WEAVE 100 - PAPER IV

MD Psychiatry theory. DNB revision fits too.

Brain, Drugs and the Community

One hundred ten-mark questions, solved as answer maps

Eight examining boards. Seventy-eight sittings. A count of where the marks have actually gone in the paper that holds the brain a psychiatrist is called to, the drugs a psychiatrist prescribes, and the population a psychiatrist answers to: epilepsy, the dementias, delirium, stroke and head injury, the movement disorders, the endocrine and infective presentations, the medical wards, and community and public mental health. Fifteen areas, weighted by what was asked, then one 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 - PAPER IV

Brain, Drugs and the Community

One hundred ten-mark questions, solved as answer maps

TITLE

Weave 100, Paper IV: Brain, Drugs and the Community

Book four of Weave 100, one hundred solved ten-mark questions for each paper of the MD psychiatry written examination.

WRITTEN BY

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EDITION

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

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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.

Contents

FRONT MATTER

How to use this book 6
Where the marks have gone 7
How the hundred were picked 8
What this book cannot promise 9

THE HUNDRED, IN WEIGHT ORDER

01 **Community and public mental health** IV-01 to IV-11 · 11 slots · 13.3% 10
02 **Dementias: types and differential** IV-12 to IV-21 · 10 slots · 11.4% 23
03 **Consultation-liaison and systemic medicine psychiatry** IV-22 to IV-30 · 9 slots · 10.1% 35
04 **Clinical neurology, headache and sleep neurology** IV-31 to IV-38 · 8 slots · 8.0% 46
05 **Stroke, TBI and focal brain syndromes** IV-39 to IV-46 · 8 slots · 7.9% 56
06 **Movement and degenerative disorders in psychiatry** IV-47 to IV-53 · 7 slots · 6.7% 66
07 **Endocrine and metabolic psychiatry** IV-54 to IV-59 · 6 slots · 6.2% 75
08 **Recent advances and landmark studies** IV-60 to IV-65 · 6 slots · 5.9% 83
09 **Drug emergencies, interactions, TRD and neurostimulation** IV-66 to IV-71 · 6 slots · 5.6% 91
10 **Psychopharmacology: principles and drug classes** IV-72 to IV-76 · 5 slots · 5.0% 99
11 **Epilepsy and psychiatry** IV-77 to IV-81 · 5 slots · 4.9% 106
12 **Nutritional, toxic and metabolic encephalopathies** IV-82 to IV-86 · 5 slots · 4.2% 113
13 **Neuro-infective and autoimmune neuropsychiatry** IV-87 to IV-91 · 5 slots · 4.0% 120
14 **Delirium and amnestic syndromes** IV-92 to IV-96 · 5 slots · 3.9% 127
15 **Cognitive assessment and dementia management** IV-97 to IV-100 · 4 slots · 2.8% 134

BACK MATTER

Ten sets, ten sittings 140
Index 142
Credits and colophon 148

HOW TO USE THIS BOOK

One question, one page

Every page after the weightage chapter is one question and its answer map. Nothing continues onto a second page, so a map can be read in the time you have rather than the time it deserves. 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 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 at the back: each is ten questions and one hundred marks, 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 gives a count against a stated denominator. If it says three of seventy-eight sittings, that is three sittings out of the seventy-eight this book counted, and the boards are listed by name so you can check.

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

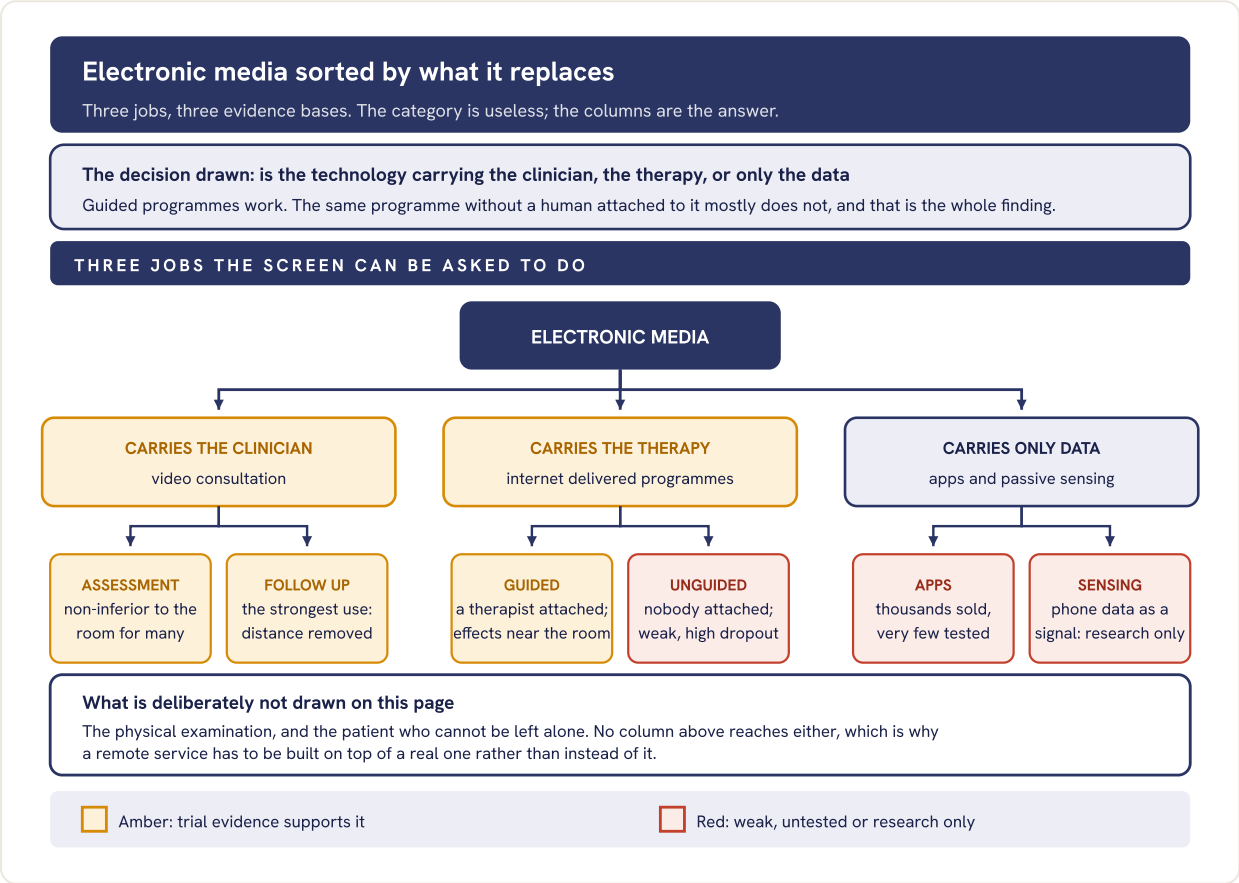

Fig 005.1 Guided and unguided sit in adjacent boxes and in different colours, because that single distinction carries more of the evidence than any other line on the page.

- **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

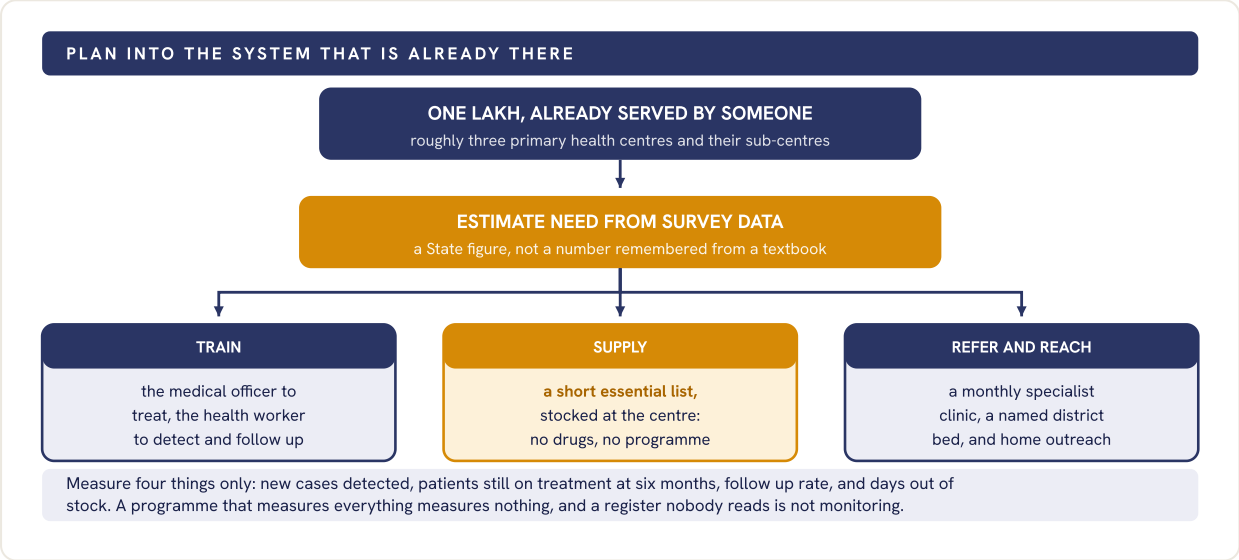

Fig 007.1 Supply sits in amber because a trained officer with an empty shelf produces referrals, not treatment, and that is the commonest way a district plan fails.

- **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

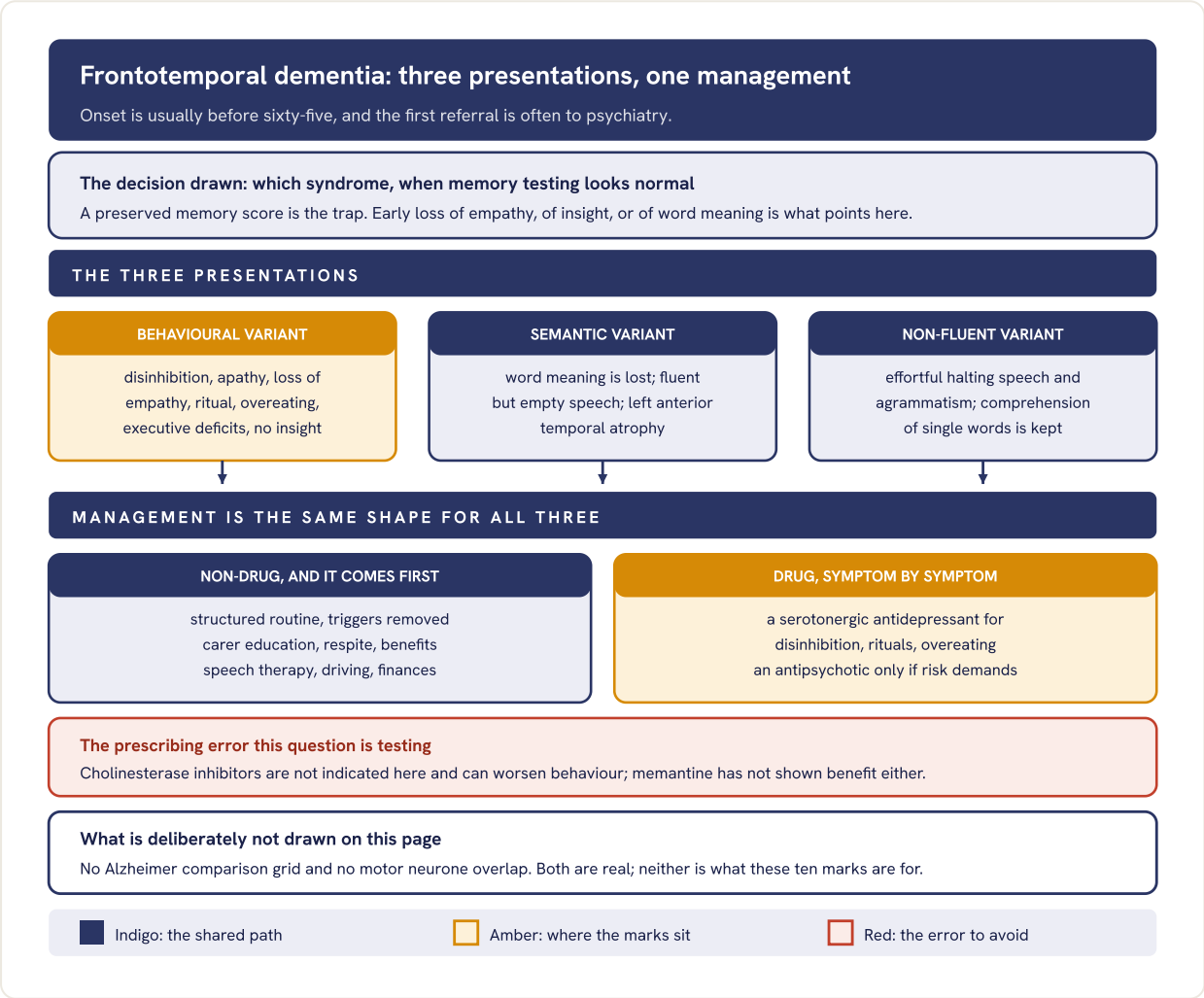

Fig 013.1 The three clinical presentations above, the single management shape below, and the prescribing error carried as a red strip because it is the line that separates this dementia from Alzheimer disease in practice.

- **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

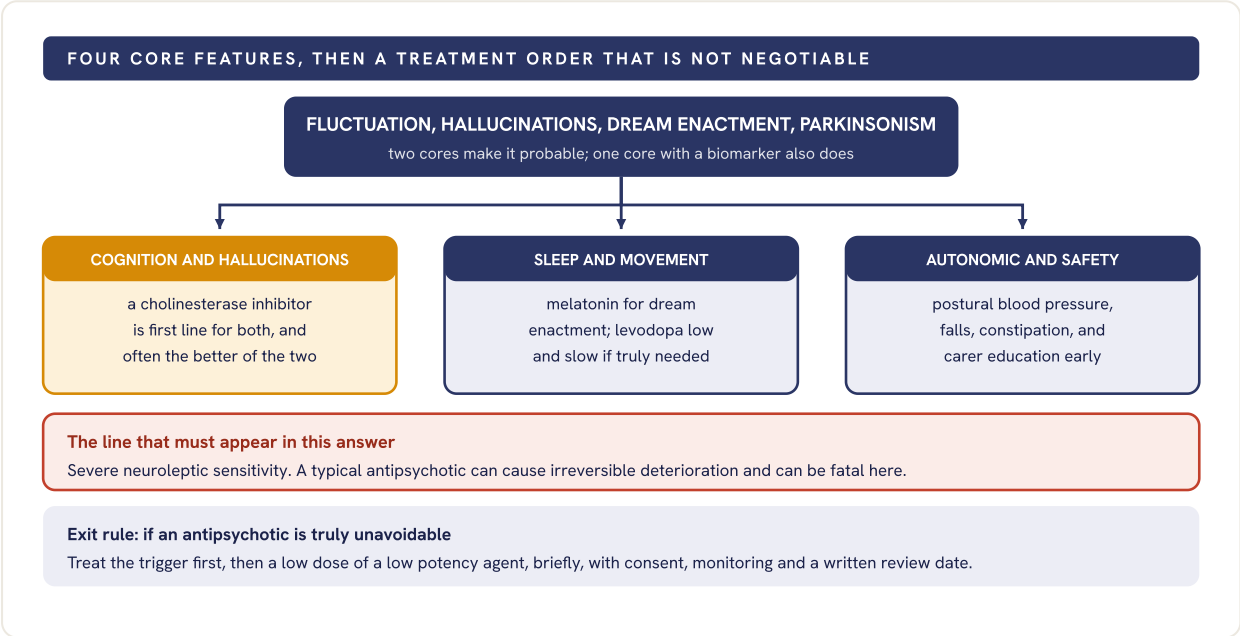

Fig 014.1 The four core features feeding three treatment strands, with neuroleptic sensitivity carried as a red strip because prescribing an antipsychotic here is the error that kills.

- **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

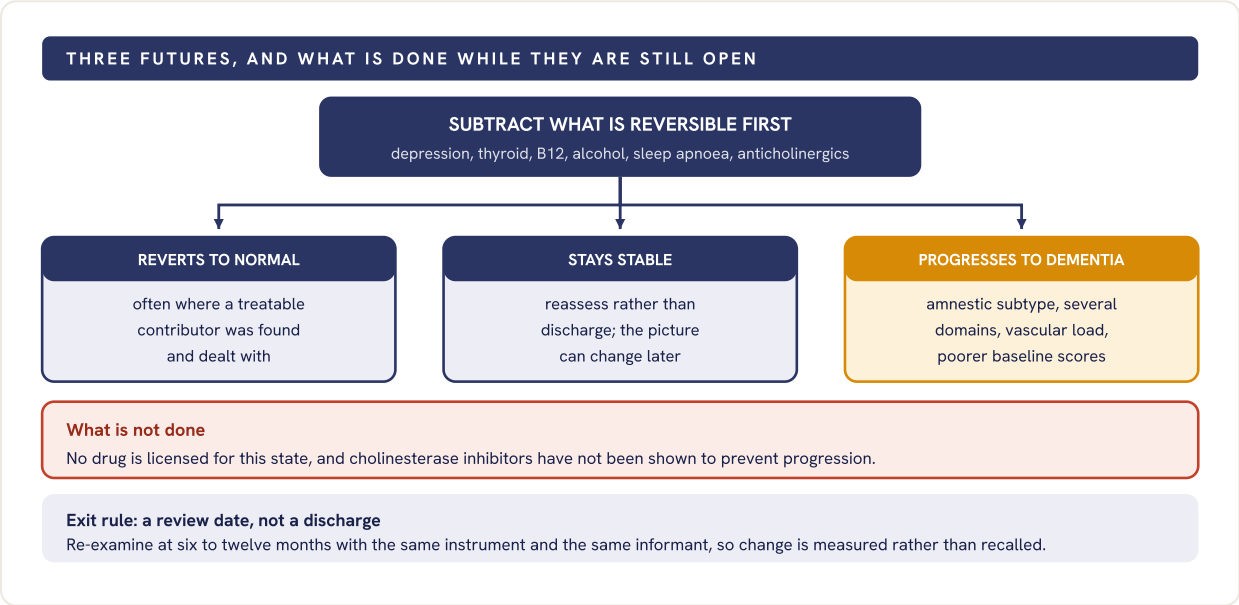

Fig 015.1 Reversible contributors are subtracted before the prognosis is discussed at all, and the three futures are then set against the features that predict each one.

- **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 acetylcholinesterase; 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

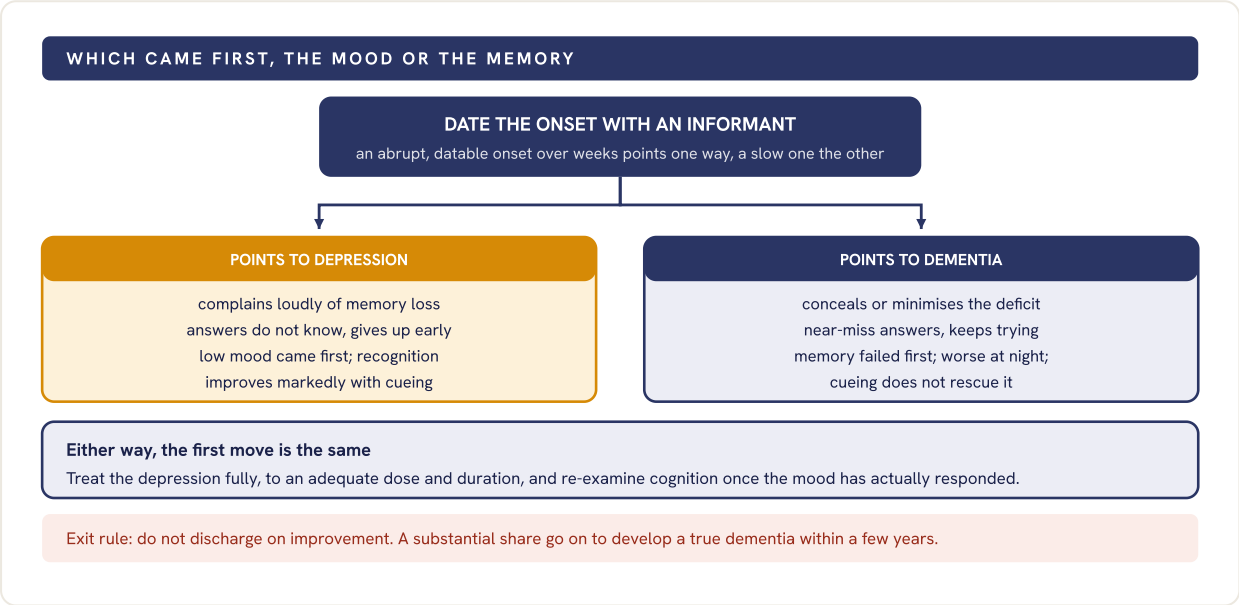

Fig 018.1 The two columns of the classical contrast, converging on one first move, with the follow-up rule carried along the foot because that is the mark most answers never reach.

- **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

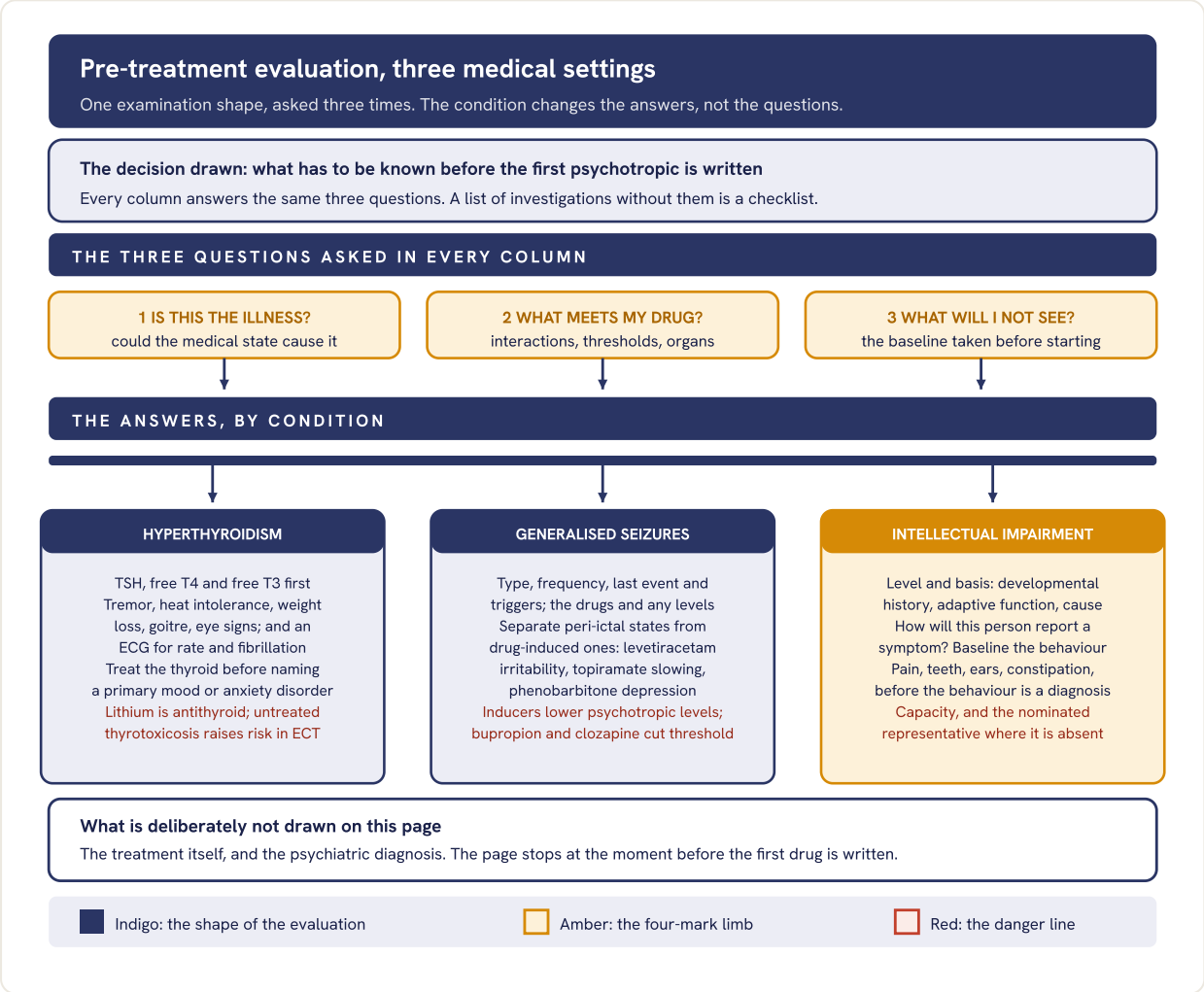

Fig 025.1 The same three questions run along one spine into all three columns, so the answer is one method applied three times, not three checklists.

- **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

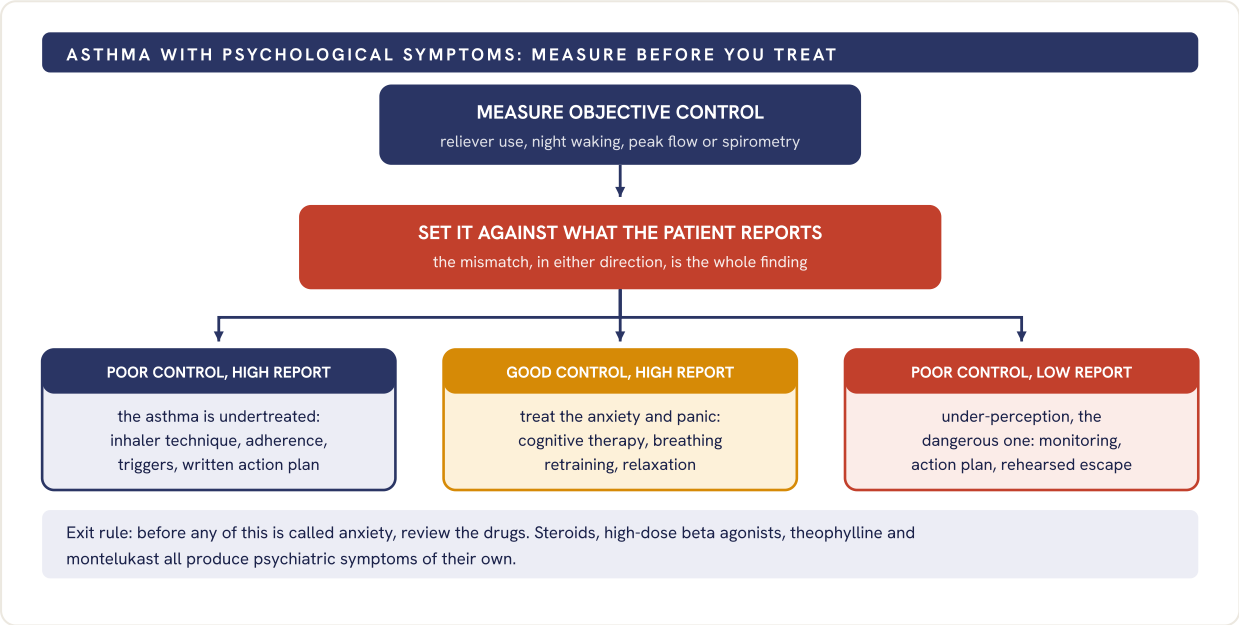

Fig 029.1 The intervention is chosen by the direction of the mismatch between measured control and reported symptoms, which is why nothing is prescribed before the measurement. Indigo optimises the asthma, amber treats the anxiety, red is danger.

- **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

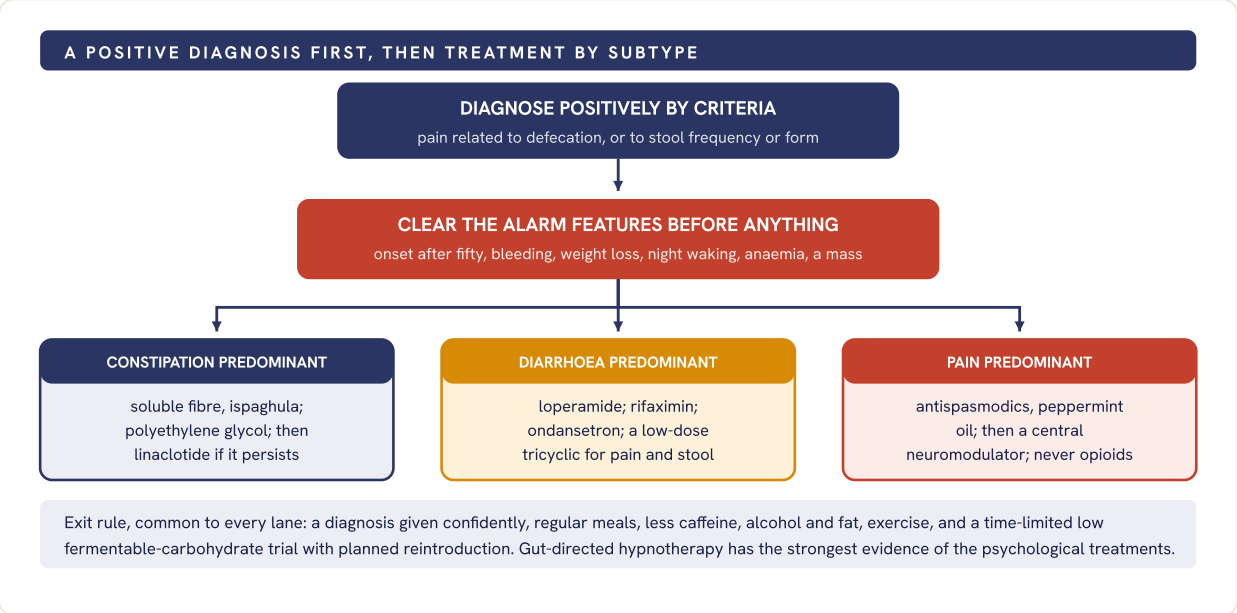

Fig 030.1 The alarm features sit between the diagnosis and every treatment lane, because they are the one thing that removes this diagnosis rather than modifying it. Indigo is structure, amber the fullest lane, red exclusion and pain.

- **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

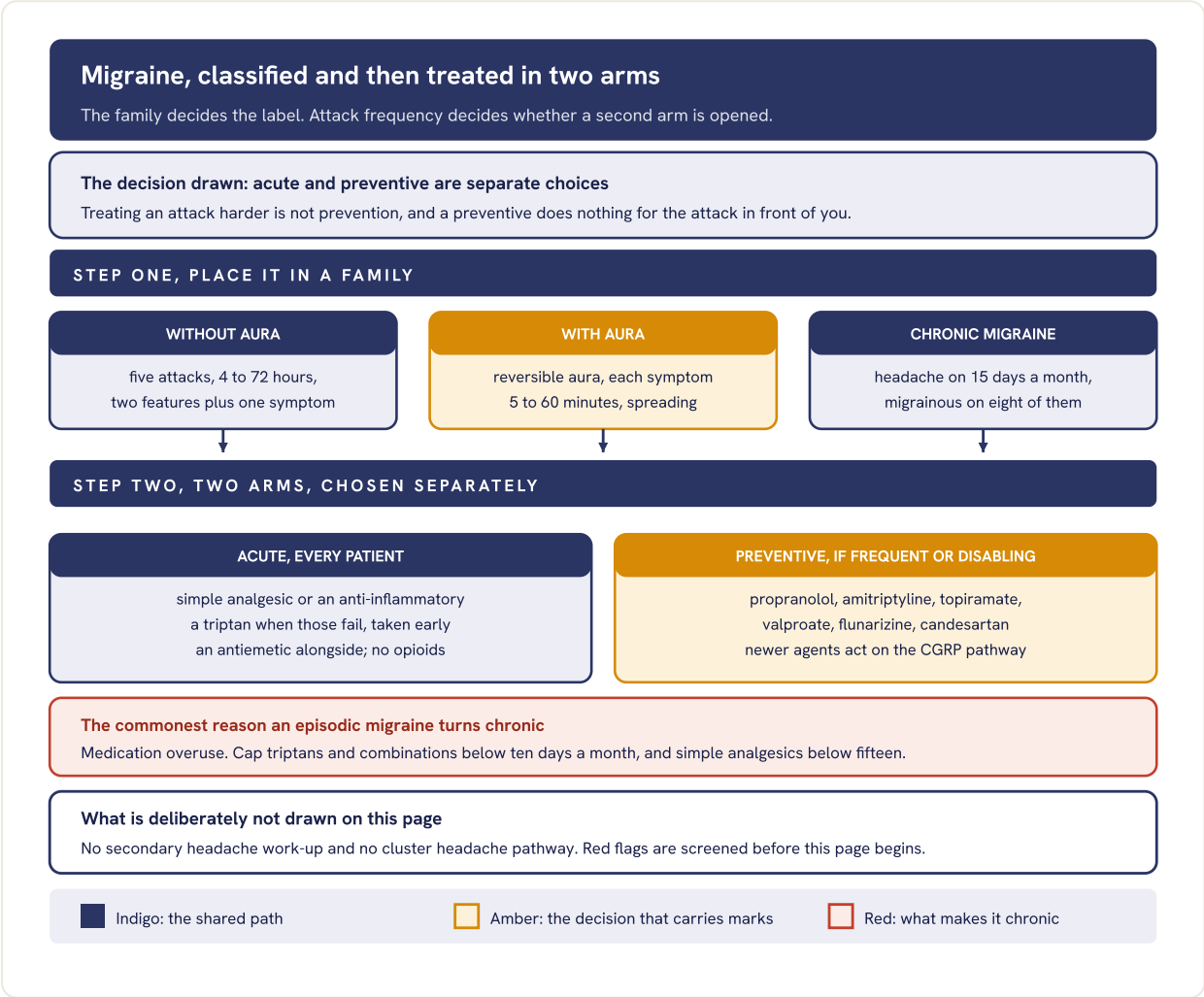

Fig 032.1 The three families most often asked about, then the two treatment arms kept apart, with medication overuse carried as a red strip because it is the modifiable driver of chronic migraine.

- **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

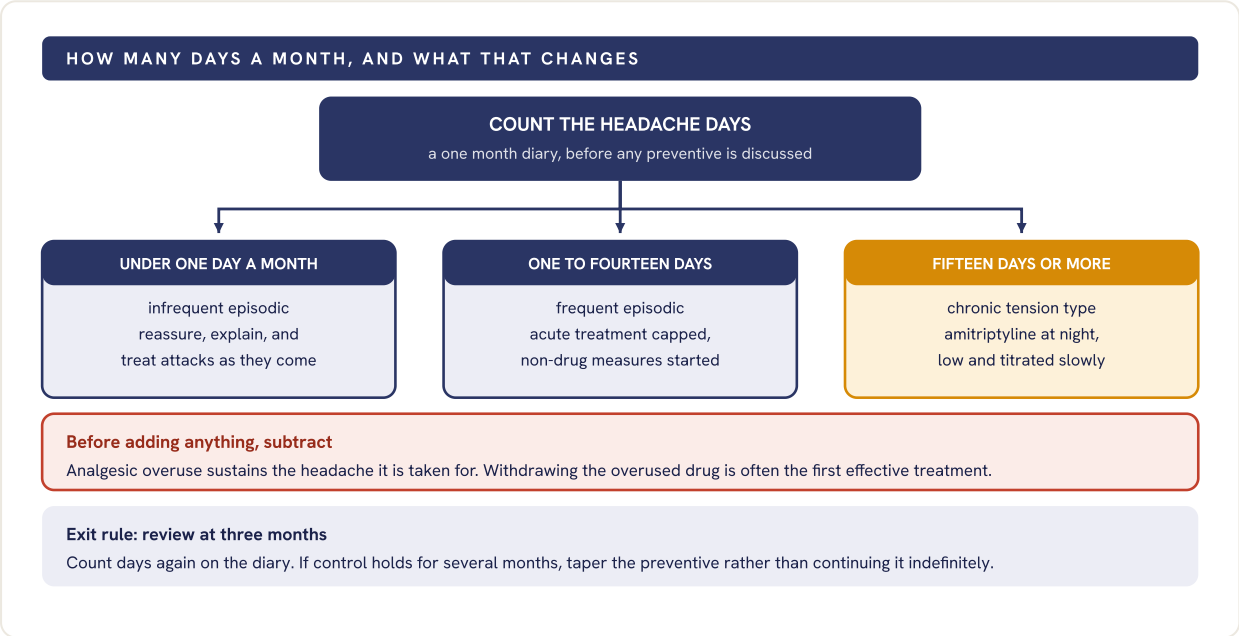

Fig 033.1 Headache days a month set the grade, and the grade decides whether a preventive is opened at all, with analgesic withdrawal placed before any addition.

- **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

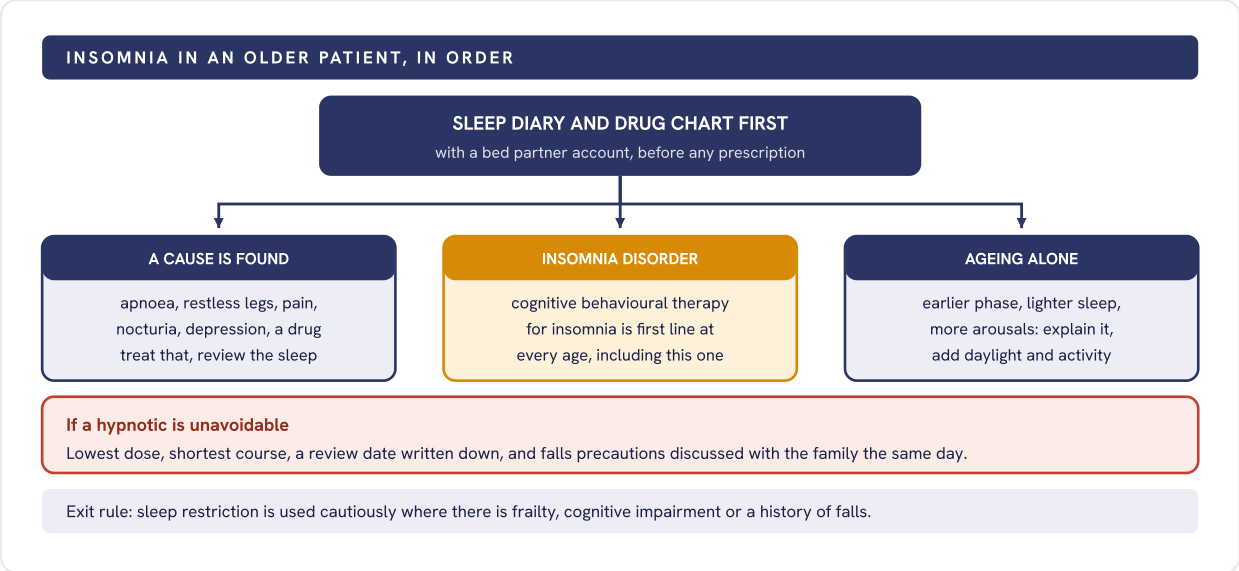

Fig 034.1 The diary and the drug chart decide which of three routes the case takes, with the hypnotic placed after all three rather than inside any of them.

- **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

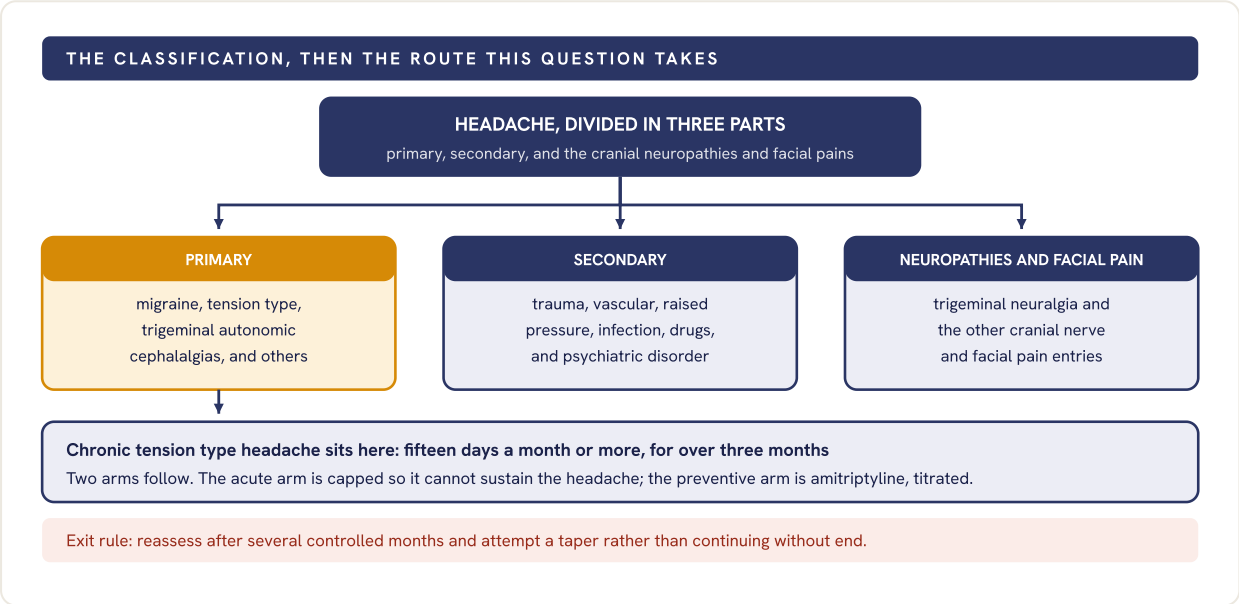

Fig 035.1 The three parts of the classification, with the route this question takes highlighted, so the four classification marks and the six treatment marks sit on one picture.

- **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

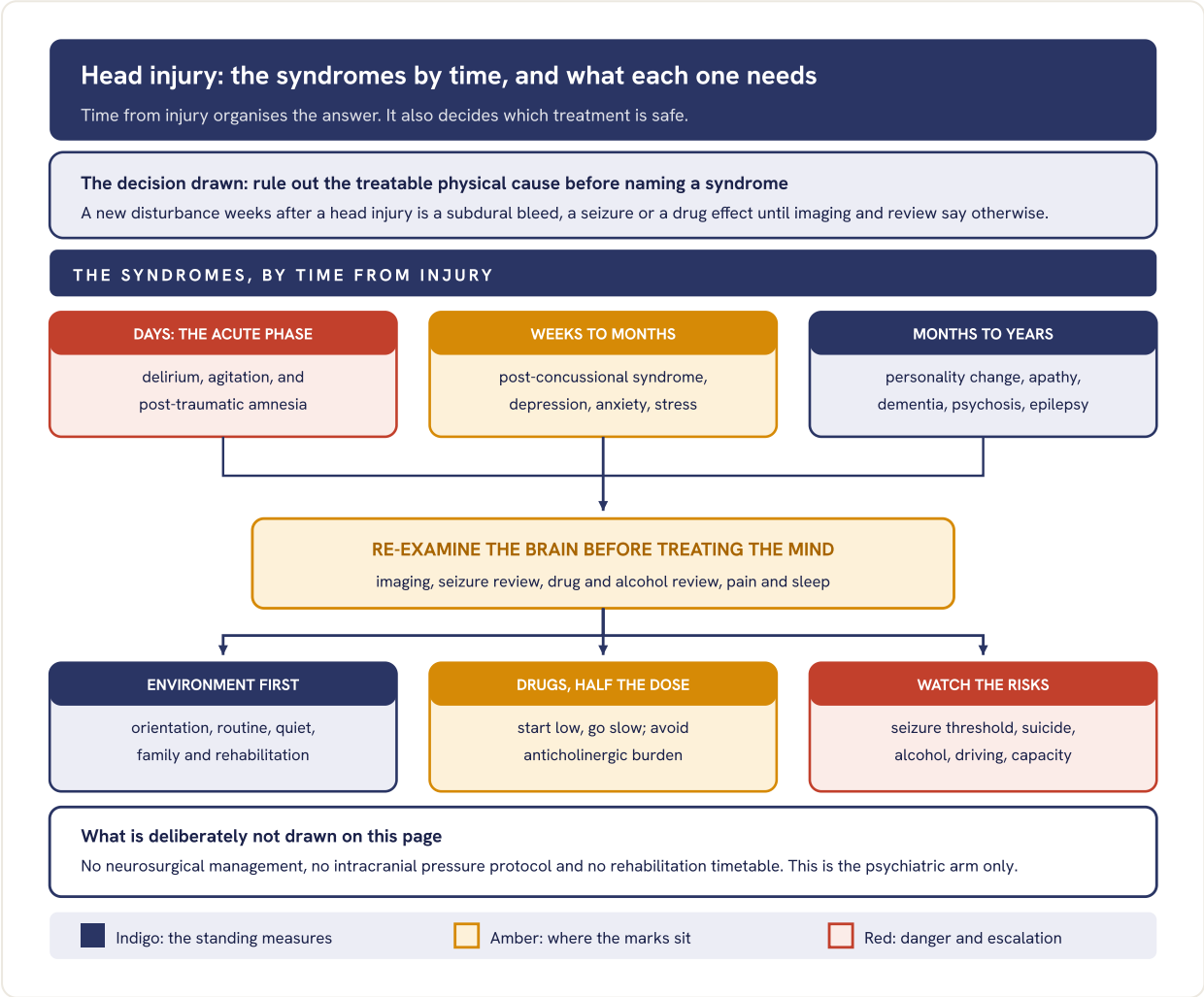

Fig 039.1 The syndromes grouped by time from injury, the physical review that must precede treatment, and the three arms of management that follow.

- **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

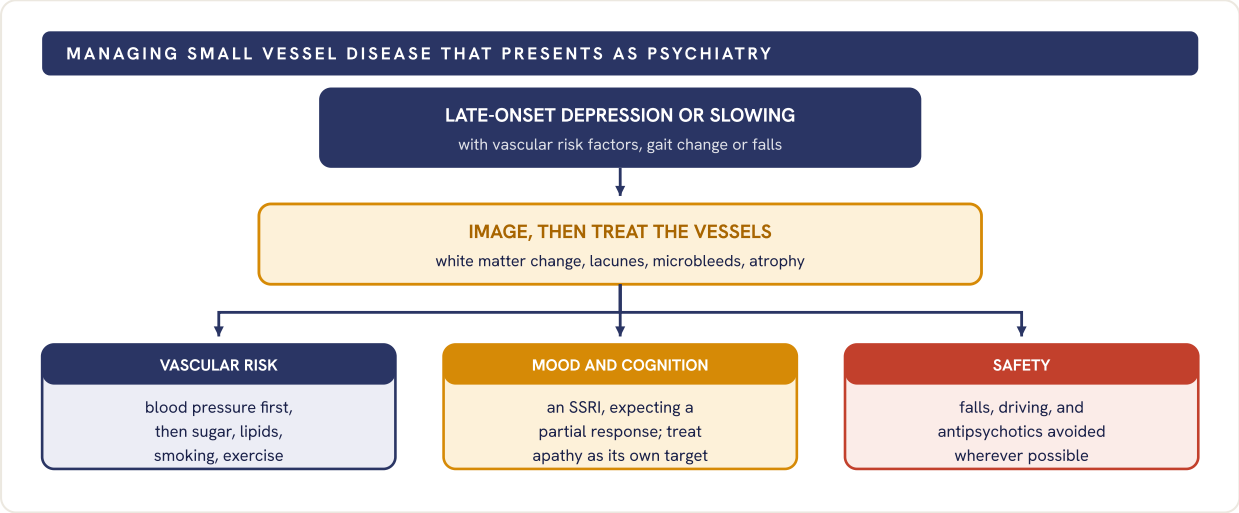

Fig 041.1 One presentation, one confirmatory step, and three arms of management with the vessels treated before the mood.

- **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

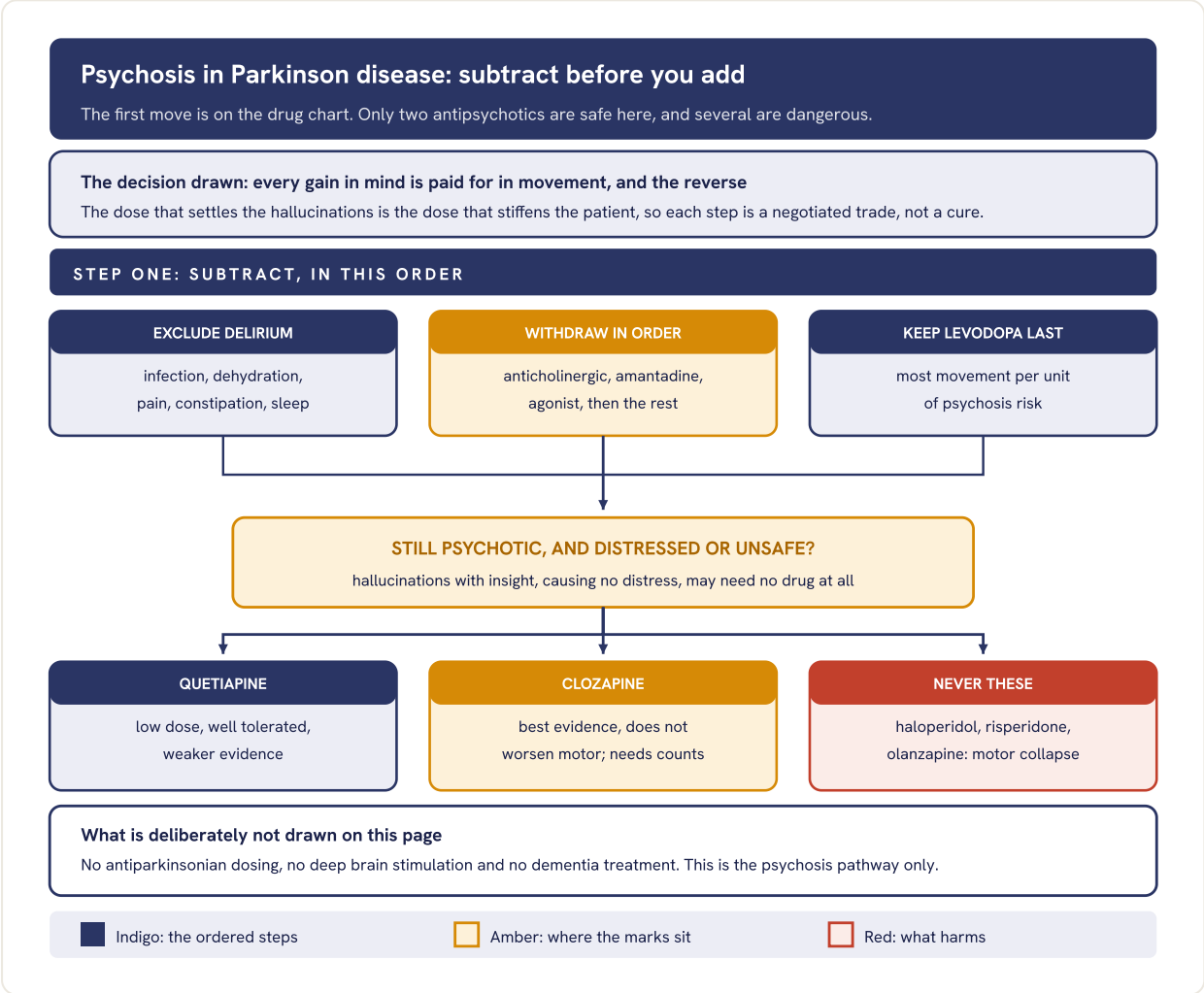

Fig 049.1 The withdrawal order that treats psychosis before any antipsychotic is added, the threshold for treating at all, and the two agents that are safe when it is crossed.

- **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.

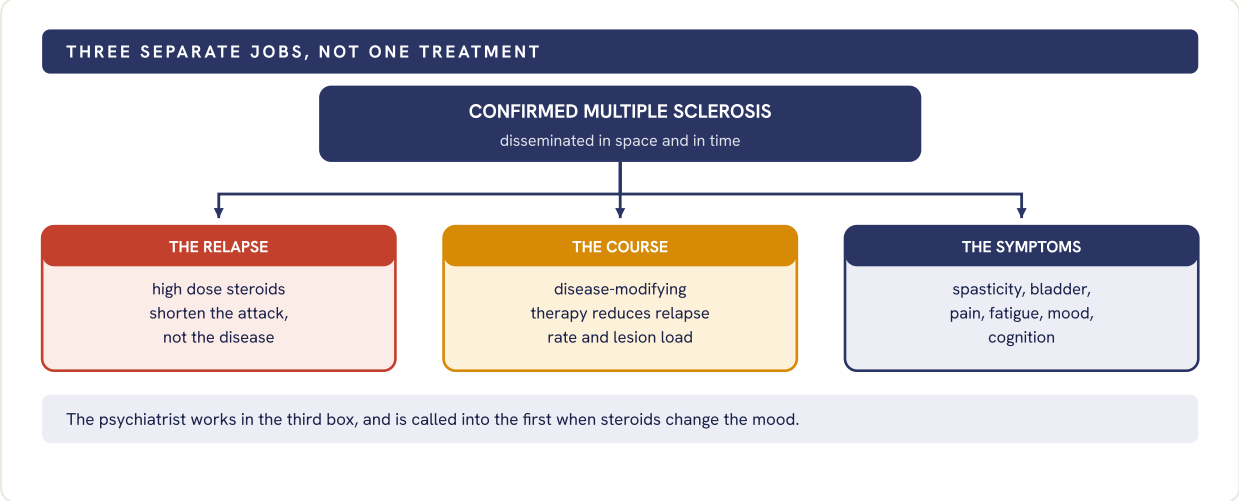

Fig 051.1 Management separated into the three jobs it actually is.

- **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.

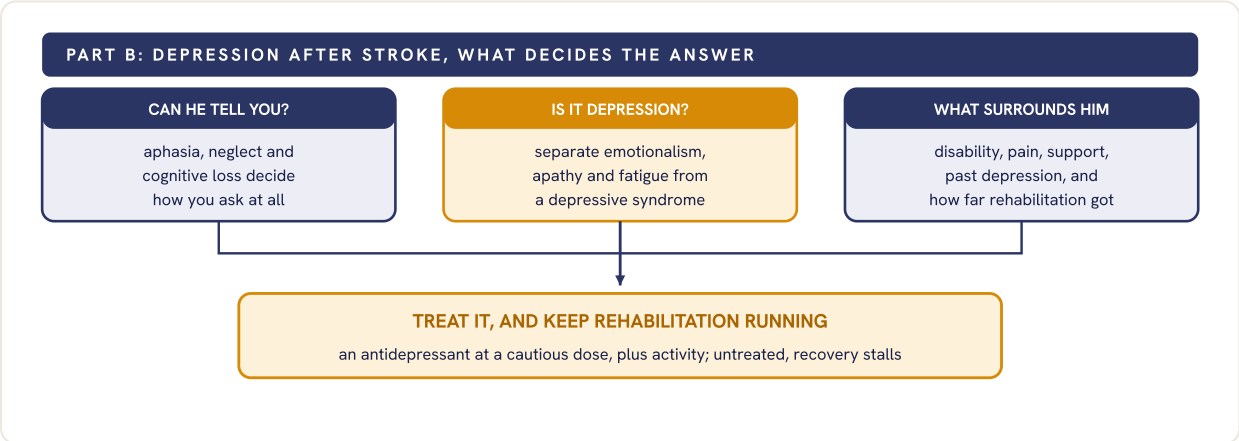

Fig 052.1 What decides the assessment, and treatment that runs alongside rehabilitation.

- **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.

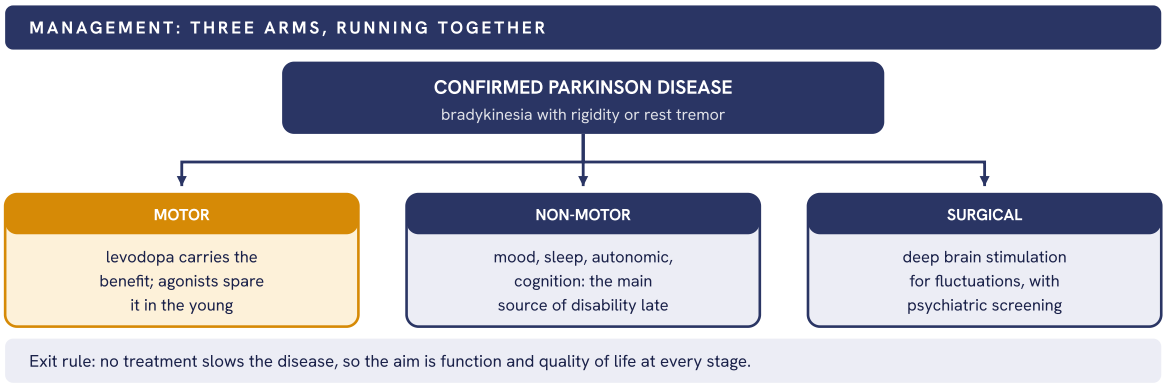

Fig 053.1 The three arms of management, with the non-motor arm marked as the main source of disability once the disease is established.

- **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

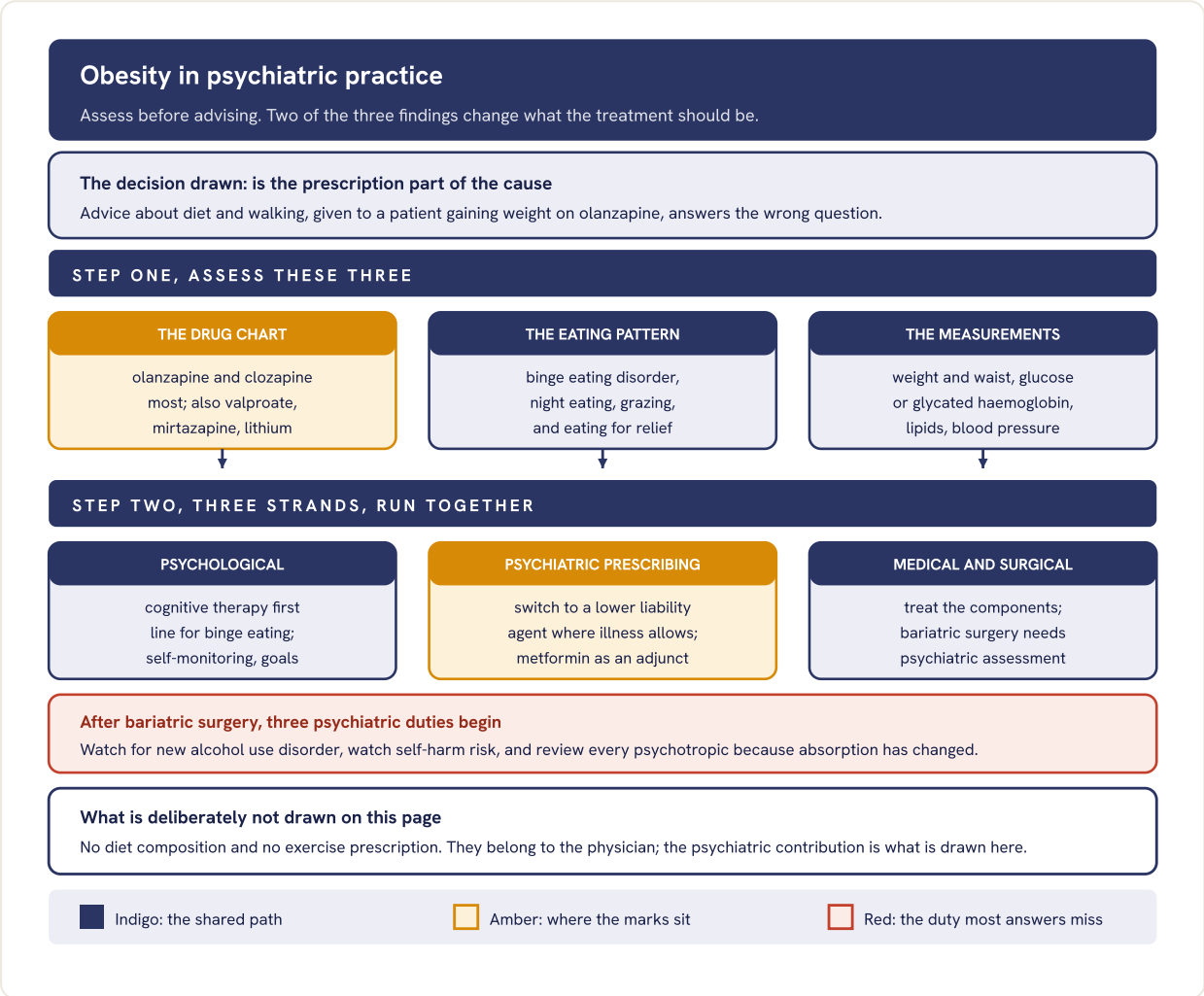

Fig 055.1 Three things assessed before anything is advised, then three strands of management run together, with the post-surgical duties carried as a red strip because they fall to psychiatry specifically.

- **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

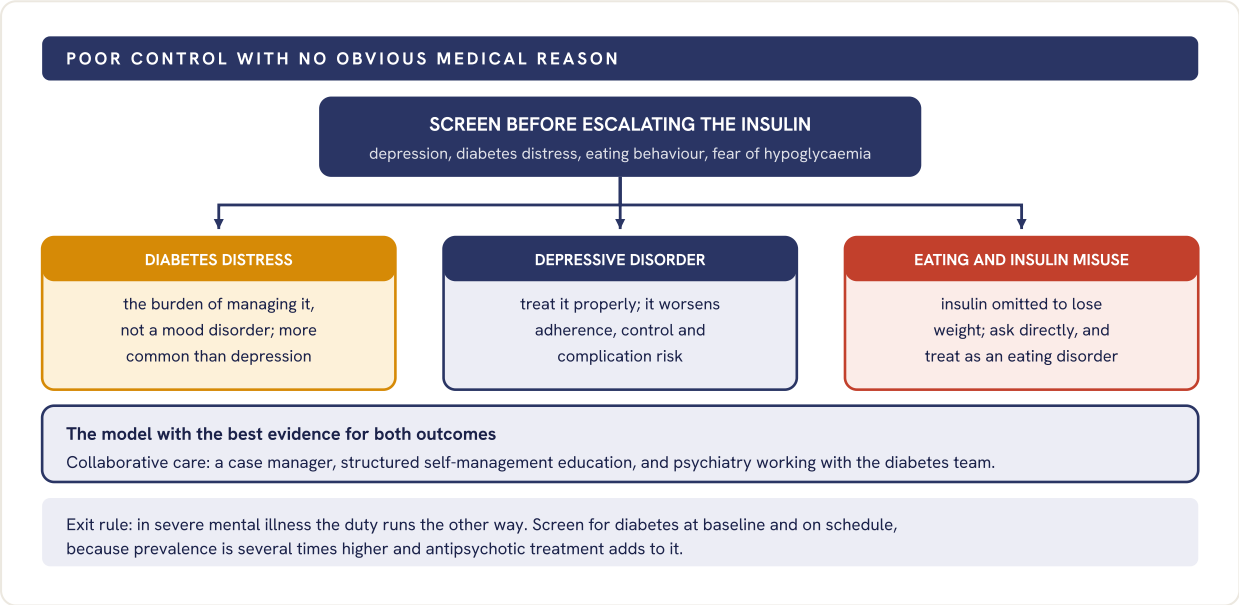

Fig 058.1 Screening placed before any escalation of the diabetic treatment, with distress separated from depression because they need different interventions and only one of them needs a drug.

- **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

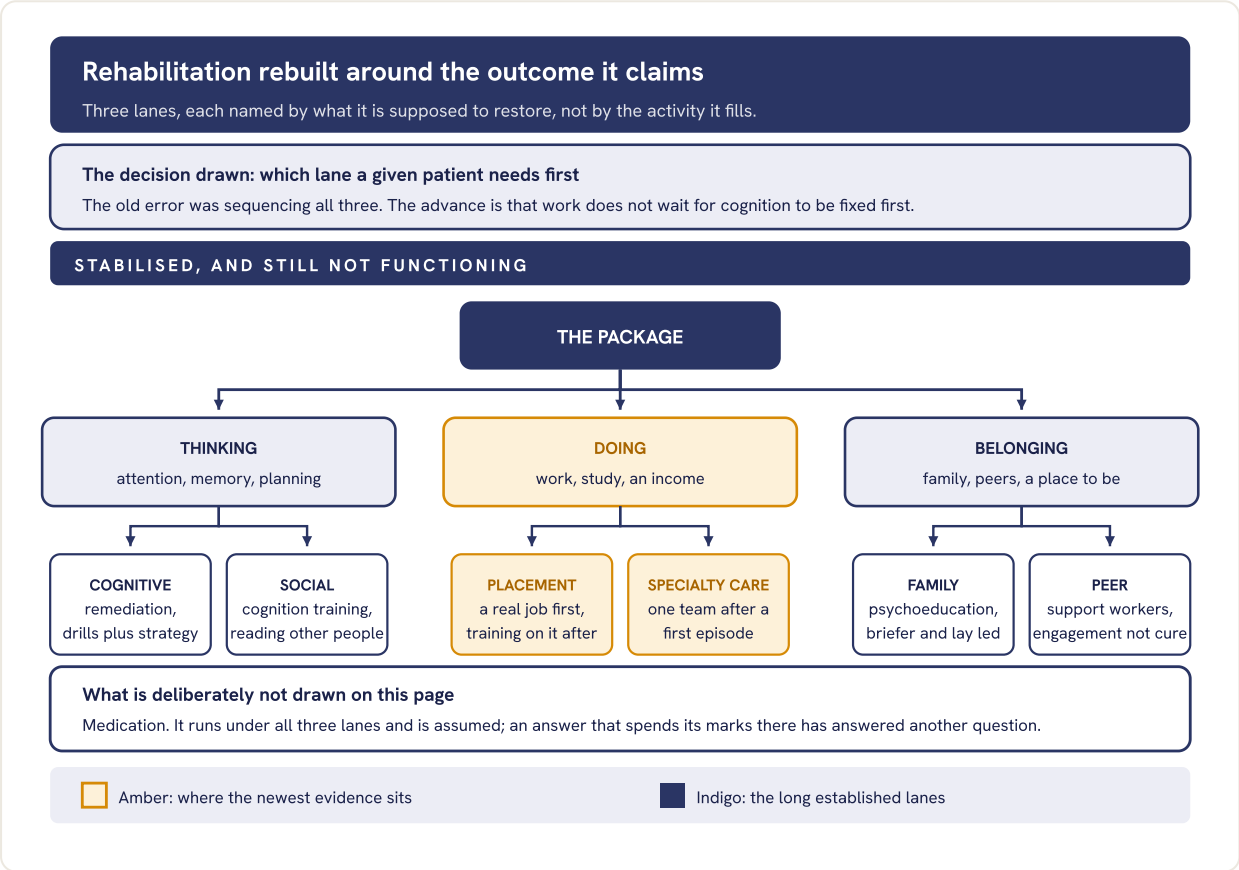

Fig 060.1 The package sorted by the outcome each lane claims, which is what makes place-then-train visible as a change of principle rather than a change of timetable.

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

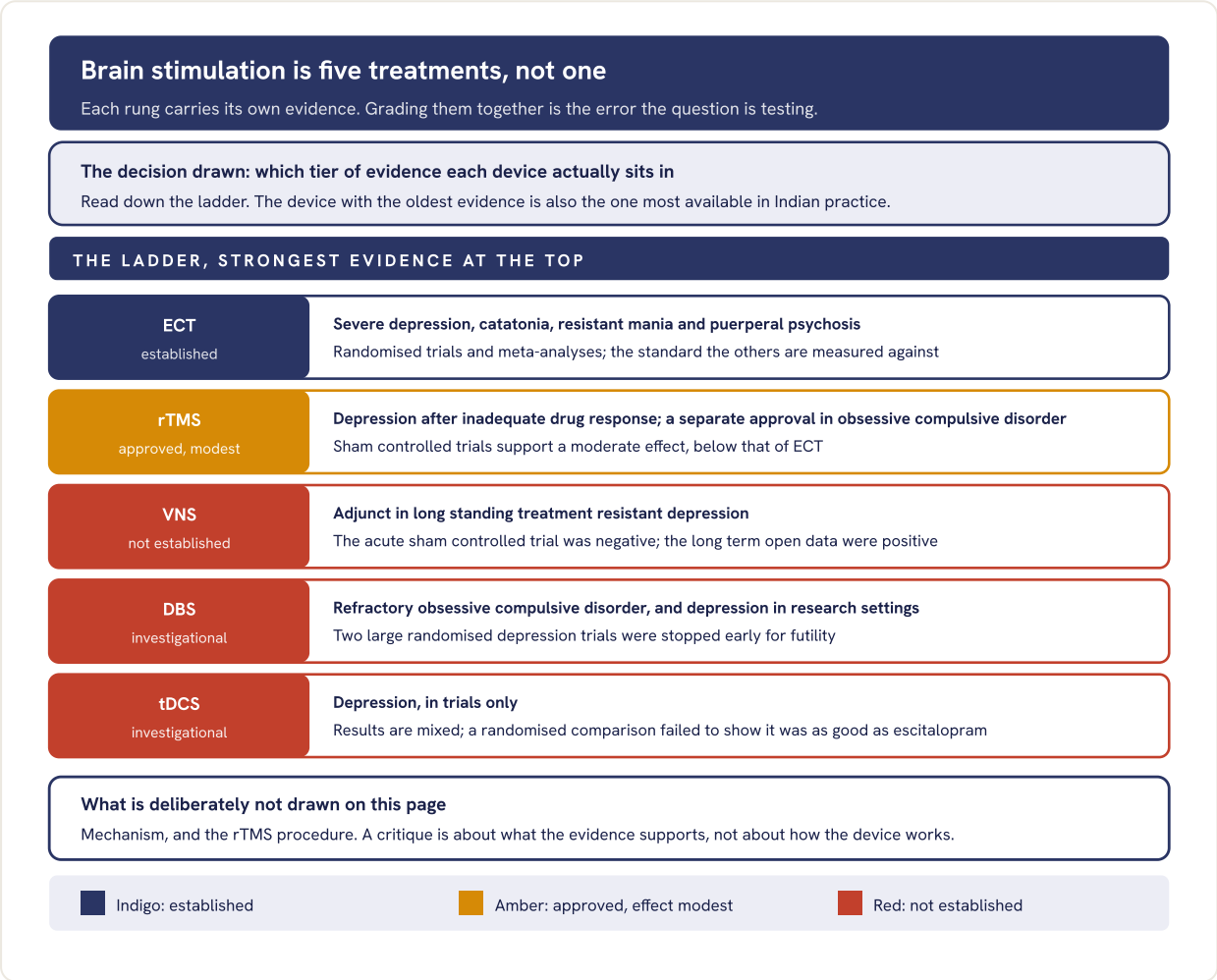

Fig 068.1 The family drawn as a graded ladder rather than a list, so that the critique is legible before a word of it is written.

- **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

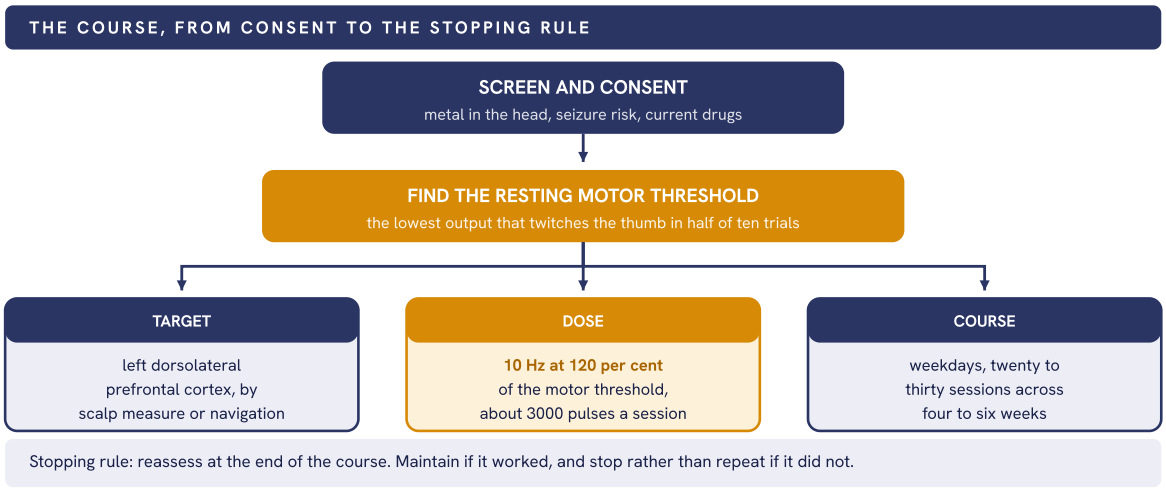

Fig 069.1 The threshold sits in amber because every later number on the page is a percentage of it, which is the step candidates omit.

- **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

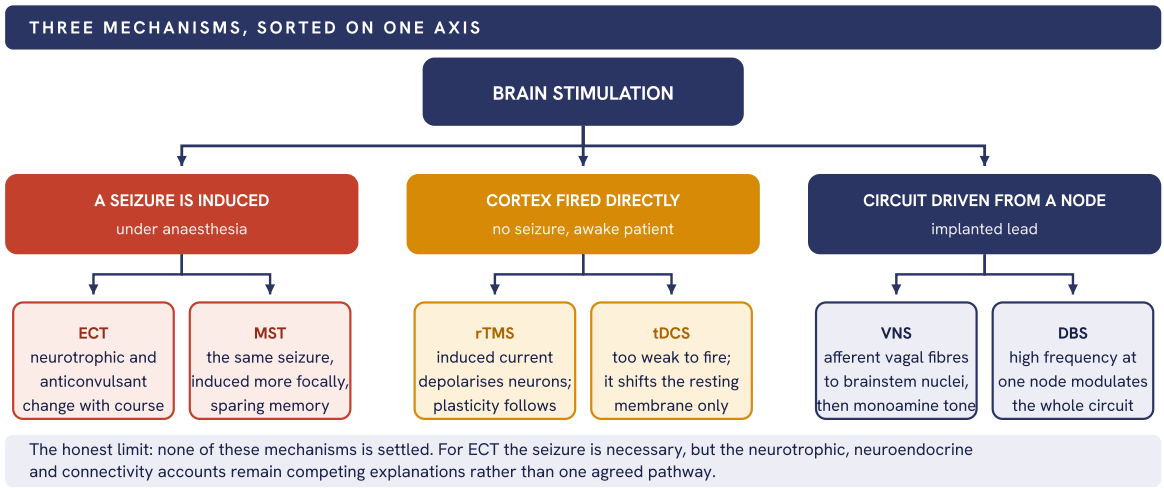

Fig 071.1 Every device sorted on one question, whether it fires neurons or only biases them, so the mechanism answer has a structure instead of six paragraphs.

- **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.

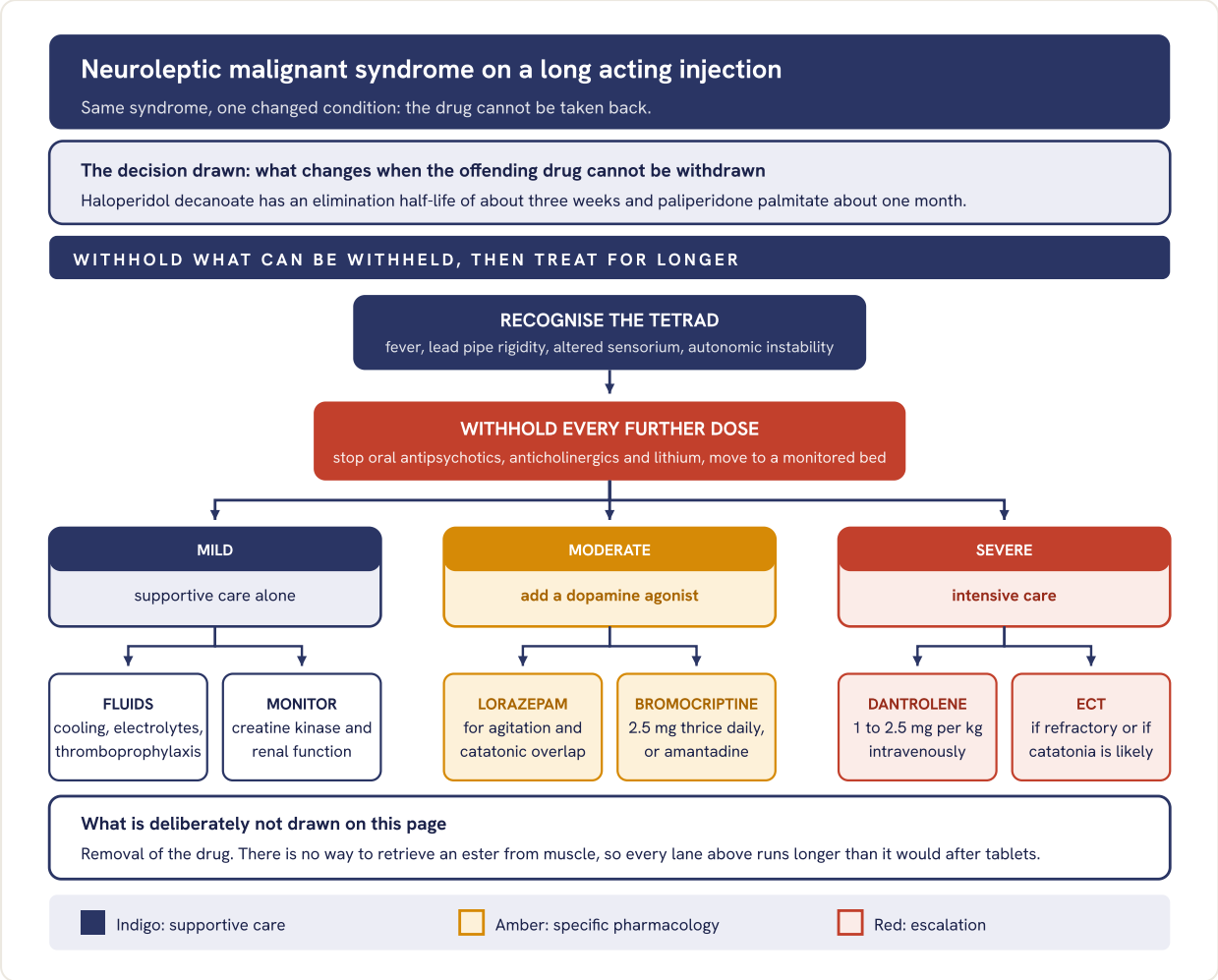

Fig 073.1 The ordinary algorithm redrawn for a drug that cannot be withdrawn, with the release half-life in the framing band so the longer course reads as a consequence.

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

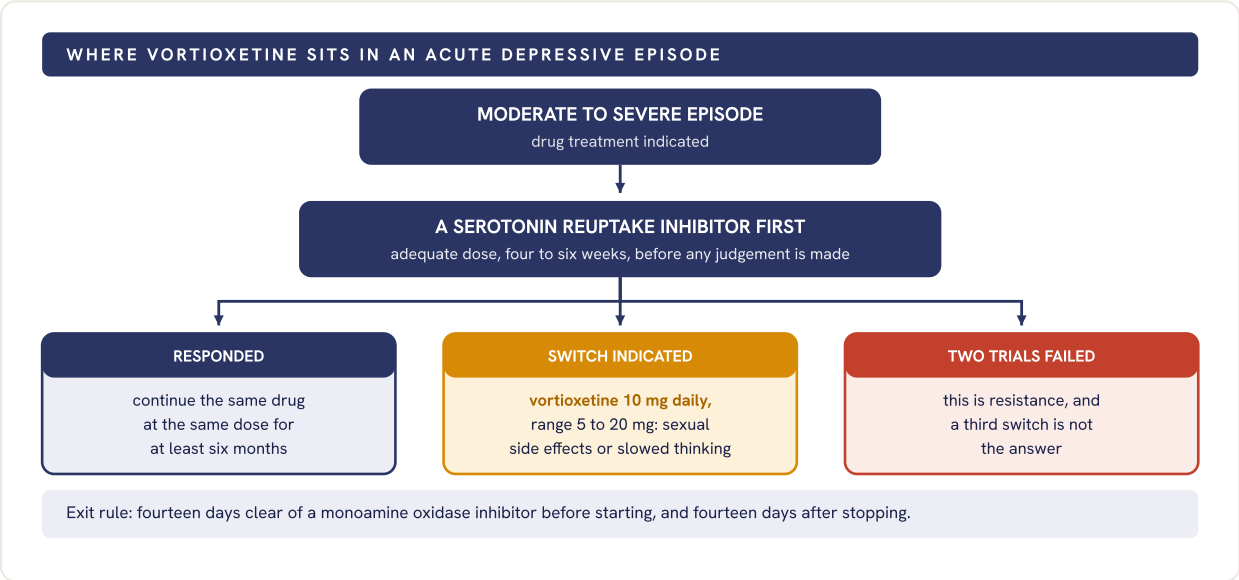

Fig 076.1 The drug placed rather than praised: vortioxetine enters at the switch, on two named grounds, and does not enter at the point where resistance has already been established.

- **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

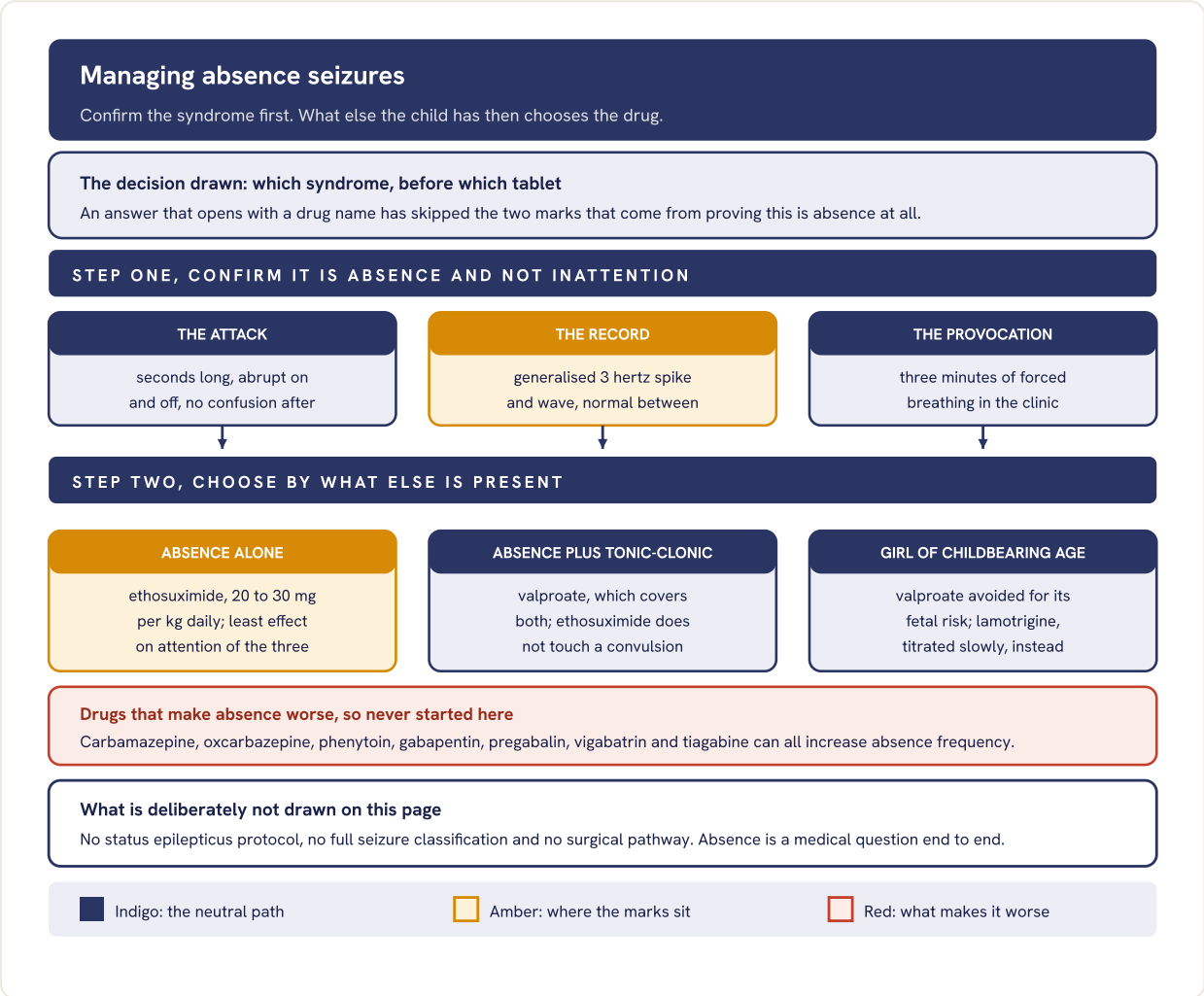

Fig 077.1 Confirmation first, then the drug chosen by what else the child has, with the aggravating drugs carried as a red strip because starting one is the commonest management error here.

- **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

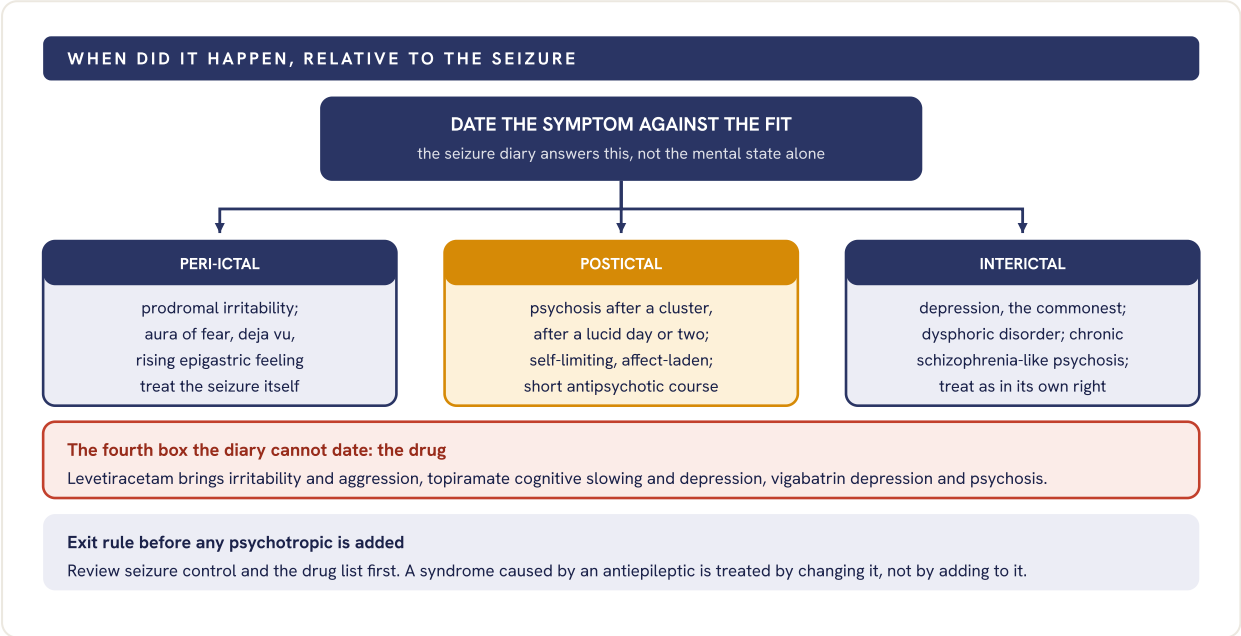

Fig 079.1 The syndromes sorted by their time relation to the seizure, with the drug-induced group set apart because it is the one the seizure diary cannot date.

- **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

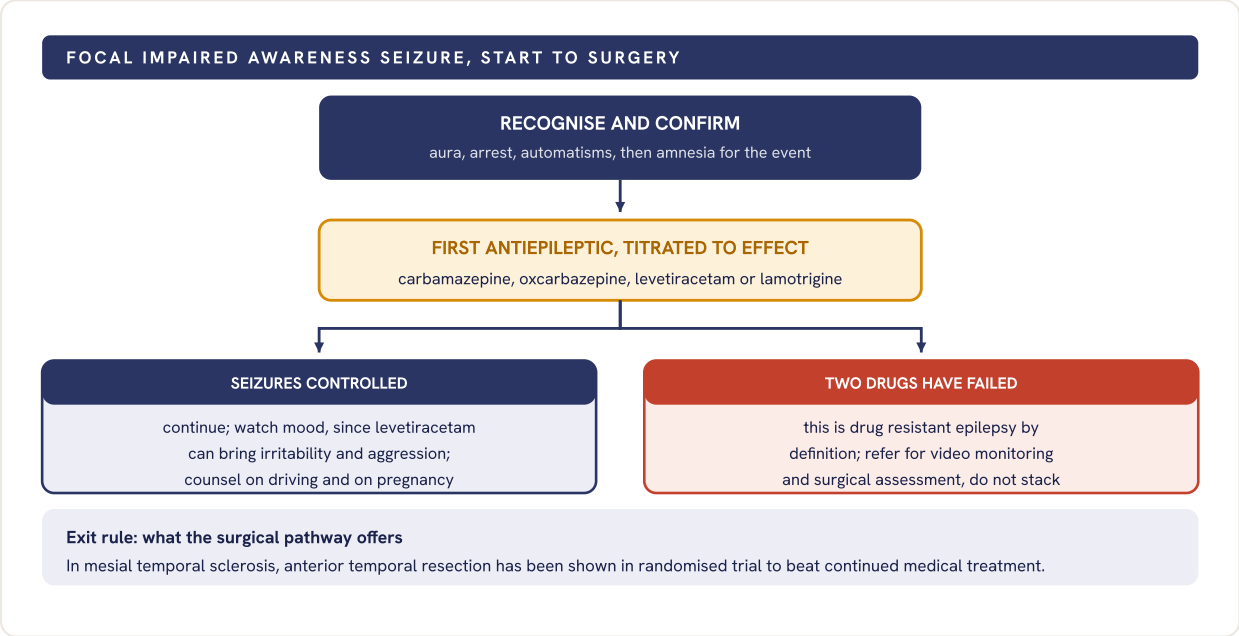

Fig 081.1 One drug, then a decision point that most answers never reach: two failed drugs is a definition, not a setback, and it routes the patient to assessment rather than to a third tablet.

- **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.

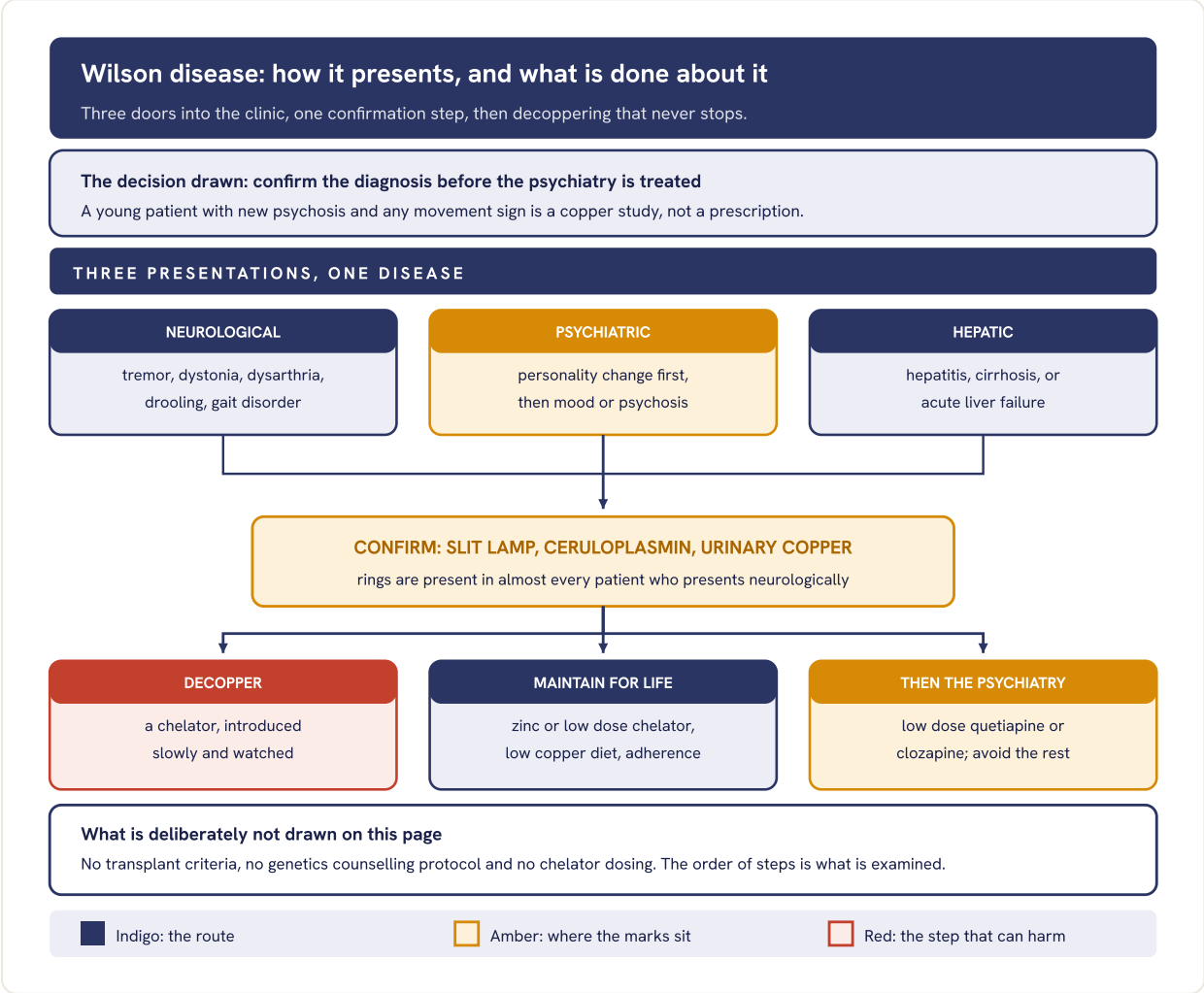

Fig 082.1 Three routes into the clinic, one confirmation step that must precede treatment, and the three arms of management that follow it.

- **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

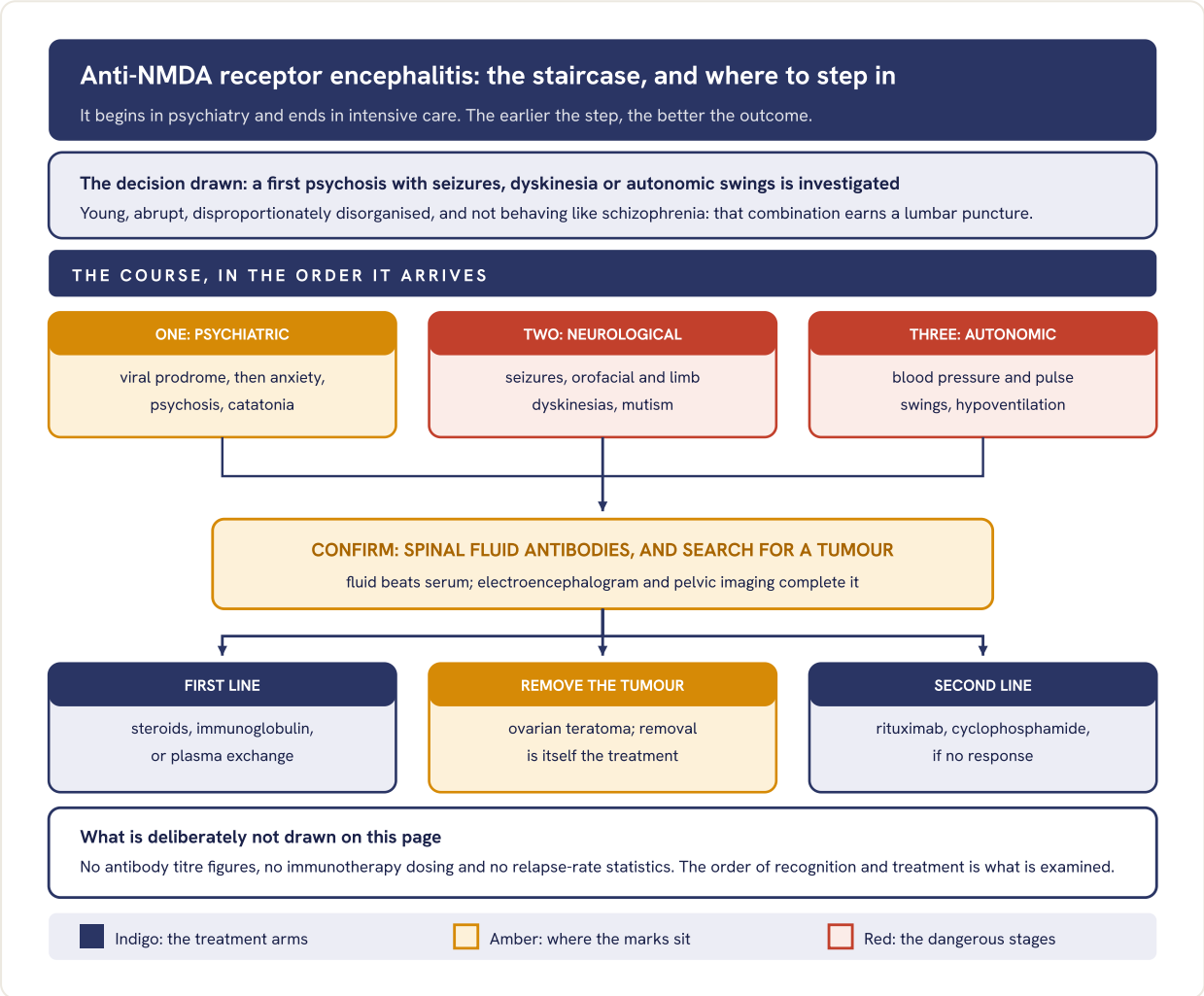

Fig 090.1 The staged course from psychiatric presentation to autonomic instability, the confirmatory step, and the three arms of treatment that follow it.

- **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.

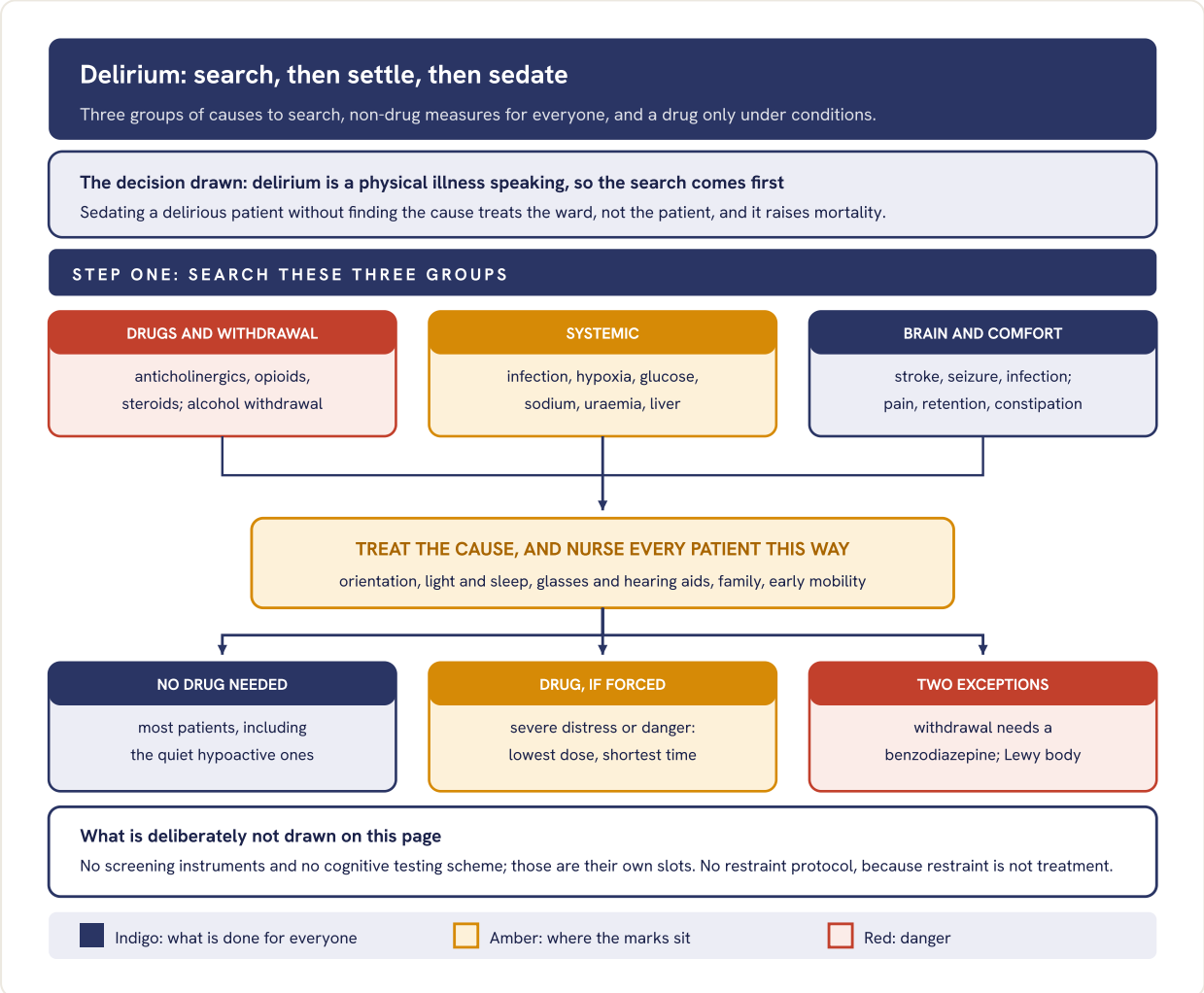

Fig 092.1 Three groups of causes to search, the non-drug care that every delirious patient receives, and the narrow conditions under which a drug is added.

- **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

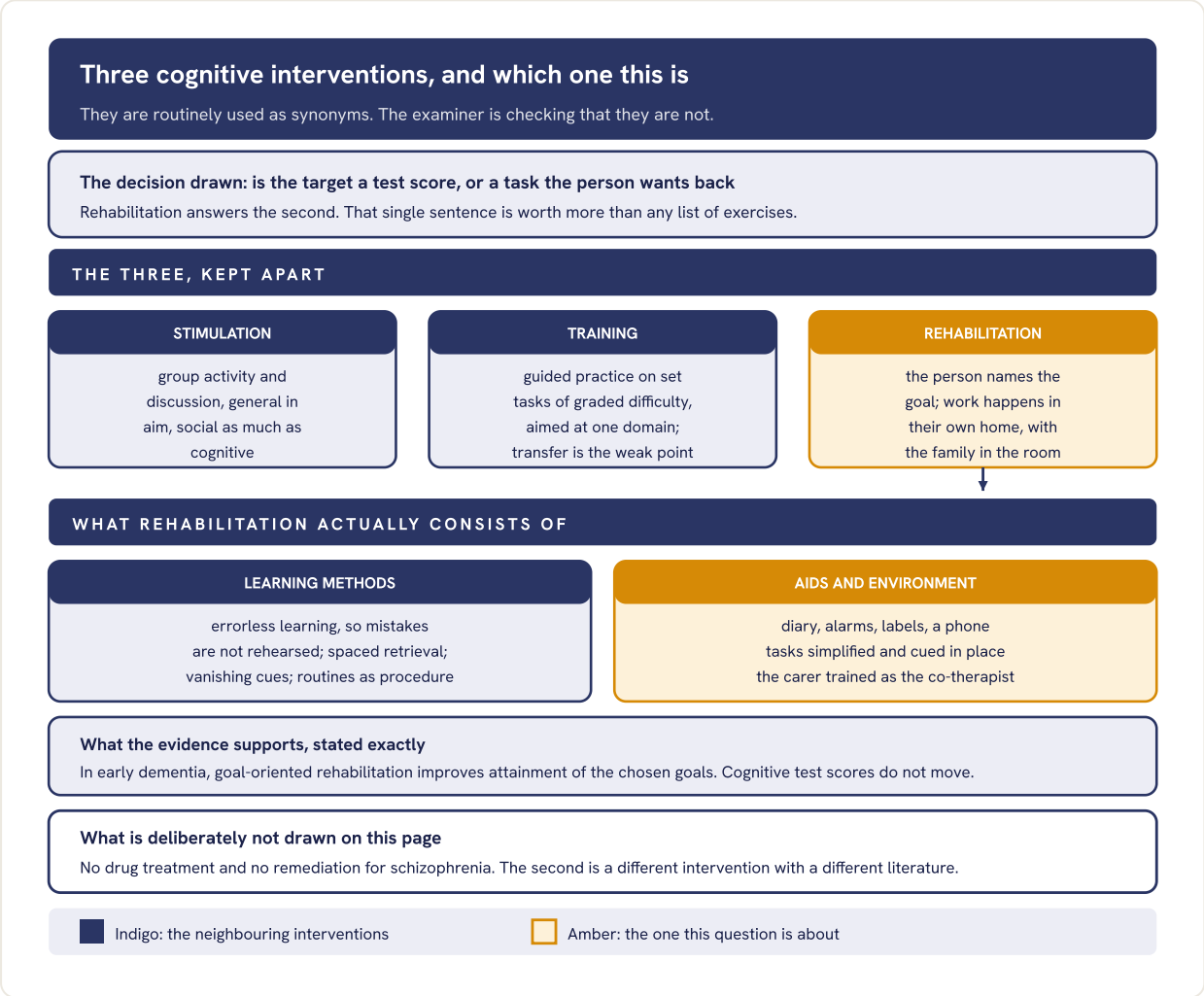

Fig 097.1 The three interventions separated at the top, then the content of rehabilitation itself below, with the evidence statement carried as a strip so the claim and its limit are read together.

- **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

APPENDIX, PAGE ONE OF TWO

Ten sets, ten sittings

The same hundred 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 24	IV-60 p 84	IV-05 p 15	IV-20 p 33	IV-66 p 92	IV-30 p 45	IV-44 p 63	IV-74 p 103	IV-52 p 73	IV-96 p 133
IV-S2	IV-13 p 25	IV-82 p 114	IV-06 p 17	IV-07 p 18	IV-09 p 20	IV-42 p 61	IV-45 p 64	IV-57 p 80	IV-71 p 98	IV-95 p 132
IV-S3	IV-01 p 11	IV-72 p 100	IV-26 p 41	IV-87 p 121	IV-34 p 51	IV-77 p 107	IV-64 p 89	IV-56 p 79	IV-58 p 81	IV-99 p 138
IV-S4	IV-14 p 27	IV-48 p 68	IV-31 p 47	IV-19 p 32	IV-67 p 93	IV-29 p 44	IV-65 p 90	IV-89 p 123	IV-93 p 130	IV-98 p 137
IV-S5	IV-15 p 28	IV-97 p 135	IV-17 p 30	IV-54 p 76	IV-10 p 21	IV-28 p 43	IV-37 p 54	IV-51 p 72	IV-53 p 74	IV-94 p 131
IV-S6	IV-39 p 57	IV-16 p 29	IV-18 p 31	IV-32 p 48	IV-11 p 22	IV-88 p 122	IV-68 p 94	IV-69 p 96	IV-59 p 82	IV-81 p 112
IV-S7	IV-02 p 12	IV-04 p 14	IV-24 p 38	IV-33 p 50	IV-35 p 52	IV-63 p 88	IV-78 p 109	IV-90 p 124	IV-38 p 55	IV-80 p 111
IV-S8	IV-40 p 59	IV-23 p 37	IV-25 p 39	IV-84 p 117	IV-41 p 60	IV-21 p 34	IV-79 p 110	IV-50 p 71	IV-85 p 118	IV-86 p 119
IV-S9	IV-92 p 128	IV-47 p 67	IV-61 p 86	IV-27 p 42	IV-08 p 19	IV-36 p 53	IV-55 p 77	IV-46 p 65	IV-70 p 97	IV-100 p 139
IV-S10	IV-22 p 36	IV-03 p 13	IV-62 p 87	IV-83 p 116	IV-49 p 69	IV-73 p 101	IV-43 p 62	IV-75 p 104	IV-76 p 105	IV-91 p 126

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.

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

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.

A

absence seizure **107**, 109, 110
add-on treatment 97
advance directive 13
affirmative counselling 22
AIDS dementia complex 121, **126**, 135
alcohol dependence 62, 116, **119**
Alzheimer disease 24, 25, **30**
amantadine **69**, 90, 101
Amitriptyline **48**, 50, 52
ammonia 117
Amnestic disorder 130
amnestic syndrome **116**, 128, 130
amyloid 29
anterior temporal resection 112
anterograde amnesia **116**, 119, 133
anti-NMDA receptor encephalitis 90, 123, **124**
anticipation 68
antiretroviral therapy 121, **126**
apathy 25, 32, **33**
aripiprazole **80**, 92
asterixis **117**, 119
ATP7B 114
augmentation **92**, 97, 105
authorisation committee **38**, 43
autoimmune encephalitis **79**, 123

B

B12 **24**, 28
bariatric surgery **77**, 116
behavioural addiction 20
behavioural variant 25
binge eating 77
Biofeedback **50**, 52
biomarker **27**, 87
bipolar affective disorder 88
bromocriptine 101
bronchial asthma 42, **44**

C

CAG trinucleotide repeat 68
Cambridge Neurological Inventory 55
cancer 41
candidate gene 86
capsulotomy 93
carbamazepine **39**, 86, 107
Cariprazine 88, **104**
catatonia 47, 87, **90**
central pontine myelinolysis 119
cerebellar degeneration 119
cerebral salt wasting 118
cerebral small vessel disease **60**, 73
cerebrospinal fluid 34, **122**, 124
ceruloplasmin 114
Chlorpromazine 80
cholinesterase inhibitor 25, 27, **30**
chorea 32, **68**, 123
chronic migraine 48
Chvostek and Trousseau signs 82
cingulotomy 93
Citalopram 42
clock drawing 132
clozapine 39, **69**, 73
cluster headache 48
cognitive rehabilitation **135**, 137, 138
Cognitive Remedial therapy 138
cognitive remediation 12, **84**
cognitive reserve **29**, 61, 137
Cognitive therapy 44
complex partial seizure 110, 111, **112**
computed tomography 47
confabulation 116, **130**
Confusion Assessment Method 131
consultation liaison psychiatry **36**, 37, 38
contusion **61**, 133
conversion practice 22
cortical dementia 25, 27, **32**
cortisol 87
COVID-19 20
creatine kinase 101
Creutzfeldt-Jakob disease 34
cryptococcal meningitis 121
CYP2C19 **42**, 86
CYP2D6 **41**, 86, 105
CYP3A4 38

D

dantrolene 101
DBS 94, 98
deep brain stimulation 69, 74, 94
delirium 20, 36, 128
Delirium Rating Scale 131
delirium tremens 119
demyelination 118, 119
depot antipsychotic 100, 101
depression after stroke 60, 73
Desvenlafaxine 104
diabetes mellitus 81
dialysis 43
diffuse axonal injury 61
disinhibition 25, 57, 59
dissociative amnesia 133
District Mental Health Programme 11, 12, 15
Donepezil 30
dopamine agonist 69, 73, 101
dorsolateral 65, 93, 96
dorsolateral prefrontal cortex 93, 96
drug resistant epilepsy 112
DSM-5 20, 87, 88

E

ECT 39, 89, 93
Efavirenz 121
electroconvulsive therapy 13, 31, 109
electroencephalogram 124
electronic media 15, 18, 20
Endophenotype 53
epilepsy 39, 57, 111
Epstein-Barr virus 72
errorless learning 135
Escitalopram 104
ethosuximide 107
event related potential 47, 53, 55

F

fatal familial insomnia 34
fibromyalgia 37, 45
first-generation antipsychotic 80
fluphenazine decanoate 100, 101
forced normalisation 110
frontotemporal dementia 25, 30, 32

G

Galantamine 30
gambling 20, 67
gaming disorder 20
gender incongruence 22
genome-wide association 86
Gerstmann-Straussler-Scheinker syndrome 34
Glasgow Coma Scale 59
glutamate 53, 90, 139
glycaemic control 29
Goitre 79

H

half-life 101, 105
haloperidol 39, 69, 101
haloperidol decanoate 101
Hashimoto thyroiditis 79
head injury 29, 57, 61
Healthcare Act 2017 19, 93
hepatic encephalopathy 47, 114, 117
herpes simplex encephalitis 64, 124, 133
high density lipoprotein 80
Hijra 22
hippocampus 47, 130
HIV disease 32, 33, 121
HLA-B 86
Hoehn and Yahr scale 74
Huntington disease 32, 33, 68
hypercalcaemia 41
hyperparathyroidism 82
hyperthyroidism 39
hyperventilation 107
hypoactive delirium 128
hypoglycaemia 81
hyponatraemia 79, 105, 118
Hypoparathyroidism 82
hypothyroidism 76, 79, 82

I

ICD-11 20, 22, 90
immunotherapy 124
Impulse control disorders 67
inattention 107, 128, 131
independent admission 13, 14
infertility 41
informed consent 93
insomnia 34, 43, 51
Interferon 71
interictal 110
irritable bowel syndrome 37, 45

ischaemic heart disease 36, 42, 44
Isoniazid 122

K

Kayser-Fleischer ring 114
ketamine 97
kidney transplant 38, 43
Kluver-Bucy syndrome 64, 65
Korsakoff syndrome 116, 119, 133
kuru 34

L

lactulose 117
lamotrigine 39, 107, 110
levetiracetam 39, 110, 112
levodopa 27, 69, 74
Levothyroxine 79
Lewy body dementia 27, 30, 32
LGBTQIA+ 22
life skills education 11, 21
linkage 21, 86
lithium 38, 39, 103
living donor 38, 43
long acting injectable 101
Lorazepam challenge 90
loss of consciousness 59, 63
Lumateperone 88

M

magnetic resonance imaging 124
magnetic seizure therapy 96, 109
mammillary bodies 116, 130
Manodarpan 21
Marchiafava-Bignami disease 119
me too drug 89, 104, 105
medication overuse 48
melatonin 27
memantine 25, 30, 139
Mental Health Act 1987 13
Mental Health Review Board 13, 14
Mental Healthcare Act 17, 19, 93
metabolic syndrome 77, 80, 81
migraine 48, 50, 52
mild cognitive impairment 24, 28, 29
Mild neurocognitive disorder 121, 126
Mini Mental State Examination 65, 132
Mirtazapine 92
mismatch negativity 53
modifiable factor 29
monoamine oxidase inhibitor 105
Montgomery Asberg scale 105
Montreal Cognitive Assessment 132
multiple sclerosis 71, 72
myoclonus 34
myxoedema madness 76, 79

N

N400 53
narcolepsy 47, 54
National Mental Health Programme 11
neurocysticercosis 111
neuroleptic malignant syndrome 27, 69, 101
neurological soft signs 47, 53, 55
neuroplasticity 137
neurosyphilis 33, 126
NMDA receptor 90, 123, 124
nominated representative 13, 14, 39

O

obesity 29, 77, 80
Oddball 53
olanzapine 69, 73, 100
olanzapine pamoate 100, 101
opportunistic infection 121, 126
orbitofrontal 57, 65, 90
orexin 51, 54
orexin receptor antagonist 51, 54
organ transplant 36, 38, 43
osmotic demyelination 118

P

P300 53
P450 30, 54, 139
paliperidone palmitate 100, 101
PANDAS 123, 124
Papez circuit 130
parasuicide 17
parathyroid 76, 79, 82
Parkinson disease 27, 30, 69
parkinsonism 24, 27, 48
personality change 32, 57, 65
Pharmacogenomics 86
physiological insomnia 51
POCSO 17
polydipsia 118
polygenic risk score 86

Polysomnography 47
post-concussional syndrome 57, 59
post-traumatic amnesia 57, 59, 61
post-traumatic stress disorder 57, 59, 87
postictal psychosis 110
pre-treatment evaluation 39
primary health centre 11, 18
Prion disease 34
pseudobulbar affect 71, 72
pseudodementia 31, 34
pseudoresistance 97
psychiatric genetics 86
psychosurgery 93, 94, 98

Q

quetiapine 69, 73, 92

R

Rashtriya Kishor Swasthya Karyakram 21
rechallenge 101
recovery 73, 82, 84
rehabilitation 12, 42, 135
repetitive transcranial magnetic stimulation 96, 109
resting motor threshold 96
retrograde amnesia 130
Rights of Persons with Disabilities Act 12, 13
risperidone 69, 73, 100
Rivastigmine 30

S

school mental health 17, 19, 21
second generation antipsychotic 89
Seizure disorder 112
self-harm 17, 20, 21
serotonin 39, 44, 50
Sertraline 42, 43
SIADH 118
South Asian waist 80
spaced retrieval 135
SPECT 47
spike and wave 107
SSRI 60
status epilepticus 107
stigma 17, 19, 21
subcortical dementia 25, 27, 32
subdural haematoma 119
successful ageing 137
suicide prevention 11
supported admission 13, 14
supported employment 84, 138
Suvorexant 54
Sydenham chorea 123

T

tau 25, 116
TBI 57, 59, 60
TDP-43 25
Tele MANAS 15
telemedicine 15
Telemedicine Practice Guidelines 15
telepsychiatry 15
temporal lobe 47, 64, 133
tender points 37
tension type headache 48, 52
teratoma 124
thalamus 33, 54, 116
theta burst 93
thiamine 116, 119, 130
thyroid disorders 76, 79, 82
thyroxine 76, 79
toxoplasmosis 121, 126
transcranial direct current stimulation 96
transient global amnesia 133
Transplantation of Human Organs and Tissues Act 38, 43
traumatic brain injury 57, 59, 61
treatment gap 18, 19
treatment resistant 94
triglyceride 80
triptan 48
tuberculoma 111
tuberculous meningitis 121, 122
twin studies 86

V

vagus nerve stimulation 93
valproate 39, 48, 107
vascular depression 60
VNS 94, 98
vortioxetine 103, 104, 105

W

weight gain 79, 103, 105
Wernicke encephalopathy 116, 119, 133
Wilson disease 32, 33, 114

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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 of the community and public mental health area, the consultation-liaison maps, and the dementia and delirium management pages. 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.

The tools. The counting, weighting, selection and typesetting all run as code in this repository, so any number in this book can be traced back to the row it came from.

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