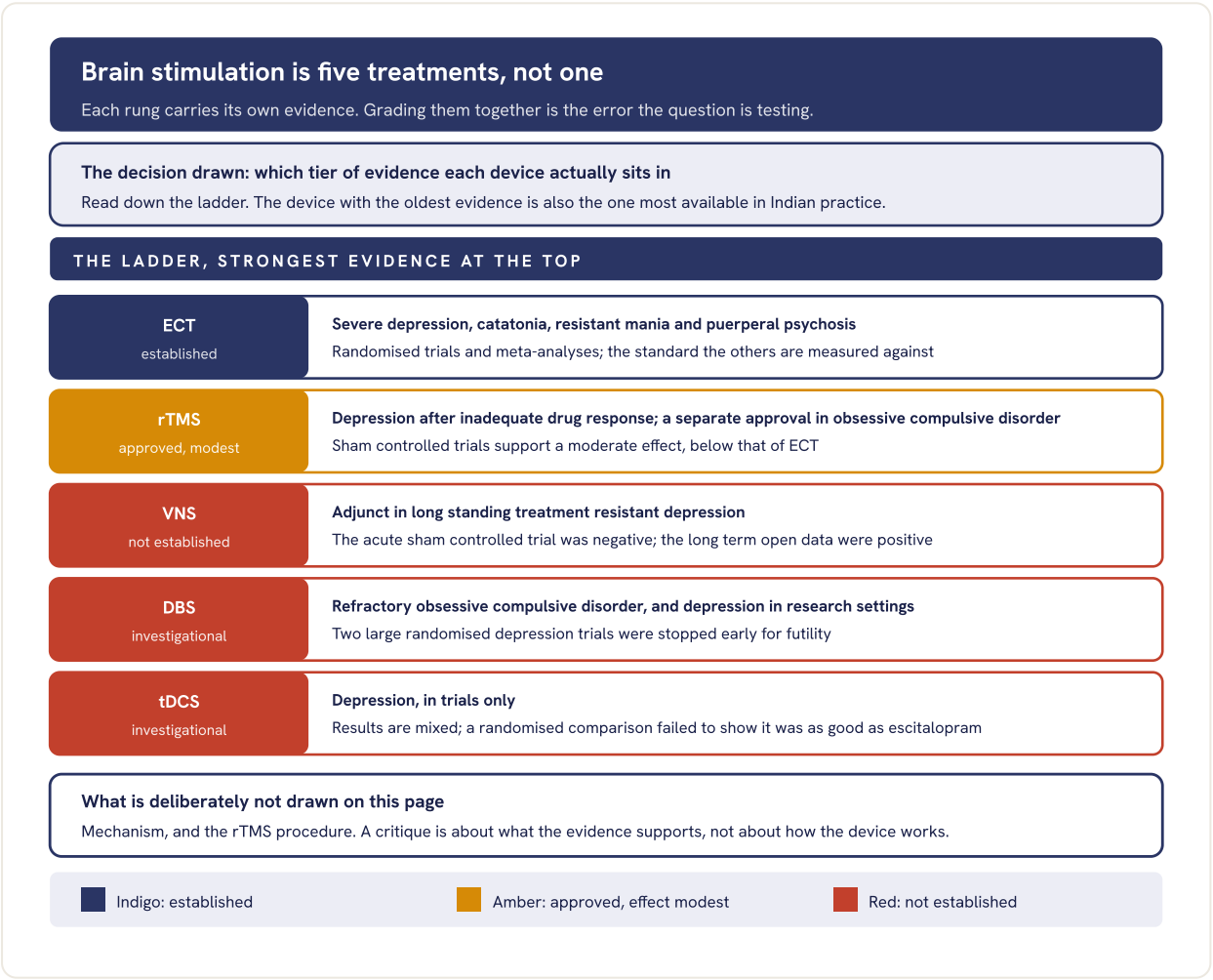


Fig 068.1 The family drawn as a graded ladder rather than a list, so that the critique is legible before a word of it is written.

- **The method problems.** Sham is the hard one: scalp sensation, muscle twitch and daily attendance cannot be blinded, so expectancy is built into every positive trial. Durability is barely studied, and maintenance schedules are empirical rather than trialled.
- **Access and ethics.** Cost and equipment concentrate these treatments in a few centres. The leucotomy era is why consent and Board approval for psychosurgery are written into statute rather than left to professional judgement.

WHAT EARNS THE MARKS

- **Refusing the category.** The first sentence should say that brain stimulation is not one evidence base.
- **Every rung graded,** including the two negative trial results most answers omit.
- **Sham named as the methodological weakness,** which is the difference between a critique and a complaint.

SEE ALSO Slot IV - 71 mechanism of brain stimulation Slot IV - 67 rTMS indications and efficacy **SPRINT**

Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 69

Describe the various physical treatment methods used in psychiatry. Discuss the indications, mechanism of action, procedure and complications of repetitive transcranial magnetic stimulation. [10]

10 | ASKED AT 15 MARKS | DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The catalogue	Grouped by whether a seizure is induced
2	Indication and mechanism	Who is offered it, and the induced current
4	The procedure	Threshold, target, dose, course
2	Complications and exclusions	What goes wrong, and who cannot have it

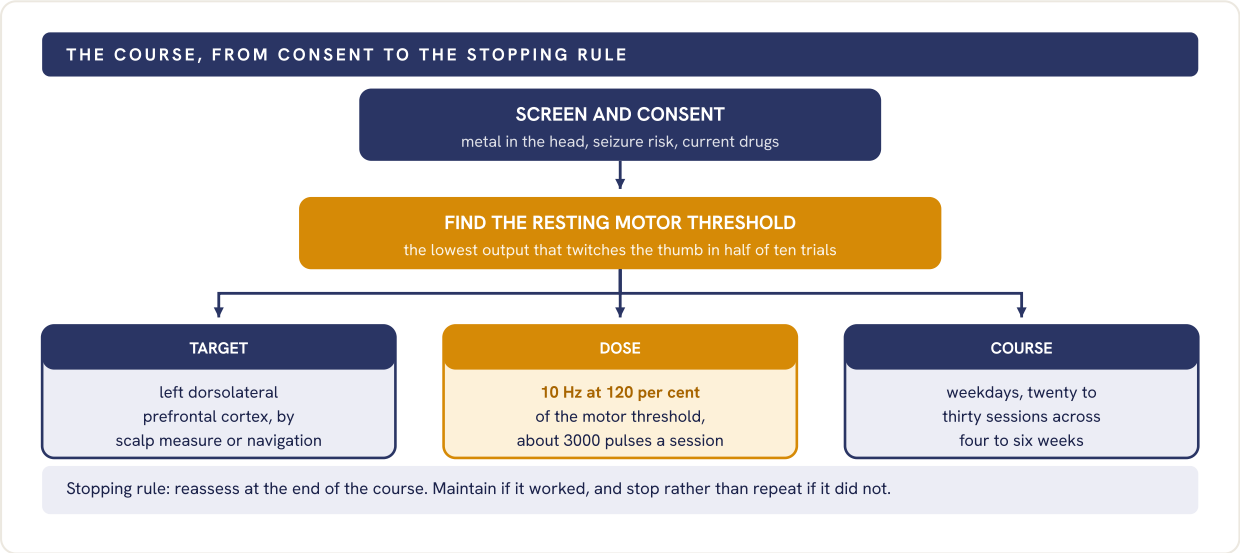


Fig 069.1 The threshold sits in amber because every later number on the page is a percentage of it, which is the step candidates omit.

- **The catalogue.** A seizure is induced: electroconvulsive therapy, magnetic seizure therapy. The cortex is stimulated without one: repetitive and deep transcranial magnetic stimulation, transcranial direct current stimulation. Implanted: vagus nerve and deep brain stimulation. Ablative: the psychosurgical procedures. Neither: bright light and sleep deprivation.
- **Complications and exclusions.** Scalp pain and headache early in the course, transient hearing change without earplugs, syncope, and a switch to mania in bipolar illness. Seizure is the serious risk: rare within published safety limits, and the reason for the exclusions. Do not treat a patient with ferromagnetic metal near the head, an implanted stimulator lead, or an untreated seizure risk.

WHAT EARNS THE MARKS

- **The catalogue grouped by a principle,** not listed alphabetically.
- **Motor threshold named as the reference for the dose,** which is the procedure mark.
- **Seizure separated from the nuisance effects,** and tied to the exclusion list rather than to a rate.

SEE ALSO Slot IV - 67 rTMS indications and efficacy Slot IV - 71 mechanism of brain stimulation **SPRINT**

Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 70

Discuss the various strategies to manage patients showing resistance to standard therapy. What is add-on drug treatment, and how is it different from augmentation treatment? [4+3+3]

4 + 3 + 3 | DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Before calling it resistance	Diagnosis, adherence, dose, duration
4	The ladder	Optimise, switch, augment, combine, then somatic
2	The two terms	Add-on against augmentation, and the test
2	Choosing on the ward	Severity and urgency, not the failure count

STRATEGY	WHAT IT MEANS	EVIDENCE	WHAT SETTLES IT
Optimise	Maximum tolerated dose, extended to six or eight weeks	Many so-called non-responders have had neither	**Do this before anything is added**
Switch	To another drug, within or across class	Remission rates were similar across switch options in the sequenced trial	Tolerability usually decides which
Augment	Add a drug that is not itself an antidepressant	Lithium and liothyronine carry the oldest data; atypicals carry approval	**The strategy with the best trial base**
Combine	Add a second antidepressant	Starting combined gave no advantage and more adverse effects	**The weakest evidence of the four**
Somatic	ECT, rTMS, or a ketamine based agent	ECT for severe, psychotic or urgent illness; rTMS for moderate resistance	**Severity and urgency, not the number of failures**

Psychological augmentation belongs on this ladder too: adding cognitive therapy performed comparably to adding a drug in the sequenced trial, and is the option least often offered.

- **The two terms.** Augmentation adds a drug that is not itself a treatment for the disorder, such as lithium or liothyronine. Add-on combination adds a second drug of the same class, so both are treating the illness. The test is simple: ask what the added drug would do if it were given alone.
- **Before any of it.** Resistance means two adequate trials in the current episode. Adequate means dose and duration, in a patient who took the drug, with the diagnosis rechecked for bipolarity, psychosis and substance use. Miss this and the ladder is climbed for the wrong illness.

WHAT EARNS THE MARKS

- **The pseudoresistance check written first,** before a single strategy is named.
- **The ladder given in order,** with the evidence attached to each rung rather than to the set.
- **The one line test for the two terms,** which is worth more than two lists of drug names.

SEE ALSO Slot IV - 66 add-on treatment critically evaluated Slot IV - 68 a critique of brain stimulation **SPRINT** Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 71

Discuss the mechanism of action of brain stimulation in psychiatry. What are the other mechanical methods used in psychiatric treatment, and what are their indications? [5+5]

5 + 5 | DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The organising question	Does the device fire neurons, or only bias them
3	Mechanism, device by device	Seizure, induced current, subthreshold shift
3	The other methods	Named, each with what it is used for
2	The honest limit	Where the mechanism is still not settled

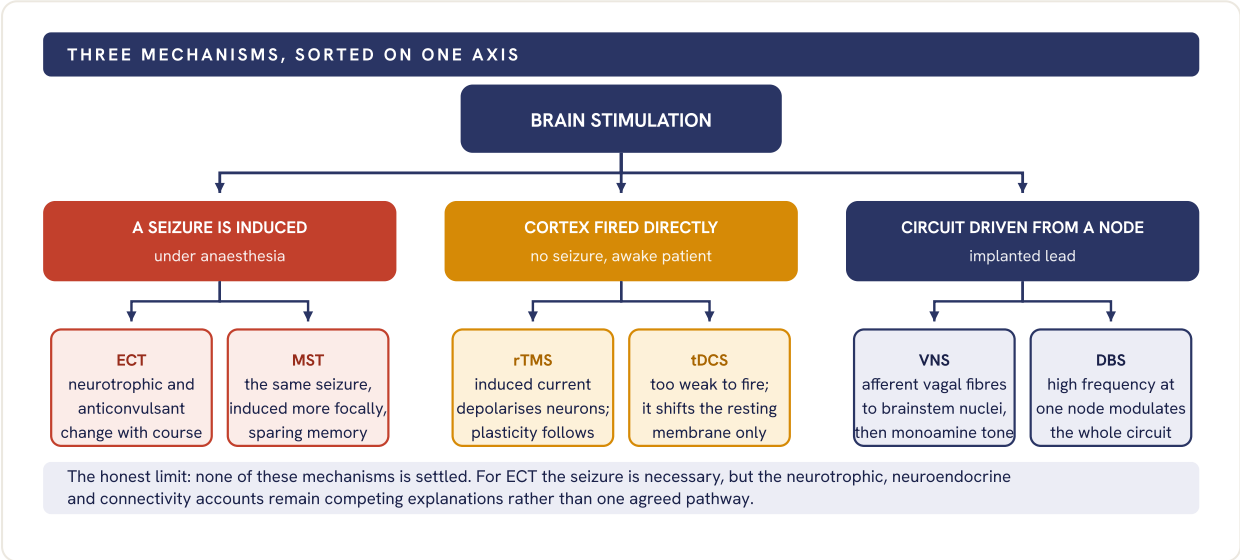


Fig 071.1 Every device sorted on one question, whether it fires neurons or only biases them, so the mechanism answer has a structure instead of six paragraphs.

- **The organising question.** Does the method induce a seizure, fire cortical neurons directly, or only bias the membrane so that whatever the brain is already doing becomes more or less likely? Sorting the devices on that axis is the mechanism answer.
- **The other mechanical methods, with indications.** Electroconvulsive therapy for severe depression with food refusal or high suicide risk, catatonia, resistant mania and puerperal psychosis. Bright light for seasonal depression. Sleep deprivation for a rapid but short lived lift, as an adjunct. Psychosurgery for refractory obsessive compulsive disorder and depression, under consent and Board approval.

WHAT EARNS THE MARKS

- **One axis stated before any device is described.** Six unsorted mechanisms is a mid-band answer.
- **The subthreshold point:** direct current stimulation does not fire neurons, and saying so separates it from the rest.
- **Each other method paired with its indication,** not listed on its own.
- **The limit strip along the foot,** which shows the field is understood rather than recited.

SEE ALSO Slot IV - 68 a critique of brain stimulation Slot IV - 69 the rTMS procedure **SPRINT**
Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

AREA 10 OF 15 · P4-A11

Psychopharmacology: principles and drug classes

5	5.0%	IV-72	IV-76
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

RECURRENT 1 SINGLE 4

REVISION ORDER

The two injectable slots together, and give the emergency limb its own time: it is the one map in this area that prints a drug dose under pressure. Lithium in youths next, where the comparison is the part that earns the marks. The me-too slot and vortioxetine last, and they share a critical-appraisal shape.

WHAT IS IN THIS AREA

The long acting injectables in two slots, one comparing the first and second generation depots and one that runs from the Indian preparations through their use in mood disorders to a suspected neuroleptic malignant syndrome. Then lithium in young people with bipolar disorder, the me-too drugs with two recent introductions to the Indian market, and vortioxetine for the acute treatment of major depressive disorder.

IV - 72

Discuss the advantages of depot antipsychotics. Compare the advantages and disadvantages of first generation with second generation depot antipsychotics. [4+6]

4 + 6 | PSYCHOPHARMACOLOGY: PRINCIPLES AND DRUG CLASSES

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What a depot is for	Adherence made visible, and steadier levels
2	The rest of the case	Relapse and admission, and what it does not fix
4	The grid	Domain by domain, discriminators marked
2	Choosing between them	Who gets which, and what decides it here

DOMAIN	FIRST GENERATION DEPOT	SECOND GENERATION INJECTION	WHAT SETTLES IT
Agents	Fluphenazine, haloperidol, flupentixol, zuclopenthixol decanoate	Risperidone, paliperidone palmitate, olanzapine pamoate	Paliperidone runs to three monthly
Starting up	Test dose, then slow titration under oral cover	Risperidone needs three weeks of oral cover; paliperidone loads on day one and day eight	**Only paliperidone needs no oral overlap**
Movement effects	Higher acute and tardive burden	Lower, dose related, never absent	**The one pharmacological reason to prefer the newer class**
Metabolic burden	Modest	Weight and lipid rise, worst with olanzapine	**The price paid for the movement gain**
Specific hazard	Effects persist for weeks after the last dose	Post-injection delirium with olanzapine pamoate	**Three hours of observation, for that one drug**

Amber cells are the discriminators. Cost is the sixth domain and the one left off the grid: first generation esters are generic and stocked, the newer injections are several times dearer.

- **Why a depot at all.** Non-adherence stops being invisible. A missed injection is a recorded event on a known date; a missed tablet is a guess.
- **The rest of the case.** Steadier levels, first-pass metabolism bypassed, and fewer relapses and admissions than the same drug taken orally in mirror-image and pragmatic studies.
- **What a depot does not fix.** It cannot be taken back, so an adverse effect resolves over weeks. It does not treat refusal, and it is not an answer to non-response.

WHAT EARNS THE MARKS

- **Advantage given as a mechanism.** Adherence made visible is the sentence that earns the first four marks.
- **A grid, with the movement and metabolic trade named as a trade.**
- **The observation rule attached to one drug,** which separates a read answer from a recalled one.

SEE ALSO Slot IV - 73 long acting injections and neuroleptic malignant syndrome **SPRINT**

Day 36, Psychopharmacology I: principles and core drug classes

IV - 73

Enumerate the long acting injectable antipsychotic preparations available in India. Describe their role in mood disorders. How would you manage a suspected neuroleptic malignant syndrome in a patient already on long acting antipsychotic treatment? [3+2+5]

3 + 2 + 5 | PSYCHOPHARMACOLOGY: PRINCIPLES AND DRUG CLASSES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 What is available	Both classes, named, with the dosing interval
2 Role in mood disorders	Maintenance in bipolar I, and the limit of the evidence
4 Managing the emergency	The flowchart: withhold, support, treat by severity
1 Rechallenge	The rule that prevents the second episode

- **Available in India.** First generation esters: fluphenazine decanoate, haloperidol decanoate, flupentixol decanoate and zuclopenthixol decanoate, given two to four weekly. Second generation injections: risperidone microspheres two weekly, paliperidone palmitate monthly and three monthly, and olanzapine pamoate.
- **Role in mood disorders.** Maintenance, never acute treatment. Randomised maintenance trials support risperidone long acting injection in bipolar I where relapses are manic and adherence is poor, with little effect on depressive relapse. No injection treats bipolar depression.
- **The severe lane, with its ceiling.** Intravenous dantrolene 1 to 2.5 mg per kg, may be repeated, to a maximum of 10 mg per kg per day.

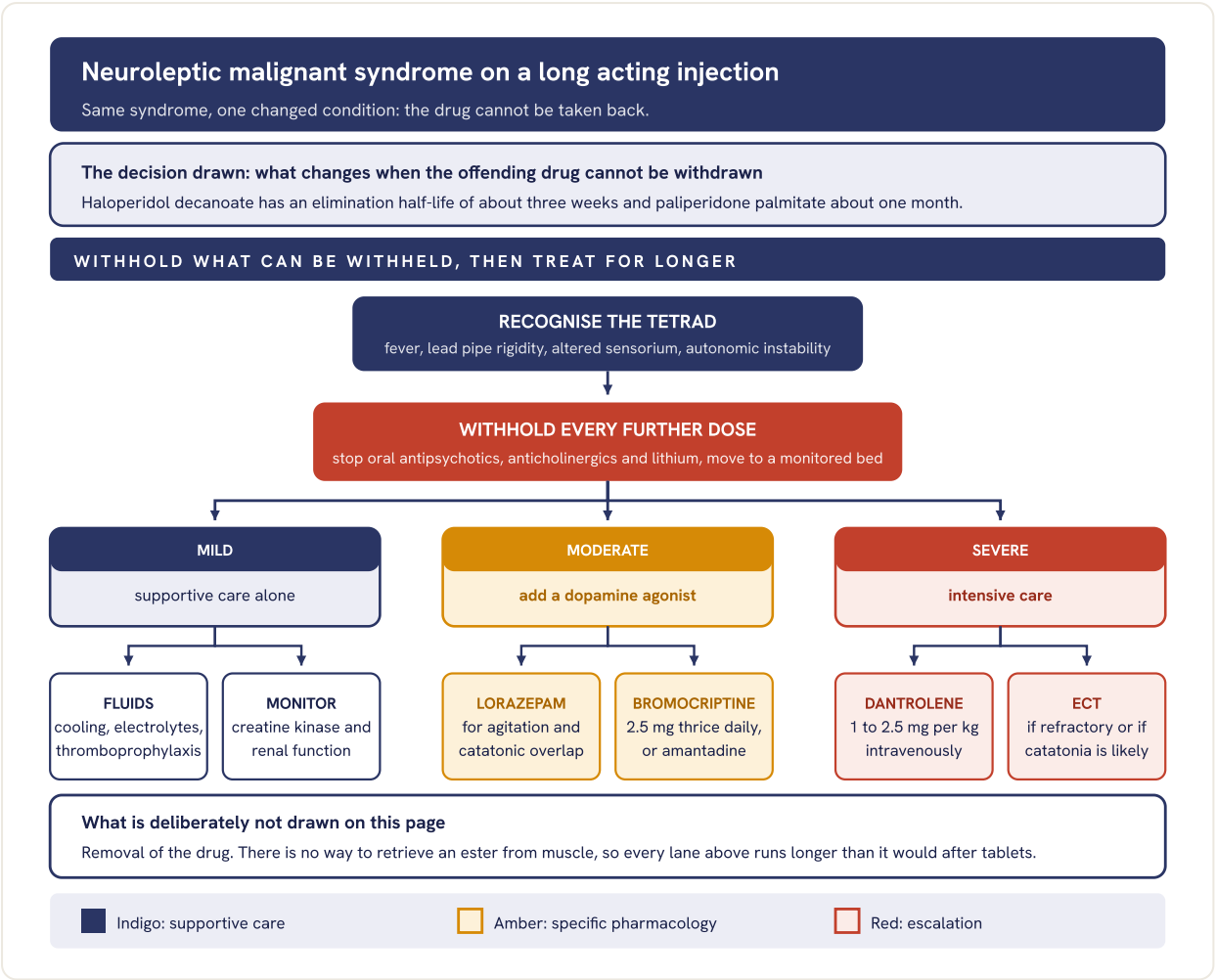


Fig 073.1 The ordinary algorithm redrawn for a drug that cannot be withdrawn, with the release half-life in the framing band so the longer course reads as a consequence.

RECHALLENGE

Never the same depot. Wait at least two weeks after full resolution, longer when the last injection was recent, then restart a lower potency oral agent slowly, watching temperature, pulse and creatine kinase.

WHAT EARNS THE MARKS

- **Both classes named with their intervals,** not the phrase "depot antipsychotics".
- **The framing band of the figure:** the drug cannot be withdrawn, so the course runs longer. Almost no answer says this.
- **The rechallenge box,** which most answers never reach.

SEE ALSO Slot IV - 72 depot antipsychotics compared Slot IV - 65 catatonia **SPRINT**

Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 74

"Lithium has advantages over other mood stabilisers in youths with bipolar disorder." Discuss this statement in the context of lithium being used as a first line mood stabiliser in adults with bipolar disorder. [10]

10 PSYCHOPHARMACOLOGY: PRINCIPLES AND DRUG CLASSES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The claim, unpacked**	What an advantage would have to mean in a child
3 **What supports it**	Licence, maintenance data, and the case against valproate
3 **What weakens it**	The head to head trial and the monitoring burden
2 **The position to defend**	Where the adult claim transfers and where it stops

ANSWER THE QUESTION THAT WAS ASKED

An advantage in a child must be one of three things: better efficacy in a head to head trial, a safety profile that suits a growing body, or an outcome that matters over decades. Lithium wins the third, argues the second, loses the first.

- **The licence and the record.** Lithium holds a paediatric licence for bipolar disorder in the United States and the longest paediatric record of any mood stabiliser. Maintenance levels of 0.6 to 1.0 mmol per litre are the usual target.
- **The comparison that matters.** Against valproate the case is strong, and strongest in girls: neural tube defects and neurodevelopmental harm after intrauterine exposure, polycystic ovary syndrome, weight gain, hepatotoxicity.
- **The head to head result.** The Treatment of Early Age Mania trial randomised children with mania to lithium, divalproex or risperidone. Risperidone gave the higher response rate, at the cost of weight and prolactin. Acute anti-manic evidence does not favour lithium.
- **The monitoring burden is the real limit.** A narrow index in a body that plays sport and gets gastroenteritis; levels pushed up by anti-inflammatories, thiazides and angiotensin converting enzyme inhibitors; toxicity above 1.5 mmol per litre; thyroid and renal tests an adolescent resents.
- **What does not transfer.** The anti-suicidal signal that anchors lithium in adults is adult data. A reasonable expectation in youths, not an established finding.

WHAT EARNS THE MARKS

- **The definition block:** naming what would count as an advantage before arguing about one.
- **The valproate comparison made sex specific,** which is where the paediatric case is won.
- **The head to head trial named against the claim,** not only the evidence that flatters it.
- **A stated position:** maintenance agent, not the acute first choice.

SEE ALSO Slot IV - 63 bipolar disorder and recent advances in treatment Slot IV - 76 vortioxetine in acute depression

SPRINT Day 36, Psychopharmacology I: principles and core drug classes

IV - 75

What are me too drugs? Discuss critically two new psychotropic drugs introduced in the Indian market. [2+4+4]

2 + 4 + 4 | PSYCHOPHARMACOLOGY: PRINCIPLES AND DRUG CLASSES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Me too, defined	The definition, and the test that settles it
4 The first agent	What is genuinely new and what is only claimed
4 The second agent	The receptor claim and the trials behind it

AGENT	WHAT IT DOES	EVIDENCE SO FAR	STATUS
Vortioxetine	Reuptake inhibition plus direct action at five serotonin receptors	**Placebo controlled trials, plus a cognitive signal on the digit symbol test**	Marketed in India, second line
Cariprazine	Partial agonist preferring the dopamine D3 receptor	**Trials in schizophrenia, mania and bipolar depression; beat risperidone on negative symptoms**	Marketed in India, specialist use
Escitalopram	The active enantiomer of citalopram, nothing else	A chiral switch; its small edge on the parent is disputed	Routine use, the standard example
Desvenlafaxine	The active metabolite of venlafaxine	No demonstrated advantage over the parent	Marketed, a follow-on

Rows one and two answer the question. Rows three and four are the contrast that makes the critique land: two drugs that reached the same market with no claim to novelty at all.

- **Me too, defined.** A drug close in structure or pharmacology to one already marketed, offering little therapeutic gain over it, developed for market share and patent life rather than unmet need. The test is a head to head trial: does it beat the incumbent on efficacy, safety or convenience?
- **Where the two stand.** Vortioxetine is ordinary by effect size; its distinct case is a cognitive signal, real but modest and still argued. Cariprazine has the stronger claim: D3 preference is a genuine departure, and its bipolar depression data do not exist for the drugs it replaces.

STATUS: WHAT A MARKETING LICENCE CERTIFIES

Approval anywhere, India included, rests on efficacy and acceptable safety, not on superiority to what is already on the shelf. A me too drug can be licensed, promoted and priced as new without ever having beaten the drug it copies.

WHAT EARNS THE MARKS

- **The definition followed immediately by the test.** Defining me too without saying how it would be recognised is half a mark.
- **Two named agents with their evidence in the same row,** not a paragraph of mechanism.
- **A different verdict for each,** which is what critically means.

SEE ALSO Slot IV - 76 vortioxetine in acute depression Slot IV - 64 therapeutic advance and prognosis **SPRINT** Day 38, Recent advances and landmark studies

IV - 76

Write briefly about the safety and efficacy of vortioxetine for the acute treatment of major depressive disorder. [5+5]

5 + 5 | PSYCHOPHARMACOLOGY: PRINCIPLES AND DRUG CLASSES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it is	Multimodal pharmacology, dose, and where it is placed
3	Efficacy	The placebo controlled trials, then the cognition claim
3	Safety	Nausea, sexual function, stopping, interactions
2	The honest verdict	What is proven, and what is only marketed

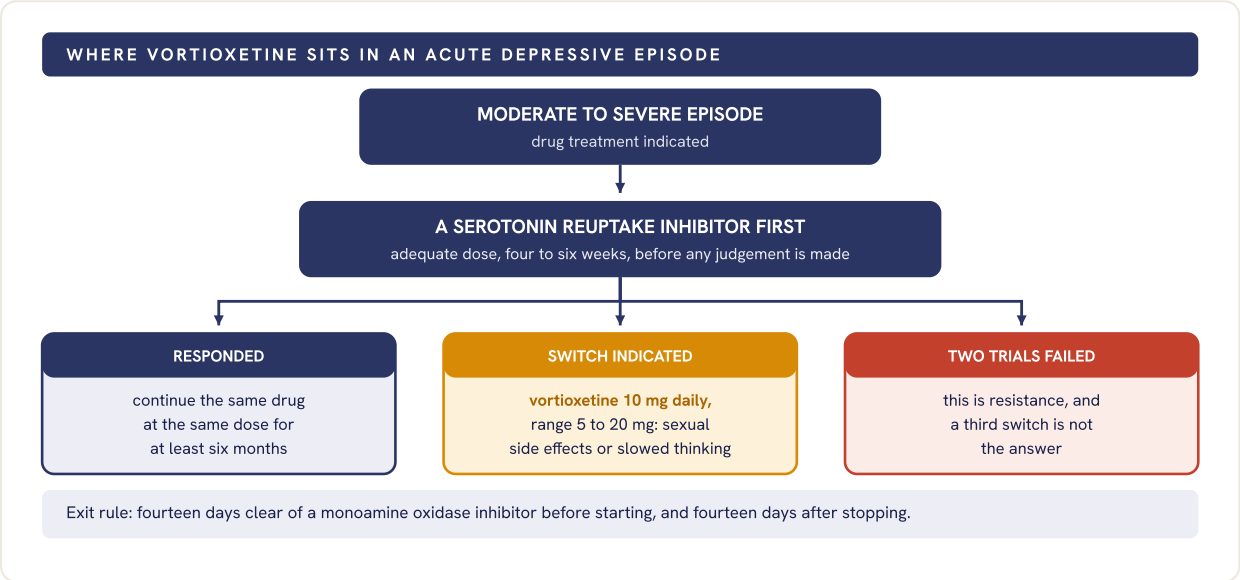


Fig 076.1 The drug placed rather than praised: vortioxetine enters at the switch, on two named grounds, and does not enter at the point where resistance has already been established.

- **Efficacy.** Short term placebo controlled trials separate on the Montgomery Asberg scale, and meta-analysis puts the effect with the other second generation antidepressants. The distinct claim is cognitive: gain on the digit symbol test, partly independent of mood change, where duloxetine did not separate. Real, modest, still argued.
- **Safety.** Nausea is commonest, dose related, worst at 20 mg and in the first fortnight. Little weight gain, less sexual dysfunction than the reuptake inhibitors, no meaningful QT effect, and few discontinuation symptoms given a half-life near sixty-six hours. Watch hyponatraemia in the elderly and reduce the dose with strong CYP2D6 inhibitors.

WHAT EARNS THE MARKS

- **Multimodal stated as pharmacology,** reuptake inhibition plus direct receptor action, not as a slogan.
- **The cognitive claim reported with its limit.** Naming the measure and the dispute is the top band answer.
- **The two grounds for the switch on the flowchart,** which is what the drug is actually for.

SEE ALSO Slot IV - 75 me too drugs and new agents in India Slot IV - 70 resistance and augmentation strategies

SPRINT Day 38, Recent advances and landmark studies

AREA 11 OF 15 · P4-A01

Epilepsy and psychiatry

5	4.9%	IV-77	IV-81
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 5

REVISION ORDER

The two classification slots together, they are the same answer at two widths and the operational classification writes both. The psychiatric manifestations slot next, it is the one most often set here. Absence seizures after that, and it is the one slot in this area that carries a drug dose. Magnetic seizure therapy last, and separately.

WHAT IS IN THIS AREA

The classification of epilepsy in two slots, one asking for it alone and one running on to the clinical features and management of complex partial seizures. Then the psychiatric manifestations of epilepsy with their management, and the management of absence seizures. The fifth slot asks for magnetic seizure therapy, which is a neurostimulation question the boards set inside the epilepsy area and is answered on its page as it was set.

IV - 77

Management of absence seizures. [10]

10 | EPILEPSY AND PSYCHIATRY | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Confirm the syndrome	Absence, not inattention: the record settles it
2	What decides the drug	Absence alone, or absence with tonic-clonic seizures
4	First line treatment	The named agent, the weight band, what it does not cover
2	Review and withdrawal	Seizure freedom, a clean record, then a slow taper

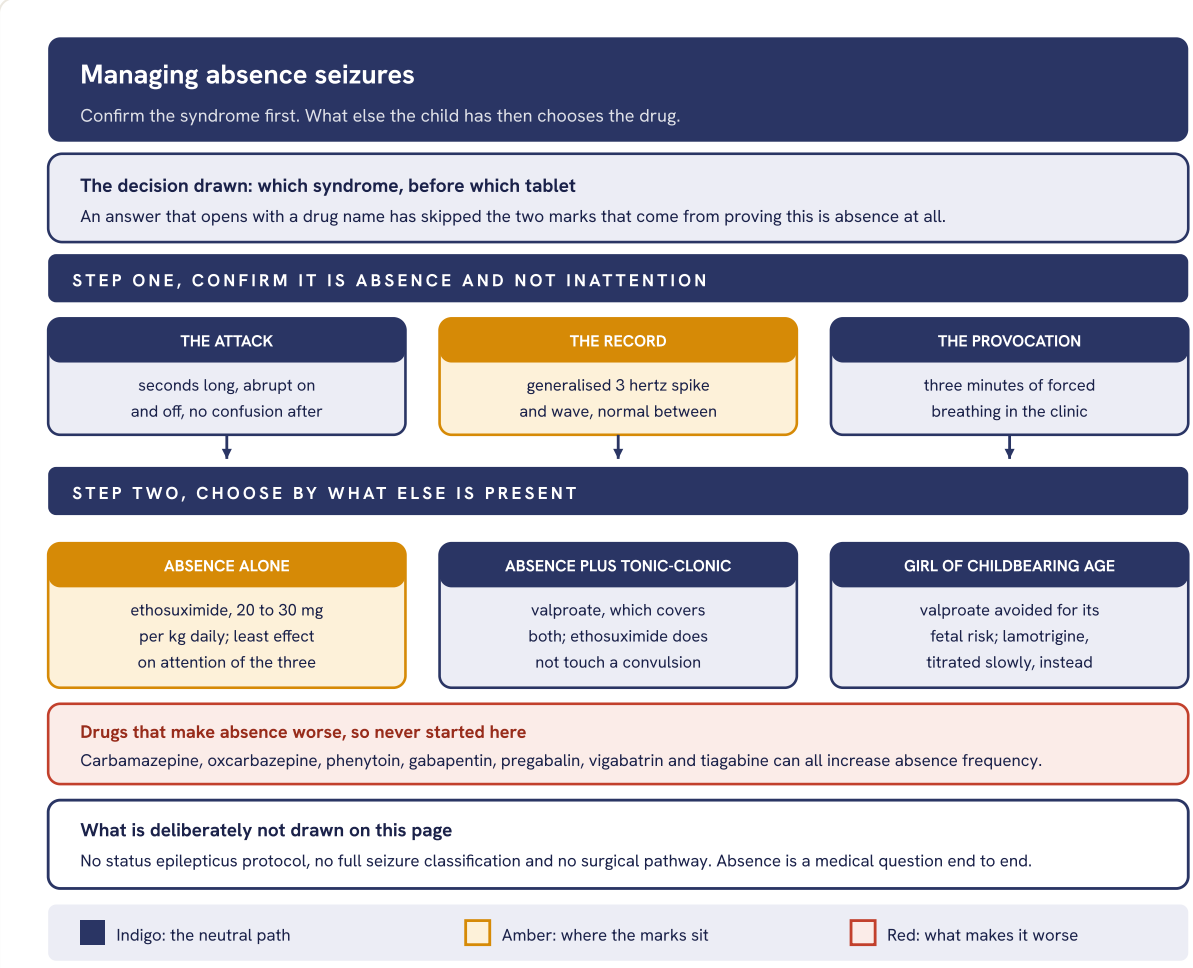


Fig 077.1 Confirmation first, then the drug chosen by what else the child has, with the aggravating drugs carried as a red strip because starting one is the commonest management error here.

- **Choosing between the three.** The three-arm comparative trial in childhood absence epilepsy found ethosuximide and valproate more effective than lamotrigine, and ethosuximide less impairing of attention. Where it is not stocked, valproate does this work.
- **Review and withdrawal.** Titrate to seizure freedom, confirmed by hyperventilation at each visit. After two seizure-free years with a clean record, taper slowly.
- **Attention does not always follow the record.** Inattention and academic underachievement often persist after the seizures stop, and need their own assessment.

PITFALL

The child called a daydreamer. Absence is not attention deficit disorder, a stimulant does not treat it, and a sodium channel blocker started for a wrongly labelled focal seizure makes it worse.

WHAT EARNS THE MARKS

- **Confirmation written before any drug.** Attack description, the record, and provocation at the bedside.
- **The branch rule stated.** Ethosuximide when absence stands alone, valproate when a convulsion has also occurred.
- **The aggravating list named.** Most answers list treatments and never say what is contraindicated.
- **The withdrawal rule.** Two seizure-free years and a slow taper is the last mark.

SEE ALSO Slot IV - 80 classification of epilepsy Slot IV - 79 psychiatric manifestations of epilepsy **SPRINT** Day 30, Epilepsy & psychiatry

IV - 78

What is magnetic seizure therapy? Discuss its role in psychiatry. [4+6]

4 + 6 | EPILEPSY AND PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	A therapeutic seizure induced magnetically, under anaesthesia
2	Why it differs	The skull does not resist a magnetic field
4	Role and evidence	Where it has been tried, and what the trials show
2	Status and limits	Investigational, scarce, and honestly stated as such

THE DEFINITION

Magnetic seizure therapy is the induction of a generalised therapeutic seizure by a high frequency, high intensity magnetic field delivered through a coil, with the patient anaesthetised and relaxed exactly as for electroconvulsive therapy. It is a convulsive treatment, not a stronger form of repetitive transcranial magnetic stimulation.

DOMAIN	ELECTROCONVULSIVE THERAPY	MAGNETIC SEIZURE THERAPY	WHAT FOLLOWS FROM IT
Energy delivered	Electric current across the scalp	Magnetic field through the scalp	**Skull and scalp do not attenuate a magnetic field**
Spread of the field	Diffuse, reaching deep structures	Focal, mainly superficial cortex	**Medial temporal structures are relatively spared**
Recovery	Slower reorientation after the fit	Faster reorientation, replicated	The most consistent trial finding
Memory	Retrograde and anterograde loss	Less autobiographical memory loss	**The whole rationale for the technique**
Efficacy in depression	Established, largest effect available	Not shown to match it	Why it stays a research treatment

Amber cells are the discriminators. The comparison is about where the field goes, and every other row follows from that one fact.

- **Role in psychiatry.** Treatment-resistant depression is the main indication studied, with smaller work in bipolar depression, schizophrenia and suicidal ideation.
- **Status, stated honestly.** Randomised comparisons with electroconvulsive therapy are few and small. The cognitive advantage is consistent; equivalent efficacy is not established. It remains investigational and is not in routine Indian practice.

WHAT EARNS THE MARKS

- **Named as a convulsive treatment.** Calling it a stronger form of repetitive stimulation loses the definition marks.
- **The physical reason for the difference.** Focality follows from the field, and the cognitive claim follows from focality.
- **Evidence separated from promise.** Better cognition is shown; matched efficacy is not.

SEE ALSO Slot IV - 77 management of absence seizures SPRINT Day 27, ECT & neurostimulation

IV - 79

Discuss psychiatric manifestations of epilepsy and their management. [6+4]

6 + 4 | EPILEPSY AND PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**The organising axis**	Time relative to the seizure names the syndrome
4	**The syndromes**	Peri-ictal, postictal, interictal, and drug induced
3	**Management**	Fix the epilepsy, then treat, watching the interaction
1	**The two traps**	Forced normalisation and the enzyme inducer

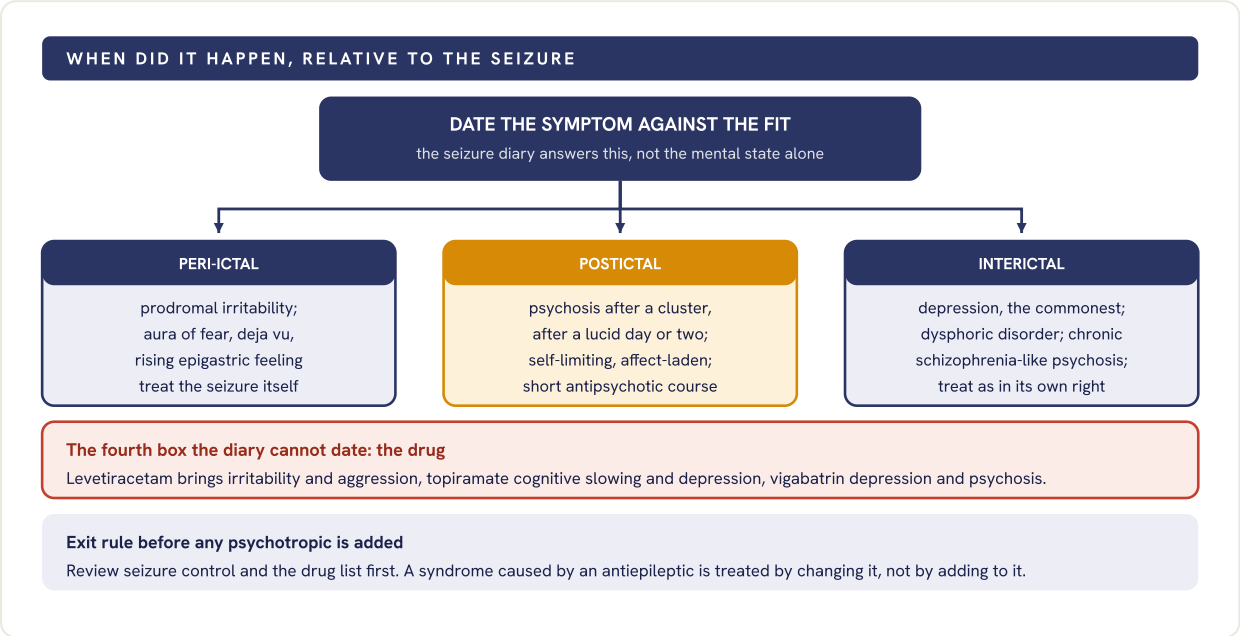


Fig 079.1 The syndromes sorted by their time relation to the seizure, with the drug-induced group set apart because it is the one the seizure diary cannot date.

- **Prescribing safely.** Selective serotonin reuptake inhibitors are the antidepressants of choice. Clozapine and chlorpromazine are the most epileptogenic antipsychotics.
- **The interaction that decides doses.** Carbamazepine, phenytoin and phenobarbitone induce enzymes and lower psychotropic levels. Valproate inhibits, so lamotrigine started alongside it needs half the usual dose to avoid a serious rash.

PITFALL

Forced normalisation. Psychosis can appear as the record normalises and the seizures stop, so raising the antiepileptic further can make the picture worse.

WHAT EARNS THE MARKS

- **The time axis named before any syndrome is listed.** A flat list of symptoms cannot earn the six description marks.
- **Postictal psychosis with its lucid interval,** separated from chronic interictal psychosis.
- **The drug reviewed before a drug is added,** and one named enzyme interaction.

SEE ALSO Slot IV - 81 complex partial seizures Slot IV - 77 management of absence seizures **SPRINT**

Day 30, Epilepsy & psychiatry

IV - 80

How is epilepsy classified. [10]

10 | EPILEPSY AND PSYCHIATRY | KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The framework	Three levels, with cause and comorbidity at each
3	Level one, the seizure	Onset: focal, generalised, or unknown
3	Levels two and three	Epilepsy type, then the named syndrome
2	Cause and comorbidity	Six aetiological groups, asked at every level

THE FRAMEWORK

The current classification is the operational scheme of the International League Against Epilepsy, published in 2017. It works in three levels: the seizure type, then the epilepsy type, then the epilepsy syndrome where one can be named. Aetiology and comorbidity are asked at every level, not appended at the end.

LEVEL	THE QUESTION IT ANSWERS	THE CATEGORIES	WHAT IT DECIDES
Seizure type	Where does the seizure begin	Focal, generalised, unknown onset	**Drug choice**
Focal, subdivided	Is awareness kept, and is onset motor	Aware or impaired awareness; motor or non-motor; focal to bi-lateral tonic-clonic	**Replaces simple and complex partial**
Generalised, subdivided	Motor or not	Tonic-clonic, tonic, clonic, my-oclonic, atonic, spasms; absence typical or atypical	Sodium channel blockers avoided
Epilepsy type	What pattern does this person have	Focal, generalised, combined, unknown	Prognosis and counselling
Syndrome	Does a named entity fit	Age at onset, seizure types, record, imaging taken together	**Prognosis and duration of treatment**
Aetiology	Why is it happening	Structural, genetic, infectious, metabolic, immune, unknown	Treatable cause found or excluded

Amber cells are the discriminators. The scheme is diagnostic in sequence, so each level narrows what the next one can be.

- **The terms retired in 2017.** Partial became focal; simple partial and complex partial became focal aware and focal impaired awareness. Use both names.
- **The Indian entry point.** Neurocysticercosis is the commonest identified cause of new focal seizures in adults here, and sits in the infectious group alongside tuberculoma, so imaging is part of classifying rather than an afterthought.

WHAT EARNS THE MARKS

- **Three levels named as levels,** in order, before any category is listed.
- **Both halves of the focal split.** Awareness and motor onset, with the retired terms mapped across.
- **The six aetiological groups,** stated as running through every level rather than as a final list.

SEE ALSO Slot IV - 81 complex partial seizures Slot IV - 77 management of absence seizures SPRINT

Day 30, Epilepsy & psychiatry

IV - 81

Classification of Seizure disorder. Discuss clinical features and management of Complex Partial Seizures. [10]

10 | EPILEPSY AND PSYCHIATRY | RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Place it in the scheme**	Focal impaired awareness in the current terms
3 **Clinical features**	Aura, automatisms, and the signs that lateralise
2 **Confirming it**	Record, epilepsy protocol imaging, video when unsure
3 **Management**	First drug, the resistance rule, then surgical referral

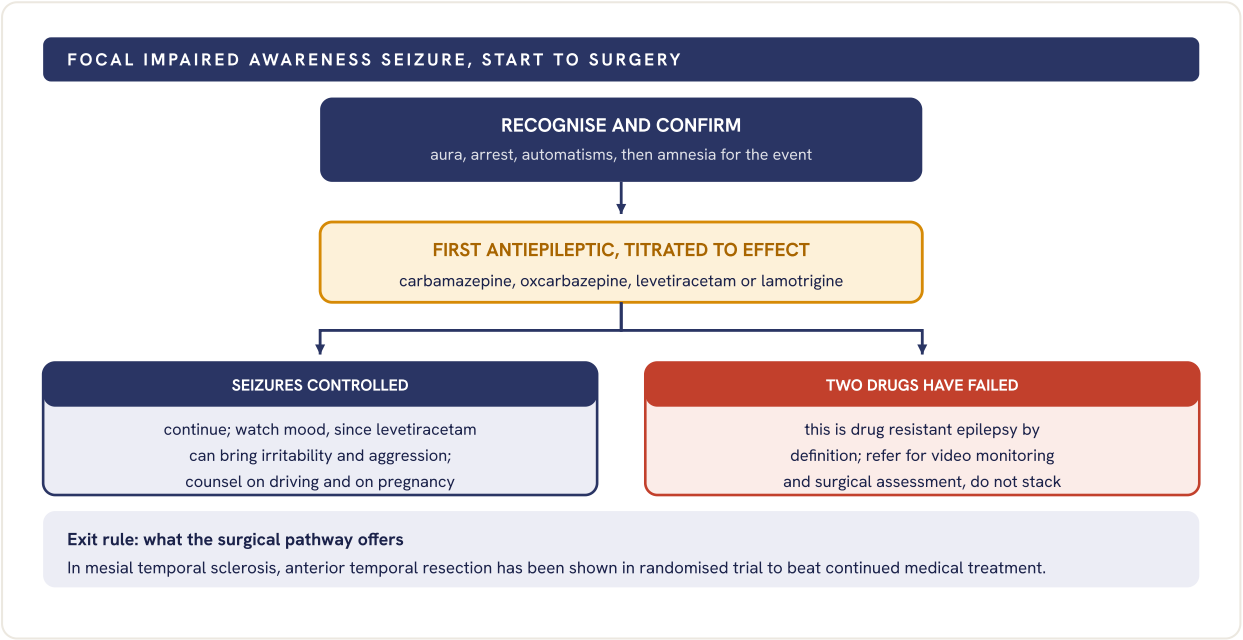


Fig 081.1 One drug, then a decision point that most answers never reach: two failed drugs is a definition, not a setback, and it routes the patient to assessment rather than to a third tablet.

- **Features, in order.** Aura of rising epigastric sensation, fear or deja vu; motionless stare; lip smacking, chewing and fumbling; one to two minutes; then amnesia.
- **What lateralises.** Dystonic posturing points away from the focus, automatisms and nose-wiping towards it, dysphasia after the fit to the dominant hemisphere.

PITFALL

Automatisms with amnesia get read as dissociation or panic: absence has no aura and no confusion after it, and a panic attack is remembered.

WHAT EARNS THE MARKS

- **The current name given alongside the old one.** Focal impaired awareness seizure, formerly complex partial.
- **At least one lateralising sign,** which is what separates a description from an assessment.
- **Drug resistance defined by two failed drugs,** and the referral that follows it.

SEE ALSO Slot IV - 80 classification of epilepsy Slot IV - 79 psychiatric manifestations of epilepsy **SPRINT** Day 30, Epilepsy & psychiatry

AREA 12 OF 15 · P4-A06

Nutritional, toxic and metabolic encephalopathies

5	4.2%	IV-82	IV-86
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 3 SINGLE 2

REVISION ORDER

Thiamine deficiency and the alcohol complications slot together, they overlap almost completely and one is the general case of the other. Hepatic encephalopathy next. Then the SIADH slot, and read its correction rate carefully rather than quickly. Wilson disease last, and it is self-contained.

WHAT IS IN THIS AREA

Wilson disease with its management, the neuropsychiatric manifestations of thiamine deficiency, hepatic encephalopathy, and the neurological complications of alcohol dependence syndrome. The fifth slot is the syndrome of inappropriate antidiuretic hormone secretion, which is an endocrine question sitting in a nutritional and toxic area, and it holds the most consequential number in this book.

IV - 82

Neuropsychiatric manifestation of Wilson disease and its management. [5+5]

5 + 5 | NUTRITIONAL, TOXIC AND METABOLIC ENCEPHALOPATHIES

DNB, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

1	The disease	Copper, one gene, and the age window it declares itself in
4	Neuropsychiatric picture	Movement, psychiatric and cognitive, in that order
4	Management	Confirm, decopper, maintain, then treat the psychiatry
1	The family rule	Screen every sibling, because it is treatable and inherited

THE DEFINITION

An autosomal recessive disorder of copper transport caused by mutation in the ATP7B gene on chromosome 13. Biliary copper excretion fails, copper accumulates in liver then brain, and the basal ganglia take the damage. It is one of the few reversible dementias, which is why it is asked.

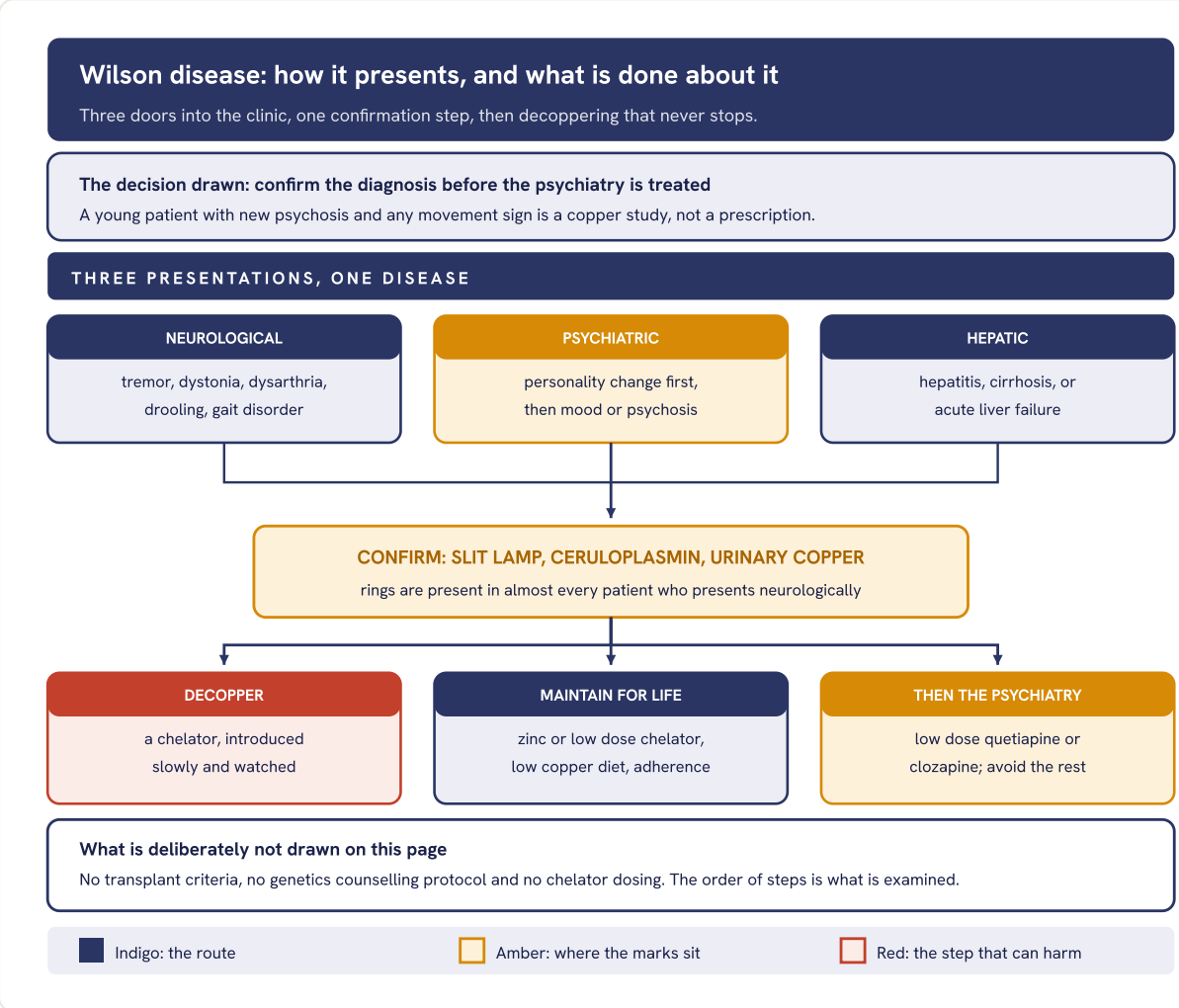


Fig 082.1 Three routes into the clinic, one confirmation step that must precede treatment, and the three arms of management that follow it.

- **The psychiatric picture.** Personality change and falling school or work performance come first, and are usually read as adolescence. Depression is the commonest syndrome, with real self harm risk; psychosis occurs but is rarer than textbooks imply. Cognitive decline is subcortical: slow, dysexecutive, memory relatively spared.
- **Confirming it.** Kayser-Fleischer rings on slit lamp, low serum ceruloplasmin, raised twenty four hour urinary copper. Rings are present in nearly all neurological presentations but may be absent in purely hepatic ones, so their absence never excludes the disease.
- **The trap.** Chelation can worsen neurological signs in the first weeks, so it is introduced slowly and the patient is warned. Never give an antipsychotic with high extrapyramidal liability to an untreated patient.

WHAT EARNS THE MARKS

- **Personality change named as the first psychiatric sign.** Most answers open with psychosis and lose the natural history.
- **The confirmation step written before any treatment.** Rings, ceruloplasmin, urinary copper.
- **Lifelong maintenance, not a course.** Stopping is why a treated patient deteriorates.
- **Screening the siblings.** One line, and it separates a good answer from a competent one.

SEE ALSO Slot IV - 48 Huntington's disease Slot IV - 84 hepatic encephalopathy **SPRINT** Day 33, Endocrine & metabolic psychiatry

IV - 83

Neuropsychiatric manifestations of thiamine deficiency. [10]

10 NUTRITIONAL, TOXIC AND METABOLIC ENCEPHALOPATHIES

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	The deficiency	What thiamine does, and who runs out of it
3	Wernicke encephalopathy	The triad, and how it actually presents
3	Korsakoff syndrome	The amnestic picture that follows an untreated attack
2	Treatment	Parenteral, high dose, and before any carbohydrate

THE DEFINITION

Thiamine is a cofactor for transketolase and for the pyruvate and alpha-ketoglutarate dehydrogenase complexes, so deficiency starves the brain of usable glucose. Body stores last only weeks. The damage falls on structures around the third and fourth ventricles: mammillary bodies, medial thalamus and periaqueductal grey.

- **Wernicke encephalopathy.** The classical triad is confusion, ophthalmoplegia and ataxia, but all three are present in only a minority; most patients show confusion alone, and the diagnosis is missed for that reason. At risk are alcohol dependence, hyperemesis, bariatric surgery, prolonged vomiting, malnutrition and refeeding.
- **Treatment.** Parenteral thiamine, in doses far above the nutritional requirement, given on suspicion rather than on proof. The commonly taught regimen is intravenous thiamine three times daily for the first two to three days, then a lower daily dose continued orally. Oral thiamine alone is inadequate in the acute phase because absorption is saturable.
- **Korsakoff syndrome.** Dense anterograde amnesia with disorientation for time, confabulation and apathy, on a background of clear consciousness and preserved immediate recall and procedural memory. It follows most untreated attacks of Wernicke encephalopathy and is only partly reversible.

PITFALL

Glucose given before thiamine can precipitate or deepen Wernicke encephalopathy, because the last thiamine is consumed driving the glucose load. In any malnourished or alcohol dependent patient thiamine goes in first. A normal blood thiamine level never excludes the diagnosis.

WHAT EARNS THE MARKS

- **The triad named, and then contradicted.** Saying that it is complete in a minority is the line that shows clinical reading.
- **Thiamine before glucose, written as an order of actions.**
- **Korsakoff described as a distinct amnestic syndrome,** not as a later stage of the same confusion.
- **The parenteral route, with the reason oral fails.**

SEE ALSO Slot IV - 86 neurological complications of alcohol Slot IV - 93 amnestic disorder SPRINT Day 33, Endocrine & metabolic psychiatry

IV - 84

Hepatic encephalopathy. [10]

10 NUTRITIONAL, TOXIC AND METABOLIC ENCEPHALOPATHIES

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Definition and mechanism	Ammonia, the astrocyte, and the shunt
2	Grading	West Haven, and the minimal grade above it
3	Clinical picture	The psychiatric presentations it is mistaken for
3	Management	Find the precipitant first, then lactulose and rifaximin

DEFINITION, AND HOW IT PRESENTS

A reversible neuropsychiatric syndrome of liver failure or portosystemic shunting. Gut derived nitrogenous substances, chiefly ammonia, reach the brain, where astrocytes swell and GABAergic tone rises. It presents as delirium, but also as depression, apathy or personality change, and in the minimal grade only as impaired driving and falling work performance. Sleep-wake reversal is often the earliest change a family reports.

GRADE	CONSCIOUSNESS	BEHAVIOUR AND COGNITION	BEDSIDE SIGN
Minimal	Normal	Normal on examination; deficits only on testing	None
One	Sleep-wake reversal	Euphoria or anxiety, short attention span	**Mild asterixis or tremor**
Two	Lethargy, apathy	Disorientation for time, personality change	**Obvious asterixis, slurred speech**
Three	Somnolent but rousable	Gross disorientation, confusion, bizarre behaviour	**Asterixis often lost, rigidity**
Four	Coma	Unresponsive	Decerebrate posturing

Amber cells are the bedside discriminator. Asterixis appears early and is lost again as the patient sinks, so its absence means either grade zero or grade four, never the middle.

- **Precipitants carry the marks.** Gastrointestinal bleeding, infection including spontaneous bacterial peritonitis, constipation, dehydration and diuretics, electrolyte disturbance, a high protein load, and sedative drugs. Finding and correcting the precipitant is the treatment; lactulose and rifaximin support it. Blood ammonia correlates poorly with grade, and a normal value does not exclude the diagnosis.

WHAT EARNS THE MARKS

- **The precipitant searched for before any drug is named.** That single line is the difference between treating and reciting.
- **A grade written with its bedside sign,** not as an unqualified word like confusion.
- **Minimal hepatic encephalopathy mentioned.** Most answers begin at grade one and lose the mark.
- **Benzodiazepines named as harmful here,** with the short acting exception if sedation cannot be avoided.

SEE ALSO Slot IV - 92 delirium Slot IV - 86 neurological complications of alcohol SPRINT

Day 33, Endocrine & metabolic psychiatry

IV - 85

Syndrome of Inappropriate Anti Diuretic Hormone Secretion (SIADH). [10]

10 | ASKED AT 7 MARKS | NUTRITIONAL, TOXIC AND METABOLIC ENCEPHALOPATHIES

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Definition and criteria	Euvolaemic hyponatraemia with inappropriately concentrated urine
2	Causes	The psychotropics first, since they are ours
2	Clinical picture	What a falling sodium looks like on a ward round
3	Management	Stop the drug, restrict fluid, respect the correction rate

THE DEFINITION

Antidiuretic hormone secretion that continues despite plasma hypo-osmolality, producing dilutional hyponatraemia in a clinically euvolaemic patient. The diagnosis needs all of: low serum sodium with low plasma osmolality, urine osmolality inappropriately high, raised urine sodium, clinical euvolaemia, and normal thyroid, adrenal and renal function in a patient not taking diuretics.

- **Why this belongs to psychiatry.** Selective serotonin reuptake inhibitors are the commonest psychotropic cause, typically in older patients within the first weeks of treatment. Carbamazepine and oxcarbazepine, antipsychotics and tricyclics all cause it. Other causes are small cell lung carcinoma, pneumonia and tuberculosis, stroke and head injury, and pain or nausea.
- **The picture.** Mild falls give nausea, headache, poor concentration and unsteadiness; moderate falls give confusion, delirium and falls themselves; severe falls give seizures and coma. Every one of these is read at first as the illness being treated, or as dementia in an older patient.
- **Two things it is not.** In psychogenic polydipsia the urine is dilute, not concentrated, because the kidney is behaving correctly. In cerebral salt wasting the patient is volume depleted rather than euvolaemic, and needs salt and water rather than restriction.

PITFALL

Correct slowly. Raising serum sodium faster than roughly eight to ten millimoles per litre in twenty four hours risks osmotic demyelination. The patient who improves for a day and then deteriorates with dysarthria, quadriparesis or a locked-in picture has been corrected too fast, and that damage is not reversible.

WHAT EARNS THE MARKS

- **The full criterion set, including euvolaemia** and the normal endocrine and renal function that must be shown.
- **Psychotropic causes named first.** The examiner is asking a psychiatrist.
- **The correction rate, with the harm it prevents.** Most answers stop at fluid restriction.

SEE ALSO Slot IV - 92 delirium Slot IV - 59 psychiatric disorders of thyroid and parathyroid disease SPRINT Day 33, Endocrine & metabolic psychiatry

IV - 86

Neurological complications of Alcohol dependence syndrome. [10]

10 NUTRITIONAL, TOXIC AND METABOLIC ENCEPHALOPATHIES

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The four mechanisms	Withdrawal, nutritional, direct toxicity, and the indirect routes
3 Acute complications	Seizures, delirium tremens, Wernicke encephalopathy
3 Chronic complications	Amnestic, cerebellar, peripheral, cognitive
2 The named rarities	The two syndromes that separate a top answer

MECHANISM	SYNDROME	THE RECOGNISING FEATURE	TIMING
Withdrawal	Seizures, delirium tremens	**Generalised fits within two days; delirium with tremor and autonomic storm**	Hours to days
Thiamine loss	Wernicke, then Korsakoff	**Confusion with eye signs and ataxia, then dense anterograde amnesia**	Days, then lasting
Direct toxicity	Cerebellar degeneration	Wide based gait and truncal ataxia, arms and speech spared	Years
Direct toxicity	Peripheral neuropathy	Symmetrical, distal, sensory first, painful burning feet	Years
Direct toxicity	Alcohol related dementia	Dysexecutive, with relative sparing of recognition memory	Years
Indirect, hepatic	Hepatic encephalopathy	Asterixis, sleep reversal, a known cirrhotic liver	Variable
Indirect, traumatic	Subdural haematoma	Fluctuating consciousness after a fall that may not be recalled	Weeks
Osmotic and rare	Central pontine myelinolysis; Marchiafava-Bignami	Over-fast sodium correction; corpus callosum demyelination	Days to weeks

Amber rows are the emergencies. Organising the answer by mechanism rather than by an alphabetical list is itself worth marks, because it shows why each complication happens.

- **The two that separate a top answer.** Marchiafava-Bignami disease is demyelination of the corpus callosum, giving dementia, gait disturbance and interhemispheric disconnection signs. Central pontine myelinolysis follows too rapid correction of hyponatraemia and gives dysarthria, quadriparesis and a locked-in state.
- **The overlap trap.** These rarely arrive alone. A dependent patient who is confused may be withdrawing, thiamine depleted, hepatic, post-ictal and bleeding under the dura at once, so each mechanism is excluded rather than assumed.

WHAT EARNS THE MARKS

- **The answer organised by mechanism,** not as an undifferentiated list of names.
- **Acute separated from chronic,** because the two demand different actions on the day.
- **The two rare named syndromes.** They are the cheapest distinguishing marks on this page.

SEE ALSO Slot IV - 83 thiamine deficiency Slot IV - 43 subdural haematoma SPRINT

Day 33, Endocrine & metabolic psychiatry

AREA 13 OF 15 · P4-A09

Neuro-infective and autoimmune neuropsychiatry

5	4.0%	IV-87	IV-91
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 SINGLE 4

REVISION ORDER

The two HIV slots together, the second sits inside the first. Anti-NMDA receptor encephalitis next, it is the longest answer here and the only one asked across five limbs. Tuberculous meningitis after that. PANDAS last, and it is the shortest map in the area.

WHAT IS IN THIS AREA

HIV in two slots, the neuropsychiatric manifestations of the disease and the AIDS dementia complex. Then the psychiatric manifestations of tuberculous meningitis, anti-NMDA receptor encephalitis asked from aetiology through to management, and PANDAS.

IV - 87

Describe the neuropsychiatric manifestations of HIV disease. [10]

10 NEURO-INFECTIVE AND AUTOIMMUNE NEUROPSYCHIATRY

KUHS, NTRUHS, RGUHS - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2	The framework	Five routes by which HIV reaches the mind
3	Direct effects	The neurocognitive spectrum, and its subcortical shape
3	Indirect effects	Opportunistic infection, tumour, and the drugs
2	Management	Treat what is treatable, and prescribe around the antiretrovirals

THE FRAMEWORK

Answer by route, not by symptom. HIV reaches the brain directly, permits opportunistic infection and tumour, is treated with drugs that carry neuropsychiatric effects, and brings stigma and loss of its own. Naming the routes before listing anything is what organises this answer.

ROUTE	WHAT IT PRODUCES	THE CLUE
Direct viral	Neurocognitive impairment, from asymptomatic through mild disorder to HIV-associated dementia	Subcortical profile, tracks viral load
Opportunistic and neoplastic	Cerebral toxoplasmosis, cryptococcal meningitis, tuberculous meningitis, progressive multifocal leukoencephalopathy, primary central nervous system lymphoma	**Focal signs, fever, headache; low CD4 count**
Iatrogenic	Efavirenz causing vivid dreams, mood change and rarely psychosis; steroids; interferon	**Temporal link to starting a drug**
Psychosocial	Depression, adjustment disorder, substance use, suicide risk	May precede or follow the diagnosis

Amber marks the two clues that change management on the day: a low CD4 count reframes any new neurological sign as an opportunistic infection until imaged, and a recent drug change is the cheapest explanation to exclude.

- **The cognitive profile.** Subcortical: psychomotor slowing, poor attention and executive function, apathy and withdrawal, with motor clumsiness and tremor. Memory is relatively spared early, and aphasia and agnosia are absent, which is what separates it from a cortical dementia. In India tuberculous meningitis is the opportunistic infection to think of first.
- **Prescribing.** Depression is the commonest psychiatric diagnosis here and is under-treated, though it responds normally. Watch enzyme interactions between antiretrovirals and psychotropics, and combined effects on the QT interval. Never stop antiretroviral therapy to manage a psychiatric side effect without the treating physician.

WHAT EARNS THE MARKS

- **The five routes named before any list.** It converts recall into a structure.
- **The cognitive picture identified as subcortical,** with what is spared.
- **Drug interactions mentioned.** Most answers stop at naming the syndromes.

SEE ALSO Slot IV - 91 AIDS dementia complex Slot IV - 88 tuberculous meningitis SPRINT

Day 35, Neuropsychiatry of systemic & neurological disease

IV - 88

Describe Psychiatric manifestations of Tuberculous Meningitis. [10]

10 NEURO-INFECTIVE AND AUTOIMMUNE NEUROPSYCHIATRY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Why psychiatrists see it	A subacute basal meningitis whose first changes are behavioural
3	The psychiatric picture	Prodrome, delirium, and the syndromes it imitates
3	Diagnosis	The fluid, the images, and why treatment does not wait
2	The drugs	The treatment carries psychiatric hazards of its own

WHY THIS REACHES A PSYCHIATRIST

Tuberculous meningitis is a subacute meningitis centred on the base of the brain, with exudate, hydrocephalus and infarction rather than the abrupt prostration of pyogenic disease. It remains common in India. Its prodrome is measured in weeks and is behavioural before it is neurological, so the first doctor consulted is often a psychiatrist.

- **The presentation.** Weeks of malaise, low grade fever, night sweats, weight loss and headache, with apathy, irritability and personality change that families describe as depression. Then confusion and delirium, cranial nerve palsies with the sixth and third most affected, meningism, seizures and the papilloedema of hydrocephalus.
- **The syndromes it imitates.** Depression with marked apathy, an acute or subacute psychosis, delirium of unclear cause, and occasionally catatonia. Later, after treatment, a residual amnestic or dysexecutive picture from infarction and hydrocephalus, which is the part that does not recover.
- **Diagnosis, and the drugs.** Cerebrospinal fluid shows a lymphocytic pleocytosis with high protein and low glucose; culture is slow, so nucleic acid amplification and imaging of basal exudates carry the early diagnosis, and treatment is never delayed for confirmation. Isoniazid causes psychosis and neuropathy, prevented by pyridoxine; cycloserine causes depression, psychosis and suicidal ideation; steroids alter mood; and rifampicin induces liver enzymes, lowering levels of many psychotropics.

PITFALL

A young adult with weeks of headache, low fever and a change in personality is imaged and tapped before an antipsychotic is started. Treating this as a first episode of psychosis costs weeks, and the harm done by hydrocephalus and infarction in that time does not reverse.

WHAT EARNS THE MARKS

- **The behavioural prodrome named as the reason for the referral.**
- **The cerebrospinal fluid picture, all three values.**
- **Antitubercular drugs named as psychiatric causes,** with pyridoxine for isoniazid.
- **The residual deficit acknowledged.** Recovery is not always complete, and saying so is honest.

SEE ALSO Slot IV - 87 neuropsychiatric manifestations of HIV Slot IV - 92 delirium **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease

IV - 89

PANDAS. [10]

10 NEURO-INFECTIVE AND AUTOIMMUNE NEUROPSYCHIATRY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition and criteria	The five, named, with the mechanism proposed
3	Clinical picture	What the abrupt onset actually looks like
2	Investigation	What supports it, and what has to be excluded
3	Management and status	Three arms, and an honest word on the evidence

THE FIVE CRITERIA

Paediatric autoimmune neuropsychiatric disorders associated with streptococcal infection. Obsessive compulsive disorder or a tic disorder; onset before puberty; abrupt onset with an episodic course of dramatic relapse and remission; a temporal association with group A streptococcal infection; and associated neurological signs, particularly choreiform movements. The proposed mechanism is molecular mimicry, with antibodies to streptococcus cross-reacting with basal ganglia.

- **The picture.** A previously well child develops obsessions, compulsions or tics almost overnight, and parents can usually name the day. Around it come separation anxiety, urinary frequency, deteriorating handwriting, emotional lability, irritability and restricted eating. The abruptness, not the content, is what is distinctive.
- **Investigation.** A throat swab and antistreptococcal antibody titres support recent infection but never confirm the diagnosis, since raised titres are common in healthy school-age children. There is no diagnostic test. Exclude Sydenham chorea, Wilson disease, autoimmune encephalitis, and an ordinary tic or obsessive compulsive disorder with a coincidental infection.
- **Management.** Treat proven streptococcal infection with antibiotics. Treat the psychiatric disorder on its own terms: cognitive behavioural therapy with exposure and response prevention, and a selective serotonin reuptake inhibitor started at a low dose, because these children are often sensitive. Immunomodulation is confined to severe cases in specialist hands.

THE HONEST POSITION

The entity is contested. PANS is the broader construct, which drops the streptococcal requirement and accepts any abrupt onset presentation. Saying that the diagnosis is disputed, and giving the reason, earns more than presenting it as settled fact; long term antibiotic prophylaxis in particular is not established.

WHAT EARNS THE MARKS

- **All five criteria, with abruptness and the episodic course.**
- **Molecular mimicry named as the proposed mechanism.**
- **Titres described as supportive but not diagnostic.**
- **The controversy stated, with PANS distinguished.**

SEE ALSO Slot IV - 90 anti-NMDA receptor encephalitis Slot IV - 82 Wilson disease **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease

IV - 90

Discuss in detail about the etiology, clinical features, differential diagnosis, assessment and management of Anti-NMDA receptor encephalitis. [10]

10 | ASKED AT 15 MARKS | NEURO-INFECTIVE AND AUTOIMMUNE NEUROPSYCHIATRY

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Aetiology	The antibody, the tumour, and the post-viral route
3	Clinical features	The stages, in the order they arrive
2	Differential and assessment	What it imitates, and what confirms it
3	Management	Immunotherapy, the tumour, and supportive care

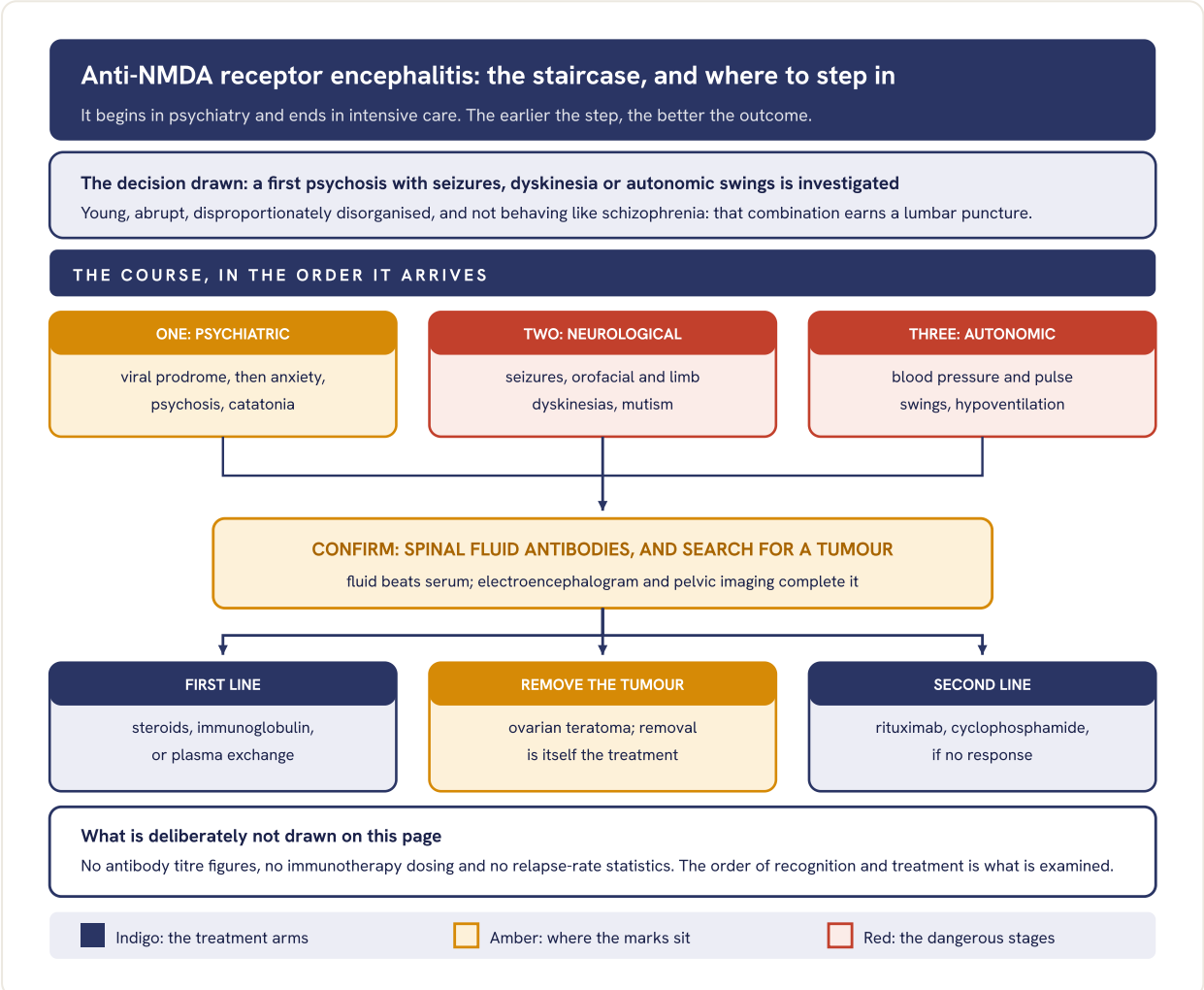


Fig 090.1 The staged course from psychiatric presentation to autonomic instability, the confirmatory step, and the three arms of treatment that follow it.

- **Aetiology and differential.** An antibody against the NR1 subunit of the NMDA receptor. Some cases are paraneoplastic, classically an ovarian teratoma in a young woman; others follow herpes simplex encephalitis; many have no trigger found. It is mistaken for a first episode of schizophrenia, for a drug induced psychosis, for catatonia, and, once rigid and febrile on antipsychotics, for neuroleptic malignant syndrome.

- **What confirms it.** Antibodies in cerebrospinal fluid, which is more sensitive than serum and may be positive when serum is negative, so a negative blood test never excludes it. The electroencephalogram is diffusely slow and may show the extreme delta brush pattern; magnetic resonance imaging is often normal, which misleads. Tumour search includes pelvic imaging.

WHAT EARNS THE MARKS

- **The stages given in order,** which is what makes the diagnosis recognisable in a psychiatric ward.
- **Spinal fluid preferred over serum,** stated as a rule with its reason.
- **Tumour search named as treatment,** not merely as investigation.
- **Normal imaging noted as common,** so a clean scan does not close the question.

SEE ALSO Slot IV - 89 PANDAS Slot IV - 92 delirium **SPRINT**
Day 35, Neuropsychiatry of systemic & neurological disease

IV - 91

AIDS Dementia Complex. [10]

10 NEURO-INFECTIVE AND AUTOIMMUNE NEUROPSYCHIATRY

KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition and naming	The older term, and where it sits in the current scheme
3	Clinical picture	The subcortical triad: cognitive, motor, behavioural
2	Investigation	A diagnosis of exclusion, and what must be excluded
3	Management and course	What antiretroviral therapy changed, and what it did not

THE DEFINITION

A subcortical dementia caused by HIV itself rather than by opportunistic infection. The older name AIDS dementia complex now sits as the most severe tier of HIV-associated neurocognitive disorder, and using the current three-tier scheme while naming the old term is the safer answer.

TIER	COGNITIVE TESTING	EFFECT ON DAILY FUNCTION
Asymptomatic neurocognitive impairment	Impairment in two or more domains on formal testing	None reported or observed
Mild neurocognitive disorder	Impairment in two or more domains	Mild interference with work or daily activity
HIV-associated dementia	**Marked impairment in two or more domains**	**Marked interference; this is the old AIDS dementia complex**

Amber marks the severe tier the question asks about. The three tiers differ by severity of testing impairment and by functional effect, and quoting both axes is what the definition marks are for.

- **The picture is subcortical.** Cognitive: psychomotor slowing, poor attention and concentration, impaired executive function, with memory affected through retrieval rather than storage. Motor: clumsiness, tremor, unsteady gait, hyperreflexia, and later frontal release signs. Behavioural: apathy, social withdrawal and loss of drive, often mistaken for depression. Aphasia and agnosia are absent, which separates it from a cortical dementia.
- **Exclusion, then treatment.** Imaging shows atrophy and diffuse white matter change; spinal fluid is examined mainly to exclude cryptococcal disease, toxoplasmosis, tuberculosis, lymphoma and neurosyphilis. Depression is the commonest and most treatable mimic and is excluded before dementia is diagnosed. Antiretroviral therapy is the treatment: severe dementia has become much less common since it, though milder impairment persists in treated patients.

WHAT EARNS THE MARKS

- **The current three-tier scheme, with the old name placed inside it.**
- **Subcortical named, with the cortical features that are absent.**
- **Depression excluded explicitly** before the dementia is diagnosed.
- **Antiretroviral therapy given as the treatment,** with what it did and did not change.

SEE ALSO Slot IV - 87 neuropsychiatric manifestations of HIV Slot IV - 19 subcortical dementias **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease

AREA 14 OF 15 · P4-A08

Delirium and amnestic syndromes

5	3.9%	IV-92	IV-96
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 RECURRENT 1 SINGLE 3

REVISION ORDER

The three delirium slots together and in that order: define it, screen for it, then examine. Inattention is the core feature on all three and all three stems require it. The two amnestic slots next, they are one answer split by axis and both fragments declare the split.

WHAT IS IN THIS AREA

Delirium in three slots: the definition with the causes and the management, the screening instruments, and the examination of cognitive function at the bedside. The amnestic syndromes in two, one asking for the disorder itself and one for the criteria and the causes sorted by mechanism.

IV - 92

Define delirium. What are the common causes of delirium? Discuss the management of delirium. [2+2+6]

2 + 2 + 6 | DELIRIUM AND AMNESTIC SYNDROMES

DNB, KUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 Definition	Attention first, then the acute onset and the fluctuation
2 Causes	Grouped so that they can actually be searched
5 Management	Cause, then environment, then a drug if forced
1 Outcome	Why it is never a benign episode

THE DEFINITION

An acute, fluctuating disturbance of attention and awareness, with an additional disturbance of cognition, developing over hours to days, and caused by a medical condition, a substance or withdrawal. Inattention is the core feature; the disturbed conscious level is what separates it from dementia.

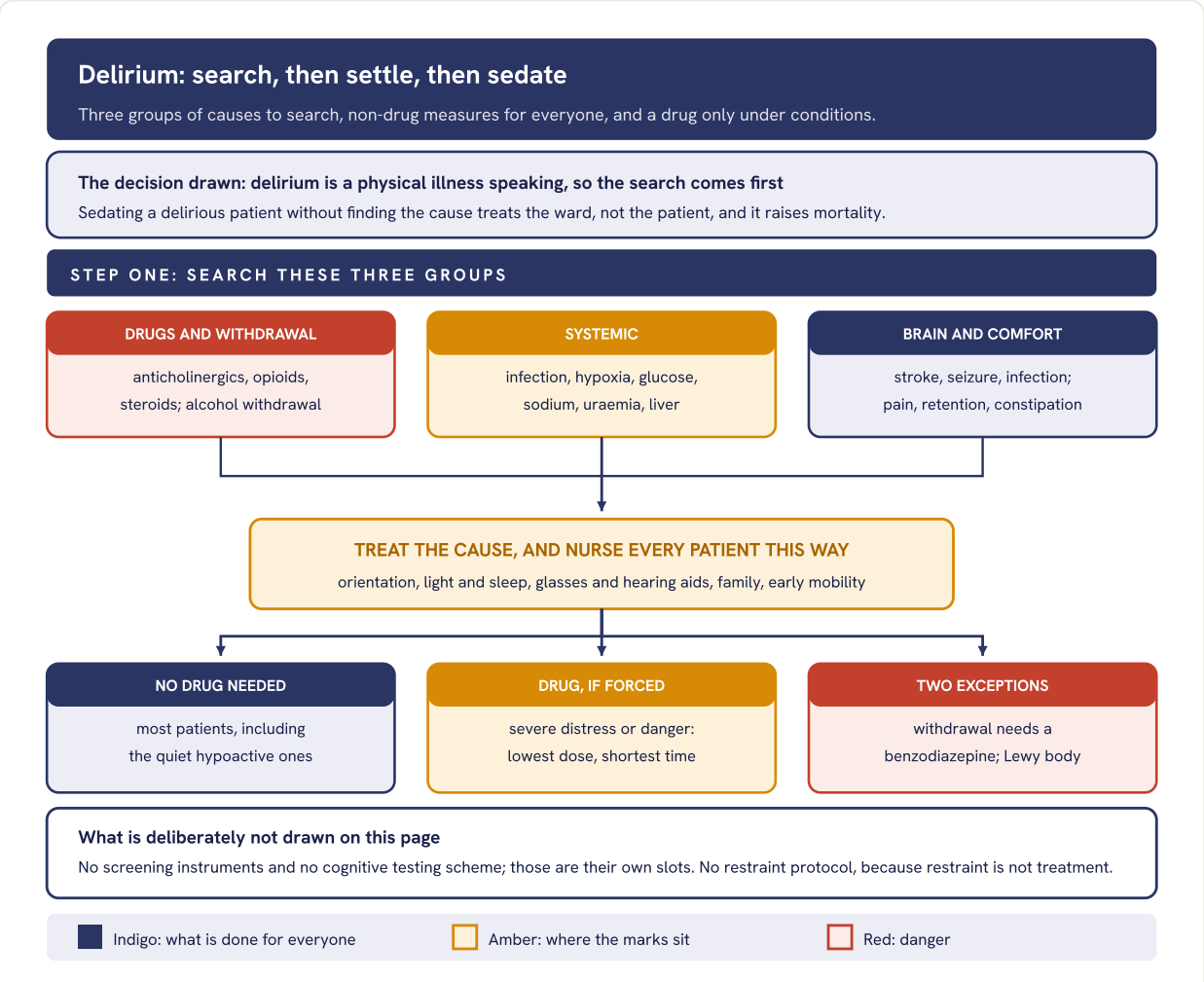


Fig 092.1 Three groups of causes to search, the non-drug care that every delirious patient receives, and the narrow conditions under which a drug is added.

- **If a drug is unavoidable.** A low dose antipsychotic, for the shortest possible time, reviewed daily. Haloperidol in small doses is the usual choice. In Parkinson disease and Lewy body dementia antipsychotics can be dangerous, and quetiapine or clozapine are preferred. Benzodiazepines are first line only in alcohol or sedative withdrawal.
- **The one most often missed.** Hypoactive delirium is commoner than the agitated form, is mistaken for depression or for tiredness, and carries the worse outcome. A quiet patient who is inattentive is delirious until proven otherwise.

WHAT EARNS THE MARKS

- **Inattention named as the core feature** in the opening definition.
- **Causes grouped, not listed,** so the answer reads as a search strategy.
- **Non-drug measures written before any drug,** and given to every patient.
- **The hypoactive subtype named,** with the fact that it does worse.

SEE ALSO Slot IV - 94 screening assessment in delirium Slot IV - 95 cognitive assessment in delirium **SPRINT** Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 93

Amnestic disorder. [10]

10 DELIRIUM AND AMNESTIC SYNDROMES RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Definition	One domain lost, everything else standing
2	The anatomy	Two circuits, and why either one produces it
4	Clinical picture	What is lost, what is spared, and confabulation
2	Management	Treat the cause, then rehabilitate around the deficit

THE DEFINITION

An acquired impairment of memory severe enough to disrupt daily function, occurring in clear consciousness and without the general cognitive decline of dementia. Anterograde memory carries the damage; the isolation of the deficit is what defines the syndrome.

- **The anatomy.** Two circuits, and damage to either produces the same syndrome. The medial temporal route through hippocampus, fornix and mammillary bodies, and the diencephalic route through the mammillary bodies and dorsomedial thalamus. Both are links in the Papez circuit, which is why bilateral damage anywhere along it is enough.
- **Lost and spared.** Lost: new learning, so the patient cannot lay down day to day memory, with retrograde amnesia that is worst for the period nearest the injury. Spared: immediate recall and digit span, procedural and skill memory, language, perception and, at least early, social manner. That preserved surface is why families and wards underestimate the disability.
- **Confabulation.** Filling gaps with plausible or fantastic material, not deliberately, and it is most marked early and where frontal damage accompanies the amnesia. Correcting the patient repeatedly is futile and distressing; it fades as the syndrome stabilises.

PITFALL

Clear consciousness separates this from delirium, and the intact non-memory domains separate it from dementia. Test attention explicitly before concluding amnesia: an inattentive patient cannot register anything, so their apparent memory failure is delirium wearing an amnestic mask.

WHAT EARNS THE MARKS

- **The deficit named as isolated,** with the spared domains listed as evidence.
- **Both anatomical routes,** not the hippocampus alone.
- **Confabulation described without moral language.**
- **Attention tested first,** which is the line that rules out delirium.

SEE ALSO Slot IV - 96 criteria and causes of amnesic syndromes Slot IV - 83 thiamine deficiency SPRINT Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 94

Screening assessment in Delirium. [10]

10 DELIRIUM AND AMNESTIC SYNDROMES RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Why screen at all	It is missed in most cases unless it is looked for
2	Who, and when	The at-risk groups, and the daily rhythm of screening
4	The instruments	What each one is for, and who can administer it
2	After a positive screen	A screen is not a diagnosis; what follows it

INSTRUMENT	WHAT IT DOES	WHO USES IT	SETTING
Confusion Assessment Method	Four features: acute fluctuating course, inattention, disorganised thinking, altered consciousness	Trained clinician	General ward, the reference standard
4AT	Alertness, orientation, attention by months backward, acute change; needs no training	Any staff member	Rapid bedside and admission screening
CAM-ICU	The same four features adapted for the ventilated, non-speaking patient	Intensive care staff	Critical care
Nursing Delirium Screening Scale	Observational, scored across a nursing shift	Nursing staff	Repeated observation over time
Delirium Rating Scale, revised	Grades severity rather than presence	Clinician	Monitoring response, and research

Amber marks the two that answer most questions. The Confusion Assessment Method requires features one and two to be present, plus either three or four; the 4AT is the practical instrument where no one has been trained.

- **Who and when.** Screen everyone over sixty five, everyone with dementia or prior delirium, everyone admitted with hip fracture or acute severe illness, and all intensive care patients. Screen on admission and then at least daily, because the disorder fluctuates and a single normal screen means very little.
- **A screen is not a diagnosis.** A positive screen triggers a diagnostic assessment against the full criteria, a search for the cause, and a review of the drug chart. A negative screen in a patient the nurses say is not himself is a reason to screen again, not a reason to stop.

WHAT EARNS THE MARKS

- **The instruments named with what each one is for,** rather than as a list of acronyms.
- **The rule for the Confusion Assessment Method,** which features must be present.
- **Daily repetition justified by fluctuation.**
- **The line separating a screen from a diagnosis.**

SEE ALSO Slot IV - 92 definition, causes and management of delirium Slot IV - 95 cognitive assessment in delirium

SPRINT Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 95

Assessment of Cognitive functions in Delirium. [10]

10 | DELIRIUM AND AMNESTIC SYNDROMES | KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The principle	Attention is tested first and everything is read through it
4 The domains	Each one, and how it is tested at the bedside
2 Instruments and limits	What the standard scales cannot do in a fluctuating patient
2 The discrimination	Delirium against dementia and against depression

- **Attention first.** Digit span forwards, months of the year backwards, serial sevens, or a simple vigilance task. If attention is impaired, every other score becomes a floor rather than a measurement, because a patient who cannot attend cannot register, and cannot then recall.
- **Then the domains.** Orientation to time before place; registration and delayed recall; language and naming; visuospatial function by clock drawing or intersecting pentagons; thought process for disorganisation; perception for illusions and visual hallucinations; and level of consciousness, which is observed rather than asked about.
- **The limits of the scales.** The mini mental state examination and the Montreal Cognitive Assessment were built for stable patients. In delirium they fluctuate hour to hour, they cannot separate inattention from amnesia, and a low score records how impaired the patient is, never why. Retesting at a different time of day is worth more than one score.

FEATURE	DELIRIUM	DEMENTIA	DEPRESSION
Onset	Hours to days	Months to years	Weeks
Course over a day	**Fluctuates, worse at night**	Stable, slowly progressive	Stable, often worse in the morning
Attention	**Impaired, the core feature**	Preserved until late	Variable, effort dependent
Consciousness	**Altered, hyper or hypoalert**	Clear until very late	Clear
Perception	Visual hallucinations, illusions	Later, less prominent	Uncommon, mood congruent
Memory failure	Registration fails, through inattention	Recall fails despite registration	**Answers do not know, improves with cues**

Amber cells are the discriminators. Fluctuation, inattention and a clouded conscious level settle delirium; the pattern of memory failure is what separates dementia from depression.

WHAT EARNS THE MARKS

- **Attention tested first, with a named test.** Saying assess cognition earns nothing.
- **The limits of the standard scales stated plainly.**
- **The three-way discrimination,** with depression included rather than only dementia.

SEE ALSO Slot IV - 94 screening assessment in delirium Slot IV - 92 definition, causes and management of delirium

SPRINT Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 96

Describe the criteria and causes of Amnesic Syndromes. [10]

10 | DELIRIUM AND AMNESTIC SYNDROMES | RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The criteria	What must be present, and what must be absent
4	Causes by mechanism	Six groups, each with the clue that points to it
2	Investigation	How the cause is actually established
1	The functional differential	What dissociative amnesia looks like instead

THE CRITERIA

Present: impairment of the ability to learn new material, or to recall previously learned material, severe enough to impair function. Absent: the clouded consciousness of delirium, the multi-domain decline of dementia, and impairment better explained by intoxication. Required: evidence of a cause capable of producing it.

MECHANISM	EXAMPLES	THE CLUE THAT POINTS TO IT
Nutritional	Korsakoff syndrome from thiamine deficiency	**Alcohol history, or a preceding Wernicke episode**
Vascular and hypoxic	Posterior cerebral artery infarction, thalamic stroke, subarachnoid haemorrhage, cardiac arrest, carbon monoxide	Abrupt onset with focal signs, or a documented hypoxic event
Infective	Herpes simplex encephalitis	**Fever and seizures, temporal changes on imaging**
Traumatic	Head injury, particularly with temporal contusion	The injury, and the length of post-traumatic amnesia
Surgical and epileptic	Bilateral temporal lobectomy, transient epileptic amnesia	The operation, or brief recurrent episodes on waking
Toxic and iatrogenic	Benzodiazepines, electroconvulsive therapy, alcohol blackouts	A clear temporal link to the agent, and reversibility

Amber marks the two that are both common and treatable, and are therefore the two an examiner most wants named early.

- **Investigation.** Informant history, imaging of the medial temporal and diencephalic structures, thiamine status, a metabolic screen, electroencephalography where epilepsy is possible, and neuropsychological testing to confirm the deficit is isolated.
- **The functional differential.** Dissociative amnesia typically loses autobiographical and personal identity information while new learning stays intact, which is the reverse of the organic pattern. Transient global amnesia is distinct again: an isolated episode of dense anterograde amnesia with repetitive questioning, resolving within a day.

WHAT EARNS THE MARKS

- **Criteria written as present, absent and required.** The exclusions carry as much credit as the inclusions.
- **Causes grouped by mechanism,** which makes the list defensible rather than remembered.
- **The reversed pattern in dissociative amnesia.** One sentence, and it separates the top band.

SEE ALSO Slot IV - 93 amnestic disorder Slot IV - 83 thiamine deficiency SPRINT

Day 34, Stroke, TBI, delirium & focal brain syndromes

AREA 15 OF 15 · P4-A04

Cognitive assessment and dementia management

4	2.8%	IV-97	IV-100
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 3

REVISION ORDER

The two cognitive rehabilitation slots together, they are one answer under two names and what the examiner wants is where each is used. Memantine next, a single-drug answer and the best-formed dose in this book. Successful ageing last, and it is the one slot here that is not written about a deficit.

WHAT IS IN THIS AREA

Cognitive rehabilitation and cognitive remedial therapy in two slots that ask for nearly the same body of material, the mechanisms and the interventions that support successful ageing, and memantine on its own.

IV - 97

Cognitive rehabilitation [10]

10 COGNITIVE ASSESSMENT AND DEMENTIA MANAGEMENT

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	**Definition**	Individual, goal-oriented, and aimed at function
3	**The three that get confused**	Stimulation, training, rehabilitation, kept apart
3	**How it is done**	Named learning methods, aids, and the environment
2	**Evidence and place**	What it changes, and what it honestly does not

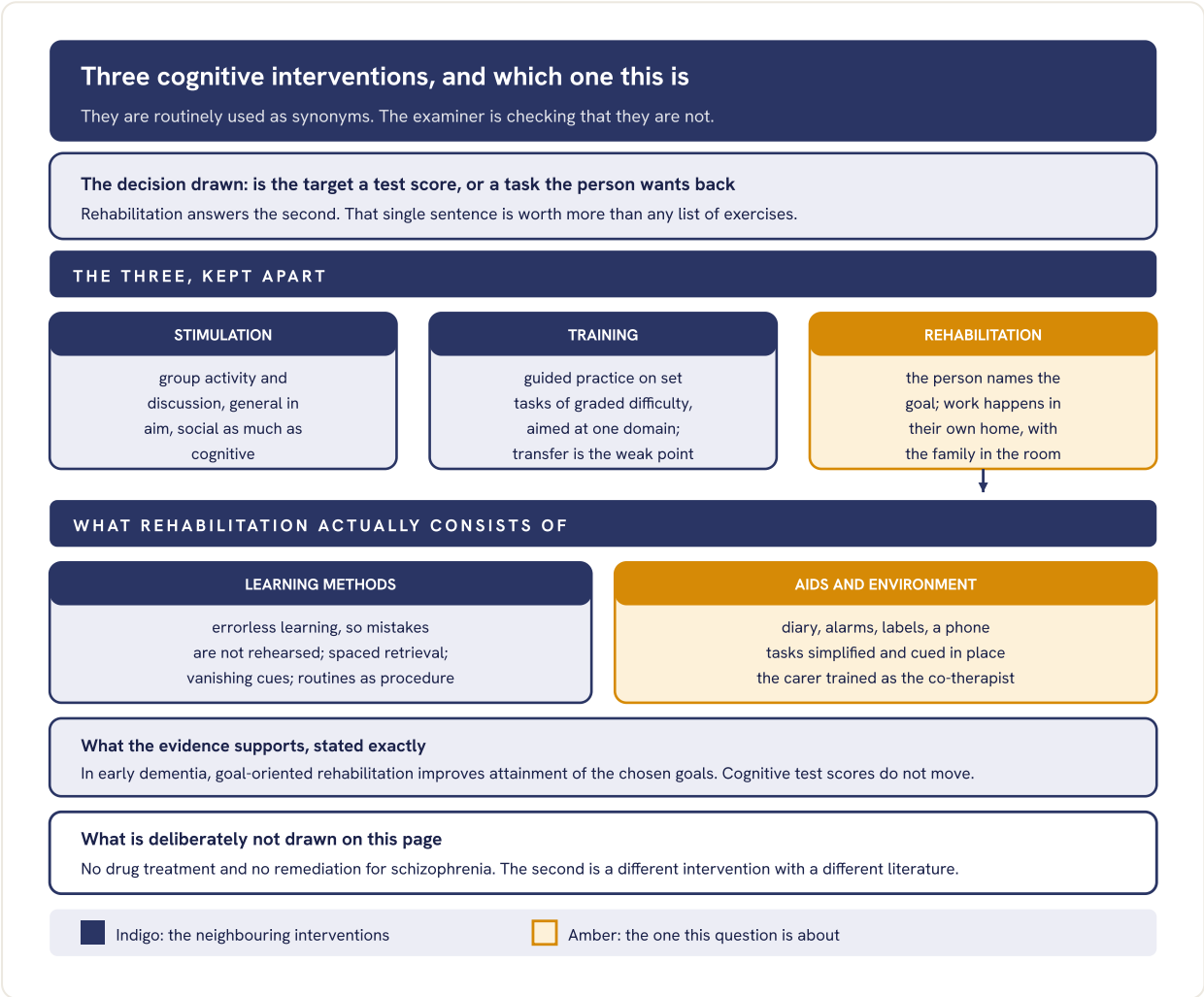


Fig 097.1 The three interventions separated at the top, then the content of rehabilitation itself below, with the evidence statement carried as a strip so the claim and its limit are read together.

- **The definition to write first.** An individualised, goal-oriented intervention in which the person and family choose everyday goals that matter to them, and strategies are built to reach those goals in the person's own setting.
- **Beyond dementia.** The same methods carry the strongest evidence after traumatic brain injury and stroke, where the deficit is stable rather than progressive.

PITFALL

Describing brain-training exercises. That is cognitive training, and its weakness is exactly the thing rehabilitation is built to solve: practice improves the practised task and rarely the life around it.

WHAT EARNS THE MARKS

- **Function named as the target,** not cognitive score, in the opening line.
- **The three interventions separated,** which is the discrimination the question exists to test.
- **Two named learning methods,** errorless learning and spaced retrieval among them.
- **The honest evidence statement,** goals improve and test scores do not.

SEE ALSO Slot IV - 99 cognitive remedial therapy Slot IV - 12 mild cognitive impairment **SPRINT** Day 32, Dementias & neurocognitive disorders

IV - 98

Explain the mechanism and interventions to enhance successful aging. [10]

10 | ASKED AT 15 MARKS | COGNITIVE ASSESSMENT AND DEMENTIA MANAGEMENT

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Define it**	The three-component model, named and attributed
3 **Mechanisms**	Reserve, plasticity, vascular and inflammatory ageing
3 **Interventions**	Ordered by the strength of the evidence behind them
2 **The critique**	Who a model built on absence of disease leaves out

THE DEFINITION, AND WHERE IT COMES FROM

Successful ageing is defined by three components together: low probability of disease and of disease-related disability, high physical and cognitive functional capacity, and active engagement with life through relationships and productive activity. Two related models sit alongside it: selective optimisation with compensation, and the shift toward emotionally meaningful goals as time horizons shorten.

MECHANISM	WHAT IT DOES	WHAT ACTS ON IT	EVIDENCE
Cognitive reserve	Buffers the same pathology behind better function	Education, occupation, lifelong learning, bilingualism	Observational
Vascular health	Protects white matter and perfusion	Blood pressure, glucose, lipids, smoking cessation	**Randomised support**
Neuroplasticity and muscle	Maintains function and slows sarcopenia	Aerobic and resistance exercise, protein intake	**Strongest single lever**
Sensory input	Keeps cognition and social contact fed	Hearing aids, cataract surgery	**Modifiable and neglected**
Engagement and role	Sustains mood, purpose and identity	Family role, work, volunteering, community	Consistent, observational

Amber cells are the discriminators. Naming the strength of evidence beside each lever is what separates a plan from a wish list.

- **Two clinical additions.** Treat depression, pain and insomnia, since each masquerades as ageing, and review the drug chart for anticholinergic and sedative burden that is quietly costing function.
- **The critique worth writing.** A model built on the absence of disease excludes most older people who have one, is culturally narrow, and ignores income and access. Adaptation within illness is a fairer standard.

WHAT EARNS THE MARKS

- **All three components of the model,** stated as required together rather than as alternatives.
- **Mechanisms before interventions,** because the stem asks for mechanism first.
- **Exercise and sensory correction named,** the two most often left out of the intervention list.
- **One honest criticism of the model.**

SEE ALSO Slot IV - 16 prevention of Alzheimer disease Slot IV - 97 cognitive rehabilitation **SPRINT**

Day 29, Consultation-liaison, geriatric, transcultural psychiatry & OSCE

IV - 99

Cognitive Remedial therapy. [10]

10 COGNITIVE ASSESSMENT AND DEMENTIA MANAGEMENT

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	Behavioural training, with transfer as the stated aim
2 The active ingredients	Four, and all four are needed for the name to apply
3 Where and how	Populations, the two approaches, and delivery
3 Evidence and limits	What improves, and what has to be added for it to count

THE DEFINITION, AND THE FOUR INGREDIENTS

A behavioural training intervention aimed at improving attention, memory, executive function, social cognition or metacognition, with durability and transfer to everyday functioning as explicit goals. The agreed ingredients are a trained therapist, repeated practice, the teaching of cognitive strategies, and procedures that engineer transfer into daily life.

DIMENSION	DRILL AND PRACTICE	STRATEGY BASED	IN PRACTICE
What it does	Repeated graded exercises to restore the process	Teaches ways around the deficit, then rehearses them	Most programmes combine both
Delivery	Often computer assisted, individual	Therapist led, individual or group	A therapist is required either way
Populations	Schizophrenia is the main one	Also anorexia nervosa, brain injury, mood disorder	Rigidity is the target in anorexia
Weak point	Gains stay near the trained task	Needs motivation and a therapist	Transfer is engineered, never assumed

Amber cells are the discriminators. The transfer problem is what the whole intervention is organised around.

- **The evidence, stated carefully.** In schizophrenia, meta-analyses show small to moderate gains in global cognition and smaller gains in functioning. Functional gain is substantially larger when remediation is delivered alongside psychiatric rehabilitation such as supported employment.
- **What it does not do.** It has little effect on positive symptoms, needs trained staff and repeated sessions, and does not replace medication or psychosocial care.

WHAT EARNS THE MARKS

- **Transfer named in the definition,** which is what distinguishes this from ordinary brain training.
- **All four ingredients listed,** the therapist among them.
- **The combination finding,** that remediation plus rehabilitation beats remediation alone.

SEE ALSO Slot IV - 97 cognitive rehabilitation Slot IV - 12 mild cognitive impairment

IV - 100

Memantine. [10]

10 COGNITIVE ASSESSMENT AND DEMENTIA MANAGEMENT

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	The receptor, and the kind of block it applies
2 Why that matters	Noise blocked, the signal for learning left alone
3 Where it belongs	Indications, the titration, and the combination
3 Safety and honest size	Adverse effects, the renal route, and the real benefit

THE MECHANISM, IN THE FORM THE MARKS ARE WRITTEN FOR

Memantine is an uncompetitive, voltage-dependent antagonist of the NMDA glutamate receptor with moderate affinity and a fast off-rate. It blocks the low-level tonic activation that drives excitotoxic noise, while still allowing the brief high-amplitude signal that learning depends on. That selectivity, not simple blockade, is the whole argument for the drug.

QUESTION	ANSWER	REASON	NOTE
Who is it for	Moderate to severe Alzheimer disease	Where the trial benefit sits	Not for mild disease
Who is it not for	Mild cognitive impairment, frontotemporal dementia	No demonstrated benefit	A common misprescription
How is it started	5 mg daily, raised weekly in 5 mg steps to 20 mg daily	Titration limits dizziness	Once daily forms exist
With what	May be combined with a cholinesterase inhibitor	Different mechanisms	No cholinergic overlap
How is it cleared	Largely renal, and not a cytochrome P450 substrate	Few drug interactions	Reduce dose in renal failure

Amber cells are the discriminators. Where a drug does not belong is as markable as where it does.

- **Tolerability.** Dizziness, headache, constipation, somnolence and confusion, occasionally hallucinations. It has none of the cholinergic gastrointestinal effects and none of the bradycardia, which is why it suits patients who cannot take a cholinesterase inhibitor.
- **The honest size of the benefit.** Symptomatic and modest: cognition, daily function and agitation shift a little. It does not modify the disease, and saying so is worth a mark rather than costing one.

WHAT EARNS THE MARKS

- **Uncompetitive and voltage-dependent stated,** not simply NMDA antagonist.
- **The tonic and phasic distinction,** which explains why blocking the receptor does not abolish learning.
- **Severity band named,** with the dementias it does not help.
- **Benefit described as symptomatic and modest.**

SEE ALSO Slot IV - 17 cholinesterase inhibitors Slot IV - 13 frontotemporal dementia SPRINT Day 32, Dementias & neurocognitive disorders

PAPER IV APPENDIX, PAGE ONE OF TWO

Ten sets, ten sittings: Paper IV

The same hundred Paper IV questions, dealt into ten papers of ten. Each cell gives the slot and the page it is answered on.

Read down a row to sit a paper. The deal is not random: the hundred are ranked by score and dealt one question from each rank decile into each set, so every set holds one of the ten strongest, one of the ten weakest, and one from each band between. That is why the sets are comparable to each other and why none of them is an easy paper.

SET	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10
IV-S1	IV-12 p 423	IV-60 p 483	IV-05 p 414	IV-20 p 432	IV-66 p 491	IV-30 p 444	IV-44 p 462	IV-74 p 502	IV-52 p 472	IV-96 p 532
IV-S2	IV-13 p 424	IV-82 p 513	IV-06 p 416	IV-07 p 417	IV-09 p 419	IV-42 p 460	IV-45 p 463	IV-57 p 479	IV-71 p 497	IV-95 p 531
IV-S3	IV-01 p 410	IV-72 p 499	IV-26 p 440	IV-87 p 520	IV-34 p 450	IV-77 p 506	IV-64 p 488	IV-56 p 478	IV-58 p 480	IV-99 p 537
IV-S4	IV-14 p 426	IV-48 p 467	IV-31 p 446	IV-19 p 431	IV-67 p 492	IV-29 p 443	IV-65 p 489	IV-89 p 522	IV-93 p 529	IV-98 p 536
IV-S5	IV-15 p 427	IV-97 p 534	IV-17 p 429	IV-54 p 475	IV-10 p 420	IV-28 p 442	IV-37 p 453	IV-51 p 471	IV-53 p 473	IV-94 p 530
IV-S6	IV-39 p 456	IV-16 p 428	IV-18 p 430	IV-32 p 447	IV-11 p 421	IV-88 p 521	IV-68 p 493	IV-69 p 495	IV-59 p 481	IV-81 p 511
IV-S7	IV-02 p 411	IV-04 p 413	IV-24 p 437	IV-33 p 449	IV-35 p 451	IV-63 p 487	IV-78 p 508	IV-90 p 523	IV-38 p 454	IV-80 p 510
IV-S8	IV-40 p 458	IV-23 p 436	IV-25 p 438	IV-84 p 516	IV-41 p 459	IV-21 p 433	IV-79 p 509	IV-50 p 470	IV-85 p 517	IV-86 p 518
IV-S9	IV-92 p 527	IV-47 p 466	IV-61 p 485	IV-27 p 441	IV-08 p 418	IV-36 p 452	IV-55 p 476	IV-46 p 464	IV-70 p 496	IV-100 p 538
IV-S10	IV-22 p 435	IV-03 p 412	IV-62 p 486	IV-83 p 515	IV-49 p 468	IV-73 p 500	IV-43 p 461	IV-75 p 503	IV-76 p 504	IV-91 p 525

No set repeats a question, a merged variant of a question, or a near-duplicate, and no set takes more than three questions from any one area.

PAPER IV APPENDIX, PAGE TWO OF TWO

How to sit one of these

SET	MARKS	MEAN SCORE	AREAS	ANCHOR OR HIGH	HEAVIEST AREAS IN THE SET
IV-S1	100	0.422	9	3	Dementias: types 2; Recent advances 1; Community 1
IV-S2	100	0.418	7	2	Community 3; Stroke 2; Dementias: types 1
IV-S3	100	0.415	9	3	Endocrine 2; Community 1; Psychopharmacology: principles 1
IV-S4	100	0.402	9	3	Dementias: types 2; Movement 1; Clinical neurology 1
IV-S5	100	0.395	8	5	Dementias: types 2; Movement 2; Cognitive assessment 1
IV-S6	100	0.394	8	2	Dementias: types 2; Drug emergencies 2; Stroke 1
IV-S7	100	0.394	6	3	Clinical neurology 3; Community 2; Epilepsy 2
IV-S8	100	0.399	6	5	Nutritional 3; Stroke 2; Consultation-liaison 2
IV-S9	100	0.392	10	3	Delirium 1; Movement 1; Recent advances 1
IV-S10	100	0.390	8	4	Psychopharmacology: principles 3; Consultation-liaison 1; Community 1

Mean score is the selection score of the ten questions in that set, on the same scale used across all four books. The spread between the strongest and the weakest set is small by construction.

Three hours, ten questions, one hundred marks. That is the shape of the real paper, so it is the shape of these. Eighteen minutes a question leaves you twenty minutes at the end, which is roughly what you will have and roughly what you will need.

Write the skeleton first, for all ten. Spend the first twenty minutes writing nothing but mark splits and section headings across all ten answers. The map on each page shows what that skeleton should look like, so you can mark your own against it before you have written a line of content. Most marks lost in this examination are lost to allocation, not to ignorance.

Then mark yourself against the anchors. Every map ends with three or four things an examiner ticks. Those are the marking scheme, as close as this book gets to one. Count how many you hit before you read the page, because reading it first will convince you that you knew it.

Sit them in order, or sit them backwards. Set one holds the strongest question in the book and set ten the weakest, but the decile deal keeps the mean scores close, so any set is a fair paper. In order simply means the first one you sit is the one most like the paper you will get.

What a set is not. It is not a mock paper from any board, it is not a leak, and it is not a prediction. It is ten questions a board set, arranged so that ten of them look like a paper. Sit five and you will have written a hundred answers in the shape the examination wants, which is the only preparation that has ever reliably worked.

Index

One index for all four papers. Every entry points at the map that answers it. A subject asked in several places carries up to three pages, and the one in bold is the map whose question is about it.

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CREDITS

Who made this, and from what

Dr. Wilfred Dsouza	Counting, area weighting, selection, and the answer maps. Lead author of the Weave 100 series.
Dr. Niharika Reddy	Clinical review across the four papers: the personality, developmental and cross-cultural areas and the psychometry maps in Paper I; the psychotherapy, personality and trauma areas and the suicide and self-harm maps in Paper II; the child, perinatal and women's mental health areas and the ethics and consent maps in Paper III; and the community and public mental health area, the consultation-liaison maps and the dementia and delirium management pages in Paper IV. Co-author.

The record. Every question in this book was read off a paper set by DNB, KNRUHS, KUHS, MUHS, NTRUHS, RGUHS, TN Dr. MGR Medical University or WBUHS. Where a paper reached us as a mirror, an archive or a transcription rather than an official file, that session carries a lower weight in the count, and the weightage chapter says by how much.

The residents. Papers, corrections and photographs of question papers came from residents across several states who had no reason to send them except that somebody else would need them. This book is mostly their work.

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