WEAVE 100 - PAPER II

MD Psychiatry theory. DNB revision fits too.

Psychoses, Mood and the Adult Disorders

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 psychoses, the mood disorders, the drugs that treat them, and the substance, anxiety, trauma and personality presentations a psychiatrist sees every week. Fourteen 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 POWERED BY TRIJOG

A NOTE ON WHO MADE THIS

Weave, powered by Trijog

Weave is a psychiatry practice and a publishing house for the things clinicians and families actually need to read. Trijog is the mental-health organisation Weave partners with for therapy delivery at scale.

WHAT WEAVE DOES	Integrative psychiatry, online and in Mumbai, and a free library for residents, clinicians and families.
WHAT TRIJOG ADDS	A therapy network with the reach to see the person you refer, quickly, and to keep seeing them.
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 II

Psychoses, Mood and the Adult Disorders

One hundred ten-mark questions, solved as answer maps

THE WEAVE 100 SERIES

TITLE

Weave 100, Paper II: Psychoses, Mood and the Adult Disorders

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

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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.

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THE HUNDRED, IN WEIGHT ORDER

01 **Eating, sleep and sexual disorders** II-01 to II-09 · 9 slots · 9.7% 10

02 **Substance use: opioids, other drugs and treatment** II-10 to II-18 · 9 slots · 9.4% 21

03 **Anxiety, OCD and related disorders** II-19 to II-27 · 9 slots · 8.9% 32

04 **Schizophrenia: aetiology, phenomenology and course** II-28 to II-35 · 8 slots · 8.4% 43

05 **Trauma, dissociative and somatic symptom disorders** II-36 to II-43 · 8 slots · 8.4% 53

06 **Schizophrenia: treatment and antipsychotics** II-44 to II-51 · 8 slots · 8.3% 63

07 **Mood disorders: pharmacotherapy** II-52 to II-58 · 7 slots · 7.5% 73

08 **Mood disorders: syndromes and nosology** II-59 to II-65 · 7 slots · 7.5% 82

09 **Psychotherapies** II-66 to II-72 · 7 slots · 6.9% 91

10 **Personality disorders** II-73 to II-79 · 7 slots · 6.6% 100

11 **Clinical assessment and cross-cutting nosology** II-80 to II-85 · 6 slots · 5.5% 109

12 **Substance use: general concepts and alcohol** II-86 to II-90 · 5 slots · 4.5% 116

13 **Other psychoses and catatonia** II-91 to II-95 · 5 slots · 4.3% 123

14 **Suicide, self-harm and impulse control** II-96 to II-100 · 5 slots · 4.0% 130

BACK MATTER

Ten sets, ten sittings 137

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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 II.

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

AREA, IN WEIGHT ORDER	UNIV POOL	DNB	WEIGHT	SHARE	SLOTS
01 Eating, sleep and sexual disorders	0.392	0.301	0.365	9.67%	9
02 Substance use: opioids, other drugs and treatment	0.304	0.470	0.354	9.39%	9
03 Anxiety, OCD and related disorders	0.318	0.380	0.337	8.93%	9
04 Schizophrenia: aetiology, phenomenology and course	0.270	0.432	0.318	8.45%	8
05 Trauma, dissociative and somatic symptom disorders	0.282	0.400	0.317	8.42%	8
06 Schizophrenia: treatment and antipsychotics	0.244	0.474	0.313	8.31%	8
07 Mood disorders: pharmacotherapy	0.243	0.377	0.283	7.51%	7
08 Mood disorders: syndromes and nosology	0.280	0.284	0.281	7.45%	7
09 Psychotherapies	0.226	0.344	0.261	6.93%	7
10 Personality disorders	0.243	0.268	0.251	6.65%	7
11 Clinical assessment and cross-cutting nosology	0.209	0.199	0.206	5.46%	6
12 Substance use: general concepts and alcohol	0.164	0.183	0.170	4.50%	5
13 Other psychoses and catatonia	0.203	0.067	0.163	4.31%	5
14 Suicide, self-harm and impulse control	0.166	0.116	0.151	4.01%	5

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

THE WEIGHTAGE CHAPTER, PAGE TWO OF THREE

How the hundred were picked

BOARD	SITTINGS COUNTED	HOW THE PAPERS REACHED US	TRUST
RGUHS	15	Hand transcription and public mirrors of the printed papers	0.806
KUHS	9	Official university portal PDFs	0.600
TN-MGRMU	35	Public archive of the printed papers, exact sitting dates recovered	0.480
KNRUHS	3	Official university portal PDFs	0.300
MUHS	2	Two watermark-confirmed papers, read by OCR. A third-party recurrence compilation was tested against them, matched none, and was removed from the sitting count	0.267
NTRUHS	2	Mirror text of the printed papers	0.160
WBUHS	2	Public archive of the printed papers	0.180
DNB	10	Official NBE material; 32 of 40 files verified by checksum against the board's own published path	fixed 0.30

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

1 Count the sittings

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

2 Weight the areas

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

3 Turn weight into slots

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

4 Score every eligible question

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

5 Fill the slots

The highest scorers in each area, up to that area's slot count, near-duplicates merged first. All 100 were filled from questions actually set: 15 anchor, 13 high, 5 recurrent, 67 single-appearance.

THE WEIGHTAGE CHAPTER, PAGE THREE OF THREE

What this book cannot promise

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

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

Most questions were seen once. Of the 440 selectable Paper II questions in the record, 384 appeared in a single sitting. That is not a defect in the counting, it is what happens when eight boards set different papers: the same TOPIC returns constantly while the same QUESTION rarely does. So the selection is driven by AREA weight, where the signal is dense, and each map prints a band rather than a frequency claim about its own question.

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

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

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

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

AREA 01 OF 14 · P2-A11

Eating, sleep and sexual disorders

9	9.7%	II-01	II-09
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 4 RECURRENT 1 SINGLE 4

REVISION ORDER

The three impotence slots first, and together: they share one differential and one management spine, and the classification slot frames all of them. Anorexia and bulimia next, as one comparison. Gender identity, sex reassignment and the offences slot last.

WHAT IS IN THIS AREA

Seven of the nine slots are sexual: erectile dysfunction and psychogenic impotence across three of them, the classification of male sexual dysfunction, gender identity and gender dysphoria, sex reassignment surgery, and the sexual offences slot. Eating takes the other two, anorexia nervosa and bulimia nervosa. The registered name of this area also carries sleep, and no sleep question survived selection into it. Nothing on the pages that follow is a sleep question, and the area is described here by what it actually holds.

II - 01

Discuss the clinical features and complications of anorexia nervosa. [10]

10 EATING, SLEEP AND SEXUAL DISORDERS

KNRUHS, NTRUHS, WBUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 **Diagnostic core**	Low weight, fear of gain, disturbed body experience
3 **Clinical features**	Behaviour, cognition, and what starvation itself makes
4 **Complications**	System by system, then refeeding
1 **Outcome**	Mortality and the two things that drive it

THE DEFINITION

Restriction of intake producing significantly low weight for age, sex and developmental stage, with fear of weight gain or behaviour that prevents it, and disturbed experience of weight or shape. ICD-11 uses body mass index below 18.5 in adults and has dropped amenorrhoea. DSM-5-TR grades severity on body mass index and names restricting and binge-purge subtypes.

- **Behaviour and cognition.** Dietary rules, driven exercise, purging, food rituals, weighing; overvaluation of shape, denial of seriousness, poor insight.
- **Starvation is a cause, not only an effect.** Low mood, irritability, obsessionality, social withdrawal and preoccupation with food improve with weight restoration.

SYSTEM	WHAT IS FOUND	WHY IT MATTERS
Cardiac	Bradycardia, hypotension, prolonged QT interval, pericardial effusion	**Arrhythmia is a leading cause of death**
Electrolyte	Hypokalaemia; hypochloraemic alkalosis with vomiting; hyponatraemia with water loading	**Drives the arrhythmia risk**
Endocrine	Low triiodothyronine, hypercortisolaemia, hypogonadotropic hypogonadism, osteoporosis	Bone loss may not fully reverse
Gut and liver	Delayed emptying, constipation, raised transaminases, superior mesenteric artery syndrome	Explains bloating on refeeding
Marrow	Anaemia, leucopenia, gelatinous marrow transformation	Reverses with weight gain
Refeeding	Falling phosphate, potassium and magnesium; thiamine depletion; fluid overload	**Iatrogenic, and the one to anticipate**

Amber cells are what an examiner is looking for: the complications that change what you do today, not the ones that merely lengthen the list.

WHAT EARNS THE MARKS

- **Both current criteria named,** with the amenorrhoea point stated as a change rather than assumed.
- **Complications grouped by system,** not listed flat. The grid is the answer.
- **Refeeding syndrome as a separate row,** with phosphate named and thiamine given before calories.
- **Mortality attributed** to cardiac events and to suicide, which is the line most answers omit.

SEE ALSO Slot II - 07 bulimia nervosa SPRINT Day 17, Eating and sleep-wake disorders

II - 02

Management of erectile dysfunction. [10]

10 EATING, SLEEP AND SEXUAL DISORDERS

KUHS, MUHS, NTRUHS, RGUHS, TN-MGRMU - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

3 Assessment	History, vascular and endocrine screen, drug review
5 The ladder	Modify, then oral, then psychosexual, then second line
2 Special situations	Drug induced, and the Indian couple context

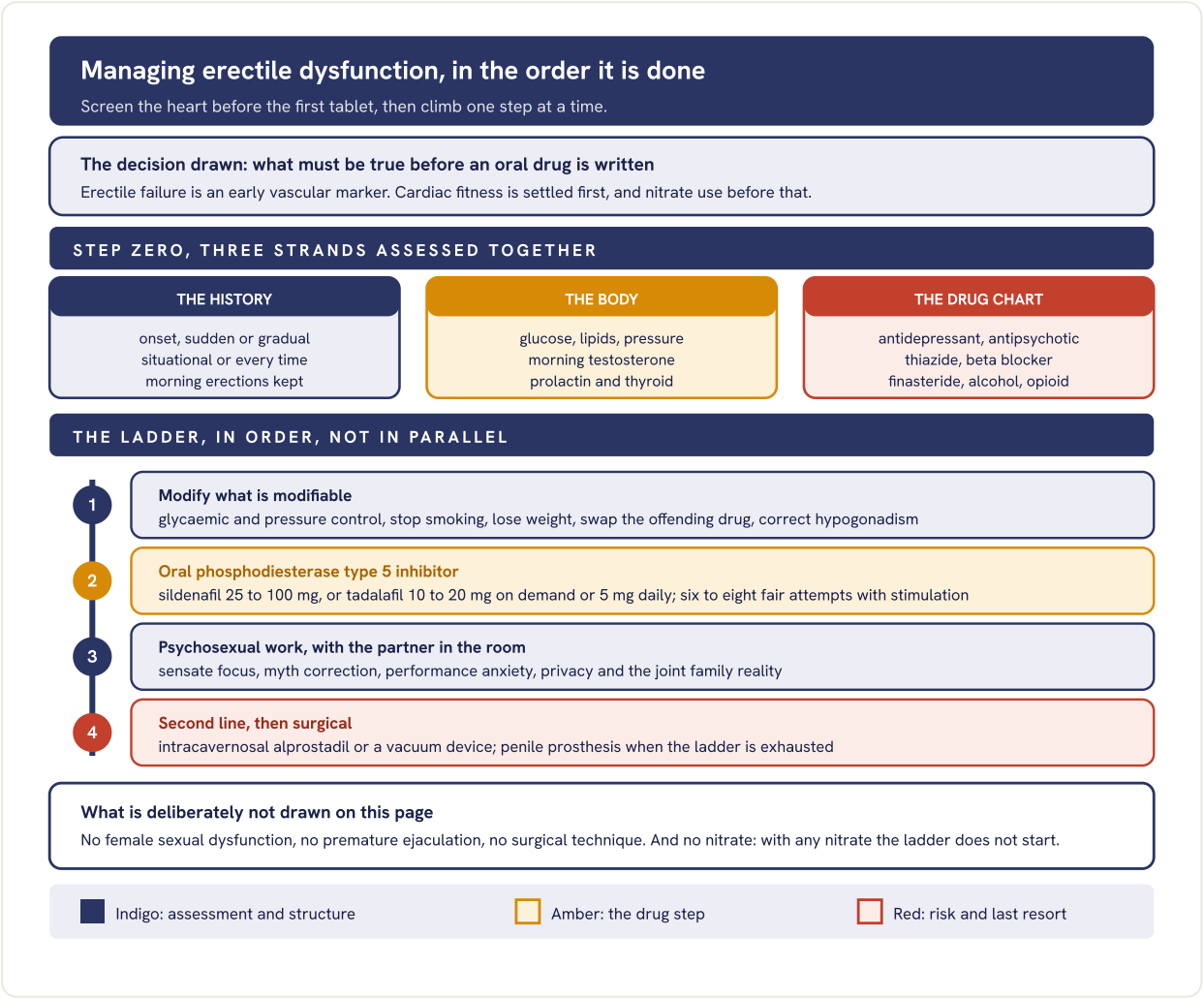

Fig 002.1 Three strands assessed together, then a four-rung ladder climbed in order, the nitrate question settled before rung two.

- **Drug induced.** Reduce the dose, switch to bupropion or mirtazapine, or add an oral inhibitor. For antipsychotics, check prolactin.

PITFALL

Calling the oral drug a failure too early. Most apparent non-response is an inadequate trial: no stimulation, a full stomach with sildenafil, or the lowest dose tried twice.

WHAT EARNS THE MARKS

- **Cardiac fitness and nitrate use settled before the prescription,** written as step zero.
- **The ladder in order,** with a named drug, a dose, and what counts as an adequate trial.
- **The partner in the plan.** Treating the man alone loses the psychosexual marks.

SEE ALSO Slot II - 03 psychogenic impotence Slot II - 09 male sexual dysfunction **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

II - 03

Define psychogenic impotence as per the current diagnostic systems. Discuss its differential diagnosis. Write in brief about management. [4+2+4]

4 + 2 + 4 | EATING, SLEEP AND SEXUAL DISORDERS | DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

4 Definition	Both systems, and the descriptors each attaches
2 Differential	Organic causes, and the near neighbours
4 Management	Couple work first, the tablet as scaffolding

THE DEFINITION, IN THE WORDS OF THE SYSTEMS

Neither system uses the word impotence. DSM-5-TR erectile disorder requires difficulty obtaining or maintaining an erection, or reduced rigidity, on most occasions for at least six months, with distress, specified as lifelong or acquired and generalised or situational. ICD-11 asks the same descriptors and adds the aetiological contributors, so psychogenic is a descriptor rather than a diagnosis.

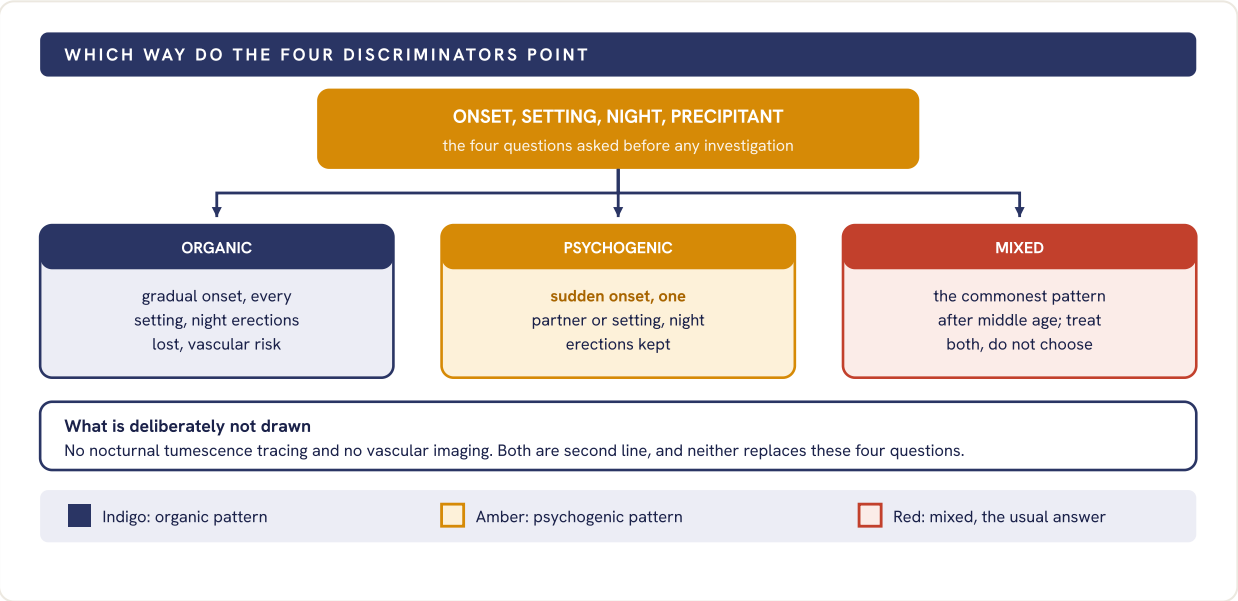

Fig 003.1 The psychogenic label rests on four bedside questions, and the honest third lane is mixed rather than either pure form.

- **Differential.** Vascular, neurogenic, endocrine and drug induced causes; early ejaculation reported as failure; low desire; depressive disorder; relationship discord; semen-loss anxiety.
- **Management.** Correct the myths, sensate focus with the partner, treat depression and anxiety, and use an oral inhibitor as short-term scaffolding.

WHAT EARNS THE MARKS

- **Naming that neither system carries the word impotence,** and giving the descriptor set instead.
- **The four discriminators from the figure,** quoted as the bedside test rather than a laboratory one.
- **Mixed aetiology named as the common answer,** which stops the artificial either or.
- **The partner in the plan,** and the tablet framed as scaffolding, not treatment.

SEE ALSO Slot II - 02 erectile dysfunction Slot II - 06 medico-legal impotence SPRINT
Day 18, Sexual, impulse-control disorders and suicide

II - 04

Discuss the indications of sex reassignment surgery. Describe the role of the psychiatrist in this matter. [4+6]

4 + 6 | EATING, SLEEP AND SEXUAL DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The terms	Gender-affirming surgery, and what Indian law now says
2	Indications	Persistence, capacity, age, stability, hormone time
5	The psychiatrist	Stage by stage, and what is documented at each
1	The limit	Surgery is not the gate to legal identity

THE ELIGIBILITY CRITERIA, WORTH WRITING OUT

Persistent and well documented gender incongruence; capacity to give informed consent; age of majority; reasonable control of any coexisting medical or mental health condition; and for genital surgery, a period of continuous hormone therapy. The eighth version of the international standards of care removed the fixed real life experience requirement and the routine two referral rule.

STAGE	WHAT THE PSYCHIATRIST DOES	WHAT IS RECORDED
Assessment	Confirm gender incongruence; exclude psychotic identity disturbance, body dysmorphic disorder and dissociative presentations	**Persistence, onset and course**
Comorbidity	Treat depression, anxiety, substance use and suicidality to stability, not to cure	Response and current risk
Consent	Capacity, realistic expectation, fertility and irreversibility discussed	**A capacity opinion, dated**
Family and work	Family sessions, disclosure planning, school or workplace liaison	Supports actually available
After surgery	Adjustment, pain and body image work, hormone adherence, regret screening	Follow up at fixed intervals

Amber cells are the two documents an examiner expects by name: a statement of persistence and a dated capacity opinion.

- **The Indian frame.** The 2014 Supreme Court judgment recognised the right to a self-identified gender, and the Transgender Persons Act of 2019 gives a certificate of identity through the District Magistrate. Surgery is not a precondition for that recognition.

WHAT EARNS THE MARKS

- **Eligibility written as a list of criteria,** not as a general statement of readiness.
- **The role split by stage,** which is what turns a description into a plan.
- **Stability rather than cure** as the comorbidity standard. The opposite reads as gatekeeping.
- **The statutory line,** that legal identity does not wait on an operation.

SEE ALSO Slot II - 08 gender dysphoria **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

II - 05

What are the sexual offences? Discuss management strategies in a case of pedophilia. Narrate neuropsychological findings in people convicted of sexual offences. [2+5+3]

2 + 5 + 3 | EATING, SLEEP AND SEXUAL DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The offences	Where they now sit in Indian statute
5 Management	Report, assess risk, treat, supervise, review
3 Neuropsychology	What group studies show, and what they cannot do

- **The statutes.** The Bharatiya Nyaya Sanhita of 2023 carries rape, gang rape, assault on modesty, sexual harassment, voyeurism and stalking, replacing the older penal code from July 2024. The Protection of Children from Sexual Offences Act of 2012 covers every sexual offence against a person under eighteen.
- **Pedophilic disorder is the clinical term.** Sustained sexual arousal to prepubescent children over at least six months, in a person aged sixteen or above and at least five years older, either acted upon or causing marked distress.

STEP	WHAT HAPPENS	WHO ACTS	CONDITION OR INSTRUMENT
1	Report an offence against a child	Any professional who learns of it	**Mandatory, without delay**
2	Structured risk assessment	Psychiatrist with the team	Static-99R, Stable-2007
3	Treat comorbidity and substance use	Psychiatrist	Disinhibition raises risk
4	Relapse prevention therapy	Therapist, in a group where possible	Offence chain, victim empathy
5	Drug treatment where urges are intense	Psychiatrist, with consent	**Fluoxetine 40 to 60 mg; antiandrogen with bone and metabolic monitoring**
6	Supervision and scheduled review	Team with probation or family	No unsupervised contact

Order, actor and standard in one view. Reporting duty comes first on the page, as it does in practice.

WHAT THE LAW AND THE TESTS DO NOT COVER

Neuropsychological findings are group level. Convicted samples show frontal and executive weakness, lower measured intelligence in pedophilic samples, raised rates of non-right-handedness, and imaging differences in orbitofrontal, amygdalar and temporal regions. None of it identifies an individual offender. A diagnosis is not a defence, and treatment does not replace the court process.

WHAT EARNS THE MARKS

- **Reporting duty written as step one,** before any clinical formulation.
- **Named risk instruments,** which separate a structured assessment from an impression.
- **Drug treatment placed after therapy and consent,** with the monitoring named.
- **The exclusions strip.** Saying the neuropsychology is group level is the mark most answers give away.

SEE ALSO Slot II - 06 medico-legal impotence SPRINT Day 18, Sexual, impulse-control disorders and suicide

II - 06

What is impotence? What are the causes and the medico-legal aspects of impotence? What are its common psychiatric sequelae? [2+4+4]

2 + 4 + 4 | EATING, SLEEP AND SEXUAL DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The two meanings	A clinical diagnosis against a legal word
2 Causes	Four organic groups, drugs, and the psychogenic set
4 Medico-legal	Where impotence decides a marriage or a charge
2 Sequelae	The man, the marriage, and the help-seeking

- **The two meanings.** Clinically the diagnosis is erectile dysfunction. In law impotence is wider: the inability to consummate a marriage, and it is judged in relation to one spouse rather than in general.
- **Causes.** Vasculogenic, neurogenic, endocrine and anatomical; drugs including antidepressants, antipsychotics, thiazides, beta blockers, finasteride, alcohol and opioids; and psychogenic causes led by performance anxiety, depression, discord and semen-loss anxiety.

STATUTE OR SETTING	WHAT IS CLAIMED	WHO DECIDES	THE STANDARD APPLIED
Hindu Marriage Act 1955	Marriage voidable, never consummated	Civil court on medical opinion	**Relative impotence with this spouse is enough**
Special Marriage Act 1954	Same ground for nullity	Civil court	Impotence at marriage and at suit
Dissolution of Muslim Marriages Act 1939	Wife seeks dissolution	Court	Husband allowed one year to show recovery
Divorce Act 1869	Nullity for Christian marriages	Civil court	Impotence at marriage and at suit
Criminal proceedings	Accused pleads incapacity	Medical board on court order	**Establishes present capacity only**

The medico-legal marks live in the last column. Every statute here asks a different question of the same finding.

WHAT A POTENCY EXAMINATION DOES NOT SETTLE

It speaks to capacity today, not to capacity on a past date. A normal finding does not exclude situational psychogenic impotence, and failure under examination conditions is not proof of incapacity. Incapacity for intercourse also does not exclude every sexual offence, because the statutory definitions extend beyond it. The psychiatrist gives an opinion; the court makes the finding.

WHAT EARNS THE MARKS

- **The clinical and the legal meaning separated in the first two lines.**
- **Relativity stated,** that impotence with one spouse is sufficient for nullity.
- **Sequelae named as a list:** depression, anticipatory anxiety, marital discord, alcohol use, semen-loss preoccupation, secrecy and delayed help-seeking.
- **The exclusions strip,** which is what separates a clinician from a reporter of findings.

SEE ALSO Slot II - 03 psychogenic impotence Slot II - 05 sexual offences SPRINT

Day 18, Sexual, impulse-control disorders and suicide

II - 07

Clinical features and management of bulimia nervosa. [5+5]

5 + 5 | EATING, SLEEP AND SEXUAL DISORDERS

DNB, RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

3 **The syndrome**	Binge, compensation, and the overvaluation behind both
2 **Signs and labs**	What the body shows while the weight looks normal
4 **Treatment**	Medical risk, then psychological, then the drug
1 **Follow through**	Laxative taper, dental review, comorbidity

- **The syndrome.** Recurrent binges with loss of control, recurrent compensation by vomiting, laxatives, fasting or driven exercise, at least weekly for three months, with self-worth judged on shape and weight. Weight is usually normal, which is why it is missed.
- **Signs and labs.** Knuckle callus, parotid swelling, dental erosion, subconjunctival haemorrhage; hypokalaemia, hypochloraemic alkalosis, raised amylase; rebound oedema when laxatives stop.

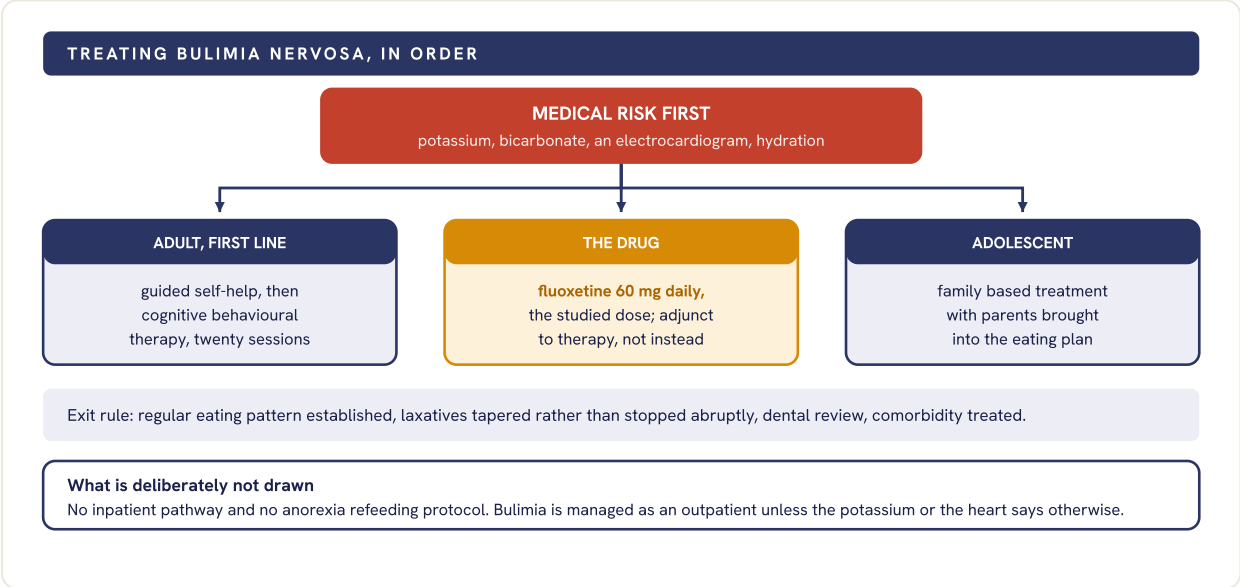

Fig 007.1 Medical risk settled first, then three treatment lanes chosen by age and by role, with the exit rule carried along the foot.

PITFALL

Reaching for bupropion. It lowers the seizure threshold and is avoided where there is purging. The second trap is reassurance about a normal weight, which is exactly what delays the diagnosis.

WHAT EARNS THE MARKS

- **Potassium and the electrocardiogram named before any therapy,** as the first line of management.
- **A named psychological treatment with a dose of sessions,** not the phrase psychotherapy alone.
- **Fluoxetine at 60 mg,** stated as an adjunct rather than a substitute.
- **The laxative taper and the dental review,** which most answers never reach.

SEE ALSO Slot II - 01 anorexia nervosa **SPRINT** Day 17, Eating and sleep-wake disorders

II - 08

What is gender identity? What are the clinical features of gender dysphoria? Briefly discuss its management. [2+3+5]

2 + 3 + 5 | EATING, SLEEP AND SEXUAL DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Gender identity	The term, and the three things it is not
3 Clinical features	Criteria, and how children differ from adults
5 Management	Affirm, assess, then stage the physical steps

THE DEFINITION

Gender identity is a person's internal sense of being male, female, both, neither or elsewhere on a spectrum. It is not the sex recorded at birth, not gender expression, and not sexual orientation. ICD-11 renamed the condition gender incongruence and moved it out of the mental disorders chapter into conditions related to sexual health, keeping a code for access while removing the label of disorder.

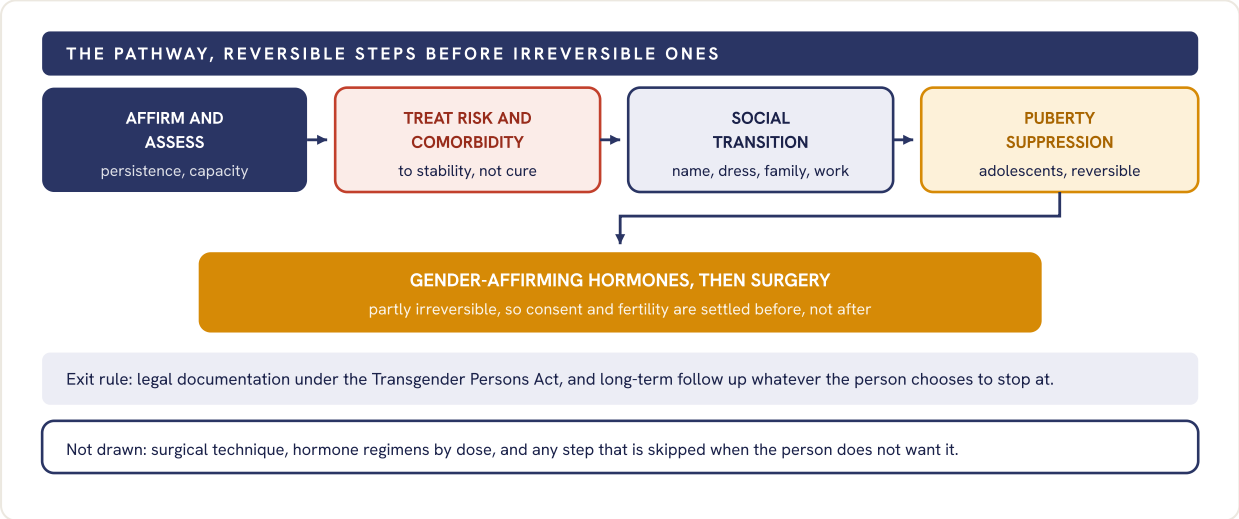

Fig 008.1 Reversible steps precede partly irreversible ones, and the person may stop at any point on the line without the pathway being incomplete.

- **Clinical features.** Marked incongruence between experienced and assigned gender for at least six months, with distress or impairment; in children, insistence on another gender with cross-gender play and aversion to one's own anatomy. Puberty is the point at which persistence becomes clearer.
- **Two things to say.** Distress often reflects minority stress rather than the identity itself. Conversion practices are unethical and have been named professional misconduct by the national regulator.

WHAT EARNS THE MARKS

- **Identity distinguished from expression and orientation** in the first two lines.
- **The ICD-11 relocation named,** which shows the current position rather than the older one.
- **Reversible before irreversible,** read straight off the figure.
- **Minority stress and the ban on conversion practices,** which carry the ethical marks.

SEE ALSO Slot II - 04 gender-affirming surgery **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

II - 09

Describe the classification of male sexual dysfunction. Discuss the approaches to their evaluation and management. [10]

10 | ASKED AT 20 MARKS | EATING, SLEEP AND SEXUAL DISORDERS

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

SKELETON

3 Classification	By phase of the response cycle, plus the descriptors
3 Evaluation	History with the partner, examination, a short lab set
4 Management	Correct the cause, couple work, then the specific drug

PHASE	THE DYSFUNCTION	HOW IT PRESENTS	WHAT SETTLES IT
Desire	Hypoactive sexual desire	Absent or reduced desire, causing distress	Depression and testosterone checked
Arousal	Erectile dysfunction	Cannot attain or keep an erection	**Night erections kept or lost**
Orgasm, early	Early ejaculation	Ejaculation before or soon after entry, with distress	**Lifelong or acquired**
Orgasm, late	Delayed ejaculation, anejaculation	Marked delay or none, often drug related	Drug chart and diabetes
Pain	Sexual pain on penetration	Pain or fear of pain during intercourse	Examination for a local cause
Culture bound	Semen-loss anxiety	Fear of loss of vitality, often with a dysfunction alongside	**Belief, not organ, is the target**

Every diagnosis above also carries descriptors: lifelong or acquired, generalised or situational, and the contributing factors from medical condition, medication, knowledge, relationship and culture.

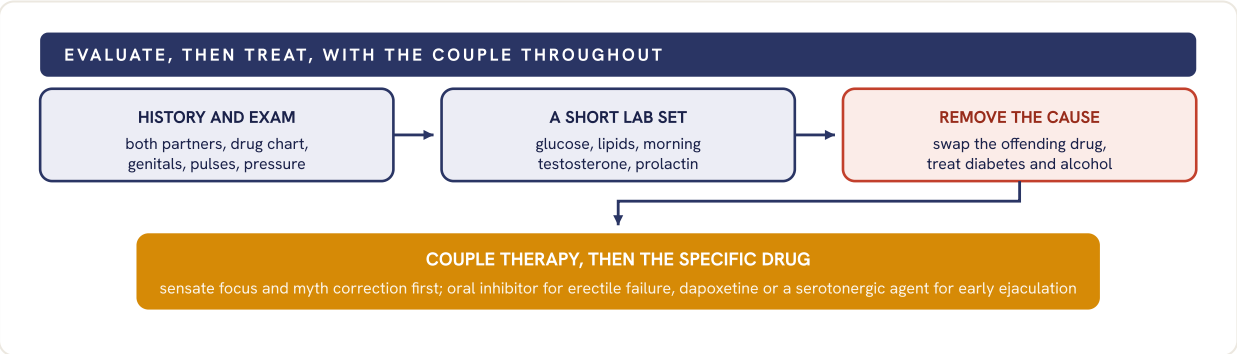

Fig 009.1 Evaluation runs left to right and converges on couple work, with the drug chosen by the phase named in the grid above.

WHAT EARNS THE MARKS

- **The response cycle used as the axis of classification,** stated before the list.
- **The descriptor set,** which is where the current systems differ from the older ones.
- **Semen-loss anxiety included,** because in Indian practice it is common and it changes the target of treatment.
- **The partner in the room,** and the drug named after the psychological step rather than instead of it.

SEE ALSO Slot II - 02 erectile dysfunction Slot II - 03 psychogenic impotence **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

AREA 02 OF 14 · P2-A07

Substance use: opioids, other drugs and treatment

9	9.4%	II-10	II-18
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 8

REVISION ORDER

Take the four opioid slots as one block; they overlap almost completely and only one of them carries the NDPS limb. Tobacco next, as a pair. Motivational enhancement therapy stands alone and is short. Caffeine and the new psychoactive substances last.

WHAT IS IN THIS AREA

Opioids across four slots (the neurobiology and psychosocial factors, substitution therapy with the NDPS Act, the naloxone challenge test with withdrawal management, and naltrexone), tobacco in two (dependence, and a locality tobacco control plan that compares gum with patch), motivational enhancement therapy, caffeine related disorders, and the new psychoactive substances.

II - 10

Describe diagnosis, clinical features and management of caffeine related disorders. [2+3+5]

2 + 3 + 5 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Diagnosis	The recognised entities, and the one still under study
3	Clinical features	The intoxication threshold and the withdrawal clock
5	Management	Taper, treat the syndrome, and the overdose lane

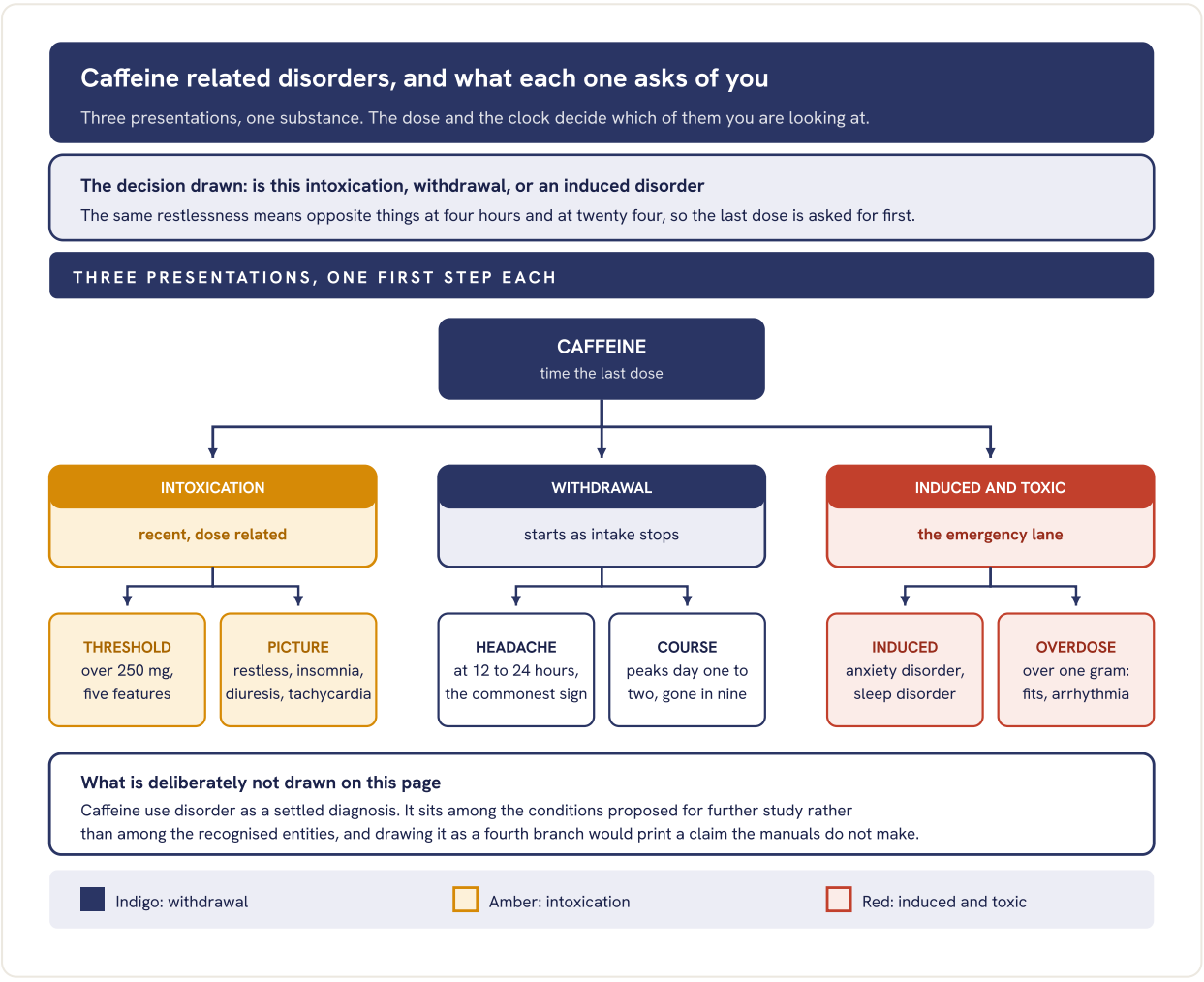

Fig 010.1 The three presentations set against the clock since the last dose, with the toxic lane kept separate because it is the only one that is managed in a resuscitation bay.

- **Management.** Withdrawal is prevented, not treated: taper by about a quarter every few days, substitute decaffeinated drinks, treat the headache simply. Intoxication needs reassurance and a stopped intake. Induced anxiety and insomnia usually clear on abstinence alone.
- **The hidden sources.** Energy drinks, colas, green tea, and combined analgesic and cold preparations. A taper planned on cups of coffee alone fails because the intake was never fully counted.

PITFALL

Do not diagnose an anxiety disorder in a heavy user without first timing the caffeine. Panic attacks are readily provoked at high intake, and the treatment is a taper, not an antidepressant.

WHAT EARNS THE MARKS

- **The recognised entities named,** with caffeine use disorder honestly placed among the conditions for further study.
- **Numbers on the page:** the 250 mg threshold, the headache at 12 to 24 hours, the gram that turns it into an emergency.
- **A taper rather than an abrupt stop,** written as the management of withdrawal.
- **Hidden sources counted,** which is what makes the plan workable.

SEE ALSO Slot II - 17 new psychoactive substances Slot II - 18 tobacco dependence **SPRINT** Day 13, Substance use disorders

II - 11

Describe the neurobiology and psychosocial factors of opioid use disorder. [4+6]

4 + 6 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4 Neurobiology	The receptor, the reward surge, the adapted state
4 Psychosocial factors	Availability, family, adversity, comorbidity
2 Where they meet	Why neither account alone predicts who becomes dependent

THE RECEPTOR, IN ONE LINE

Opioids act at the mu receptor, a G protein coupled receptor that inhibits adenylyl cyclase. In the ventral tegmental area this inhibits GABA interneurons, which disinhibits dopamine neurons and produces the accumbens surge. Everything that follows is the brain adapting to that surge.

STAGE	CIRCUIT	NEUROADAPTATION	WHAT THE PATIENT REPORTS
Intoxication	Ventral tegmental to accumbens	Dopamine surge, positive reinforcement	The rush, then warmth
Withdrawal	Extended amygdala, locus coeruleus	**Cyclic AMP pathway upregulated; dynorphin and CRF recruited**	Dysphoria, pain, autonomic storm
Preoccupation	Prefrontal cortex to striatum	Weak top down control, habit takes over	Craving on seeing a cue
Tolerance	Receptor level	**Desensitisation and internalisation of mu receptors**	The old dose does nothing

The two amber rows explain treatment: alpha two agonists work because the locus coeruleus is disinhibited, and lost tolerance is why relapse after abstinence kills.

- **Psychosocial factors.** Availability and low price, an using peer group, parental substance use with poor supervision, childhood adversity and neglect, school dropout and unemployment, and locally concentrated epidemics that make first exposure ordinary rather than deviant.
- **Comorbidity and self medication.** Conduct disorder, adult attention deficit, depression and post traumatic stress all raise risk, and iatrogenic exposure through prescribed analgesia is a route that clinicians control.
- **Where the two meet.** Most people exposed to opioids do not become dependent. Neurobiology explains what happens once use is repeated; the psychosocial field explains who repeats it.

WHAT EARNS THE MARKS

- **The disinhibition mechanism stated,** not just the phrase dopamine reward.
- **The withdrawal row tied to a drug,** so noradrenergic overactivity explains clonidine.
- **Psychosocial factors given equal weight,** which is what the six marks are for.
- **The integration line.** Answers that give only biology read as incomplete.

SEE ALSO Slot II - 16 opioid withdrawal Slot II - 15 opioid substitution therapy **SPRINT** Day 13, Substance use disorders

II - 12

Naltrexone. [10]

10 SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What it is	Pure antagonist, and how that differs from substitution
3	Indications and dose	Two licensed uses, the dose, the evidence
3	Before you start	The opioid free interval and the challenge test
2	Adverse effects	The liver, and the overdose risk after stopping

THE DEFINITION

A competitive opioid receptor antagonist with highest affinity at the mu receptor and no agonist activity. It is orally active, long acting, has no abuse liability and no street value, and it blocks the reinforcing effect of an opioid or a drink rather than replacing it.

USE	DOSE	WHAT IT ACHIEVES	THE CAUTION
Alcohol dependence	50 mg daily, orally	Fewer heavy drinking days, less craving	Works best with adherence support
Opioid relapse prevention	50 mg daily after detoxification	Blocks euphoria if a lapse occurs	**Requires full detoxification first**
Impulse control problems	50 mg daily, off label	Some benefit in gambling and self injury	Evidence thinner than for alcohol
Long acting form	Monthly injection	Removes the daily adherence decision	Limited availability in India

The amber cell is the one that turns a good answer into a safe one. Given to a dependent user it precipitates immediate withdrawal.

- **Before you start.** Opioid free for seven to ten days after a short acting opioid, and ten to fourteen after a long acting one. Confirm with a negative naloxone challenge and a urine screen. Take baseline liver function tests.
- **Adverse effects.** Nausea, headache, dizziness, abdominal pain, sleep disturbance and dysphoria; transaminase rise at high doses, so avoid in acute hepatitis or liver failure. It also blocks opioid analgesia, which matters before surgery.
- **The risk on stopping.** Tolerance is lost while the blockade is on. A patient who resumes his old dose after discontinuing is at high risk of fatal respiratory depression, and he must be told so.

WHAT EARNS THE MARKS

- **Antagonist contrasted with substitution** in the first two lines, which frames everything else.
- **The opioid free interval with its numbers,** plus the challenge test named.
- **The loss of tolerance warning.** Most answers stop at hepatotoxicity and miss the fatal risk.

SEE ALSO Slot II - 16 naloxone challenge test Slot II - 15 opioid substitution therapy **SPRINT** Day 13, Substance use disorders

II - 13

What is motivational enhancement therapy? What are the indications of motivational enhancement therapy in psychiatry? Discuss the principles of motivational enhancement therapy. [3+3+4]

3 + 3 + 4 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 What it is	Manualised motivational interviewing plus structured feedback
3 Indications	Where in psychiatry it is used, and the trial behind it
4 The principles	Four, each with the therapist behaviour it demands

THE DEFINITION

A brief, structured psychotherapy, usually four sessions, that applies motivational interviewing and adds personalised feedback from a formal assessment. It is client centred in style and directive in aim: the therapist does not install motivation, he draws out the patient's own reasons to change.

PRINCIPLE	WHAT IT MEANS	THERAPIST BEHAVIOUR	THE FAILURE MODE
Express empathy	Accurate understanding without approval or judgement	Reflective listening	Sympathy that colludes
Develop discrepancy	Widen the gap between present use and stated values	Elicit change talk	**Supplying the discrepancy yourself**
Roll with resistance	Resistance is a signal to change approach, not to push	Reframe, shift focus	**Arguing for change**
Support self efficacy	Belief that change is possible is itself predictive	Affirm past successes	Reassurance without evidence

The amber cells are the two errors that turn this into ordinary advice giving, and naming them is worth more than listing the principles alone.

- **Indications.** Alcohol and other substance use disorders, engagement of the ambivalent or poorly motivated patient, medication adherence in bipolar disorder and schizophrenia, tobacco cessation, gambling, eating disorders, and adherence to long term physical treatment.
- **The evidence.** In the large matching trial four sessions of this approach performed about as well as twelve sessions of cognitive behavioural or twelve step facilitation therapy, which is why it is the pragmatic first offer in a busy service.

WHAT EARNS THE MARKS

- **The distinction from motivational interviewing:** the added structured feedback and the fixed brief format.
- **All four principles named,** each with the therapist behaviour beside it in the grid.
- **Indications reaching beyond addiction,** since the question asks about psychiatry as a whole.
- **The matching trial cited,** which is what turns an assertion of efficacy into an answer.

SEE ALSO Slot II - 89 relapse prevention without drugs Slot II - 18 tobacco dependence

II - 14

How can you plan for tobacco control in your locality? What type of assistance would you want from the local Government? Compare nicotine chewing gum and nicotine patches. [5+3+2]

5 + 3 + 2 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

5 The locality plan	Map, protect, enforce, treat, educate
3 Government assistance	What only the administration can supply
2 Gum against patch	The grid, and why the answer is usually both

THE FRAMEWORK TO NAME FIRST

Build the local plan on the national tobacco control law of 2003 and the National Tobacco Control Programme: smoke free public places, no sale to minors or near schools, no advertising, pictorial warnings, and a cessation service in the district hospital.

DOMAIN	CHEWING GUM	TRANSDERMAL PATCH	WHAT SETTLES IT
Delivery	Intermittent, buccal, user controlled	Continuous, transdermal, fixed	Who controls the dose
Onset	Minutes, peaks within half an hour	Hours, then a steady level	**Only gum treats a craving as it happens**
Dosing	2 mg, or 4 mg if he smokes within half an hour of waking	Stepped strengths tapered over weeks	Time to first cigarette
Adherence	Frequent dosing, technique dependent	**Once daily, the best adherence of the group**	Number of decisions per day
Adverse effects	Jaw ache, hiccups, dyspepsia	Skin irritation, vivid dreams overnight	Dentition against skin

Chew and park, do not chew continuously: swallowed nicotine is not absorbed and causes the dyspepsia.

- **The locality plan.** Survey the burden and the vendors, declare schools and health facilities tobacco free, support enforcement of the public place ban, run a cessation clinic with trained counsellors, and take school and workplace education to where the young start.
- **What to ask the administration for.** Enforcement machinery and a designated officer, vendor licensing and action on sales near schools, programme funds for replacement therapy, health worker training, and the education department's cooperation for school work.
- **Combination is the answer.** A patch for the background plus gum for breakthrough craving beats either alone, so the comparison ends in a recommendation rather than a draw.

WHAT EARNS THE MARKS

- **The named law and programme,** before any local activity is described.
- **A plan divided into what you do and what you request,** as the question splits it.
- **The two amber cells:** gum for acute craving, patch for adherence.
- **The combination recommendation,** which is the point of comparing them.

SEE ALSO Slot II - 18 tobacco dependence Slot II - 86 reducing alcohol use in your area SPRINT Day 13, Substance use disorders

II - 15

Describe opioid substitution therapy. Mention the psychological and pharmacological principles underlying it. Discuss the NDPS Act in relation to it. [3+3+4]

3 + 3 + 4 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	What it is	The agents, the setting, and the stated goal
3	The principles	Cross tolerance below, harm reduction above
4	The law	What the Act enables, and what it leaves out

STEP	WHAT HAPPENS	WHO ACTS	CONDITION OR INTERVAL
1	Confirm opioid dependence and score withdrawal	Treating psychiatrist	Objective signs required
2	Induction on buprenorphine, or on methadone	Licensed centre staff	Only in mild withdrawal
3	Titrate to the dose that abolishes craving	Prescriber, daily review	Usually the first two weeks
4	Maintenance with directly observed dosing	Dispensing centre	Take home doses on stability
5	Psychosocial work, then a planned taper or continued maintenance	Team and patient together	Retention is the outcome

Induction into a patient who is not yet in withdrawal precipitates it. Order, actor and interval are exactly what this archetype is marked on.

- **Pharmacological principle.** A long acting agonist occupies the receptor, so the plasma level stays flat, withdrawal is abolished, cross tolerance blunts the euphoria of any illicit dose, and the peak and trough cycle that drives injecting disappears. Buprenorphine is a partial agonist with a ceiling on respiratory depression.
- **Psychological principle.** Harm reduction. Removing the daily hunt for money and drug frees attention for work, family and treatment, and retention in care is the measure by which the approach is judged.

WHAT THE ACT DOES NOT COVER

The Act licenses and supplies; it does not fund a service, train a team or create demand reduction on its own. It does not touch alcohol or tobacco. Consumption of a narcotic drug remains an offence, and the immunity for an addict who volunteers for treatment is conditional, not a general decriminalisation.

WHAT EARNS THE MARKS

- **Steps in order with the actor named at each,** and the mild withdrawal condition on induction.
- **Both principles answered separately,** pharmacological and psychological, because the question splits them.
- **The 2014 amendment named:** essential narcotic drugs brought under central rules, which is what made this therapy practicable.
- **The exclusions strip.** Listing provisions without their boundary reads as recall from a summary.

SEE ALSO Slot II - 16 opioid withdrawal Slot II - 12 naltrexone

II - 16

What is naloxone challenge test? Discuss management of opioid withdrawal with specific mention of non-opioid pharmacological strategies. [3+7]

3 + 7 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The challenge test	Purpose, dose, observation period, how it is read
4	Assess the withdrawal	The rating scale, the clock, the severity call
3	Non-opioid strategies	Alpha two agonist first, then symptom by symptom

THE NALOXONE CHALLENGE TEST

A safety test done before starting naltrexone, to prove that no physiological opioid dependence remains. Naloxone 0.8 mg is given, part intravenously and the remainder after observation, and the patient is watched for twenty to forty five minutes. Any withdrawal sign is a positive test: defer, and repeat later.

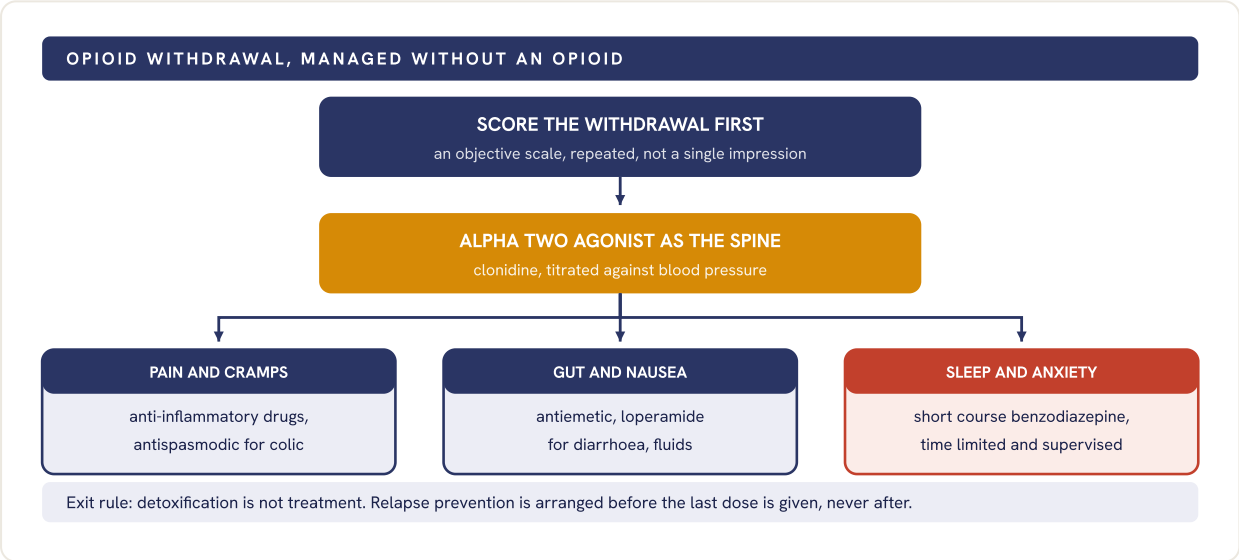

Fig 016.1 The alpha two agonist carries the syndrome and the three lanes mop up what it leaves, with the red lane marked because that is where iatrogenic dependence begins.

- **Why clonidine works.** Chronic opioid use disinhibits the locus coeruleus, and withdrawal releases a noradrenergic storm. An alpha two agonist suppresses it directly, which is why it treats sweating, cramps and restlessness but does very little for craving.

WHAT EARNS THE MARKS

- **The challenge test written with its numbers,** dose and observation period, and a stated reading rule.
- **An objective withdrawal score placed before any drug,** as the first box of the figure.
- **Clonidine explained by the locus coeruleus,** not merely listed.
- **The exit rule,** that detoxification without relapse prevention is an incomplete plan.

SEE ALSO Slot II - 12 naltrexone Slot II - 11 neurobiology of opioid use disorder **SPRINT** Day 13, Substance use disorders

II - 17

Define new psychoactive substances. How do you classify them? Write the clinical features related to use of any one of them. [2+4+4]

2 + 4 + 4 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The definition	What new means here, and what not controlled means
4 The classification	Families by mechanism, with an example in each
4 One family in detail	Synthetic cannabinoids, features and management

THE DEFINITION

Substances of abuse, pure or in preparation, not controlled by the 1961 or 1971 drug conventions but posing a public health threat. New means newly available, not newly synthesised.

FAMILY	EXAMPLE	MECHANISM	SIGNATURE EFFECT
Synthetic cannabinoids	The JWH and AM series	**Full agonist at CB1, unlike the partial agonism of cannabis**	Agitation, psychosis, seizures
Synthetic cathinones	Mephedrone	Monoamine releaser and re-uptake blocker	Stimulant, then paranoia
Phenethylamines	The 2C and NBOMe series	Serotonin 2A agonism	Hallucinations, hyperthermia
Synthetic opioids	Fentanyl analogues	Potent mu agonism	**Respiratory arrest at microgram doses**
Plant based and sedative	Kratom, etizolam	Mixed opioid, or benzodi-azepine like	Dependence, withdrawal fits

Ketamine type dissociatives form a sixth family. Classify by mechanism: street names change with every batch, receptors do not.

- **Synthetic cannabinoids in detail.** Full CB1 agonism without the cannabidiol that buffers herbal cannabis. Severe agitation, acute psychosis, catatonia, seizures, vomiting, tachycardia and hypertension, with reported acute kidney injury and myocardial ischaemia. Far more toxic than the plant it imitates.
- **The laboratory trap.** A routine urine cannabinoid immunoassay does not detect them. A negative screen in an agitated young man who says he smoked something does not exclude the diagnosis.
- **Management.** Supportive care in a quiet monitored area, benzodiazepines first for agitation and seizure risk, fluids, and watch renal function and creatine kinase.

WHAT EARNS THE MARKS

- **Both conventions named in the definition,** and new correctly glossed as newly available.
- **A classification by mechanism** with one example in each family, rather than a list of street names.
- **Full against partial CB1 agonism,** the amber cell that explains the whole clinical picture, and the negative urine screen that follows from it.

SEE ALSO Slot II - 10 caffeine related disorders Slot II - 90 dependence, abuse and harmful use SPRINT Day 13, Substance use disorders

II - 18

Discuss the clinical features, pharmacological and non-pharmacological management of tobacco dependence. [3+4+3]

3 + 4 + 3 | SUBSTANCE USE: OPIOIDS, OTHER DRUGS AND TREATMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Clinical features	Dependence criteria, severity marker, withdrawal course
4 Pharmacological	Three first line agents, each with its rule
3 Non-pharmacological	The five step framework and what follows a lapse

Fig 018.1 The five step framework drawn as one sorting question, with the amber severity marker placed where it actually changes the prescription.

- **Clinical features.** The dependence criteria applied to nicotine, with withdrawal starting within a day of stopping: irritability, anxiety, poor concentration, restlessness, low mood, insomnia and increased appetite. Most of it settles in two to four weeks; craving outlasts it.
- **Pharmacological.** Replacement therapy, best as a patch plus a rescue form; varenicline, a partial agonist at the nicotinic receptor, titrated over the first week and continued twelve weeks; bupropion, avoided where there is any seizure risk or an eating disorder.
- **Smokeless tobacco.** In Indian practice much of the burden is chewed, not smoked. Ask about it by name, examine the mouth for submucous fibrosis, and remember that replacement therapy still applies.

WHAT EARNS THE MARKS

- **Time to first cigarette used as the severity marker,** as the amber box does, not the number smoked alone.
- **Three first line agents named with a rule each,** rather than the word counselling on its own.
- **The unwilling patient given a plan,** which is the lane most answers leave empty.
- **Smokeless tobacco asked about,** the Indian half of the question.

SEE ALSO Slot II - 14 tobacco control and replacement therapy Slot II - 13 motivational enhancement therapy

SPRINT Day 13, Substance use disorders

AREA 03 OF 14 · P2-A08

Anxiety, OCD and related disorders

9	8.9%	II-19	II-27
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 · HIGH 3 · SINGLE 5

REVISION ORDER

Build one OCD answer properly and five of the nine slots open. Panic and agoraphobia next, because they share a structure. Generalised anxiety disorder and hoarding are independent and both are short.

WHAT IS IN THIS AREA

Obsessive-compulsive disorder in five of the nine slots: the definition with severe-case management, the aetiopathology, the non-pharmacological management with the course studies, treatment resistance, and the psychological-theory slot it shares with depression. Panic disorder, generalised anxiety disorder, agoraphobia and hoarding disorder take the rest.

II - 19

Discuss non-pharmacological management of OCD. Describe studies on the course of OCD. [6+4]

6 + 4 | ANXIETY, OCD AND RELATED DISORDERS

DNB, WBUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	The first line	Exposure with response prevention, and why it works
3	What is added	Cognitive work, the family, and the refractory lane
4	Course studies	What the long follow ups actually found

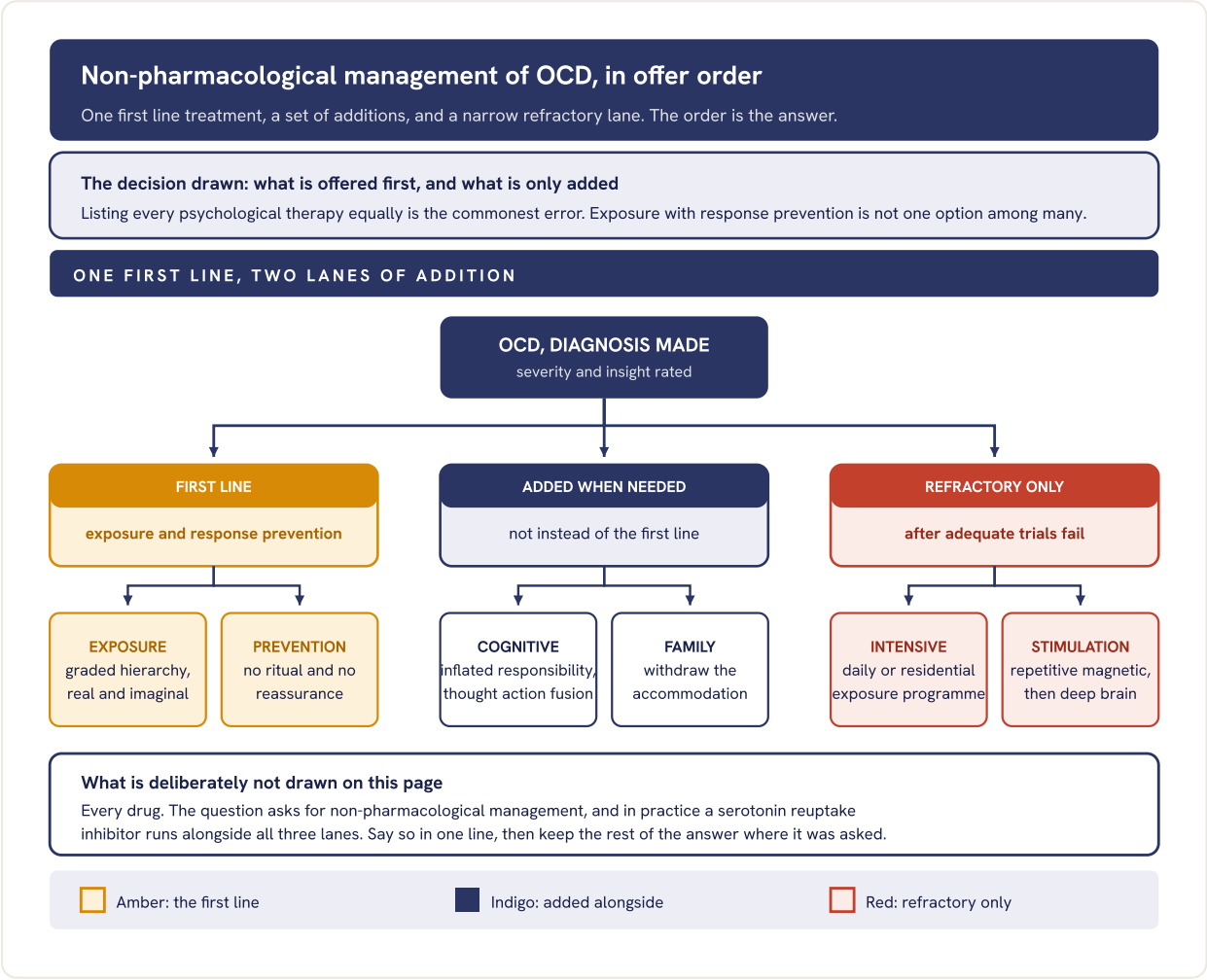

Fig 019.1 Exposure with response prevention drawn as the trunk rather than as one option among six, with the refractory lane kept visibly separate.

- **How exposure is delivered.** A shared hierarchy, prolonged exposure until anxiety falls without a ritual, homework between sessions, and no reassurance from staff or family. Response prevention is the active ingredient; exposure without it is distress with no learning.

COURSE STUDIES: WHAT THEY FOUND

Long term follow up finds a mainly chronic, fluctuating course. In the largest forty year study most patients improved and about a fifth recovered fully, while around half still met criteria at follow up. Later prospective cohorts confirm low full remission and frequent partial response. Early onset, poor insight, hoarding and long duration before treatment predict worse outcome.

WHAT EARNS THE MARKS

- **Response prevention named as the active ingredient,** the second amber leaf.
- **Family accommodation addressed,** which most answers never mention.
- **Course described with numbers,** chronic with partial remission rather than the word variable.
- **Predictors of poor outcome listed,** which is what makes it a study answer.

SEE ALSO Slot II - 22 severe OCD Slot II - 25 treatment resistant OCD **SPRINT**

Day 14, Anxiety, OCD and trauma-related disorders

II - 20

Management of panic disorder. [10]

10 ANXIETY, OCD AND RELATED DISORDERS

KNRUHS, MUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	**Confirm and exclude**	The medical and substance causes that mimic it
2	**The acute attack**	What is done in the room, and what is not
4	**Definitive treatment**	Cognitive behavioural therapy and the drug, in parallel
2	**Continuation**	How long, and how it is stopped

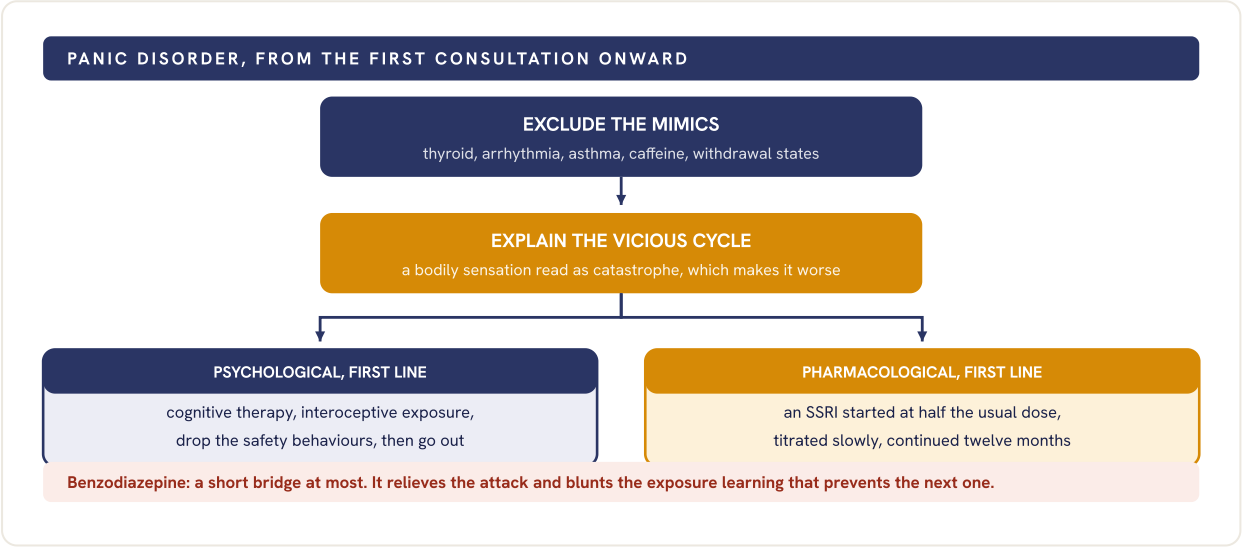

Fig 020.1 The two first line treatments drawn side by side rather than in sequence, with the benzodiazepine caution carried as a red strip along the foot.

- **In the acute attack.** Stay, speak calmly, name what is happening, and slow the breathing. Do not investigate again in a patient already worked up, because repeated tests confirm his belief that something has been missed.
- **The cognitive model.** Panic is the catastrophic misinterpretation of a benign bodily sensation. Treatment tests that interpretation directly, by inducing the sensation in the room and letting nothing be done about it.
- **Stopping.** Continue for at least a year after full response, then taper slowly. Early stopping and abrupt cessation are the two commonest reasons a treated patient relapses.

WHAT EARNS THE MARKS

- **Medical mimics excluded first,** as the top box of the figure does.
- **Both first line treatments named as first line,** not therapy offered only after drugs fail.
- **Interoceptive exposure and safety behaviours,** the specific techniques rather than the words cognitive therapy.
- **The benzodiazepine caution with its reason,** the red strip.

SEE ALSO Slot II - 26 agoraphobia Slot II - 23 generalised anxiety disorder SPRINT

Day 14, Anxiety, OCD and trauma-related disorders

II - 21

Discuss the etiopathology of obsessive-compulsive disorder. [10]

10 ANXIETY, OCD AND RELATED DISORDERS

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 The axis	One disorder read at five levels, not five theories
4 The biological levels	Genes, transmitters, the circuit, the immune route
2 The psychological level	Appraisal and learning, from normal intrusions
2 What ties them	The evidence each level rests on, named

STATE THE AXIS BEFORE THE LIST

No single cause is established. The defensible answer reads one disorder at five levels, genetic, neurochemical, circuit, immunological and psychological, and names the observation each level rests on.

LEVEL	THE PROPOSAL	THE EVIDENCE IT RESTS ON	WHERE IT IS WEAK
Genetic	Moderate heritability, higher in early onset	Twin and family studies; glutamate transporter genes	No replicated single gene
Neurochemical	Serotonin dysfunction, with glutamate excess	**Selective response to serotonergic drugs alone**	Response is not proof of cause
Circuit	Overactive cortico-striato-thalamo-cortical loop	**Orbitofrontal and caudate overactivity that normalises with treatment**	Also seen in other conditions
Immunological	Post-streptococcal autoimmunity in a subgroup	Abrupt paediatric onset with tics after infection	A small and contested subgroup
Psychological	Normal intrusions appraised as personal responsibility	Intrusive thoughts are near universal in healthy people	Does not explain onset

The amber cells are the two findings an examiner expects by name, and both are pharmacological or imaging evidence rather than theory.

- **The psychological account, in detail.** Intrusions are universal. What makes them clinical is the appraisal: inflated responsibility for harm, and thought action fusion, the belief that thinking something makes it likelier or is morally equivalent to doing it. The compulsion relieves anxiety and is negatively reinforced, so it recurs.
- **The honest limit.** Every level above describes a correlate. None of them predicts who develops the disorder, and treatment response at one level does not establish causation at that level.

WHAT EARNS THE MARKS

- **The axis stated before the list,** which turns five theories into one structured account.
- **The loop named in full,** cortico-striato-thalamo-cortical, with the regions.
- **Universal intrusions as the starting point,** the finding that makes the appraisal model plausible.
- **The weakness column.** Naming what each level cannot explain reads as command.

SEE ALSO Slot II - 22 severe OCD Slot II - 24 psychological theories and management **SPRINT**

Day 14, Anxiety, OCD and trauma-related disorders

II - 22

Define obsessive-compulsive disorder. Discuss the management of a patient with severe OCD. [3+7]

3 + 7 | ANXIETY, OCD AND RELATED DISORDERS

DNB, KUHS, NTRUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

3 **Definition**	Obsession and compulsion defined, then the disorder
2 **Assess severity**	Rate it, check insight, risk and where he is treated
4 **Combined treatment**	High dose serotonergic drug plus intensive exposure
1 **If no response**	The augmentation step, and what precedes it

THE DEFINITION

Recurrent obsessions, which are intrusive unwanted thoughts, images or impulses recognised as one's own and resisted, or compulsions, which are repetitive acts performed to reduce the distress they cause. They are time consuming or distressing, and not pleasurable in themselves.

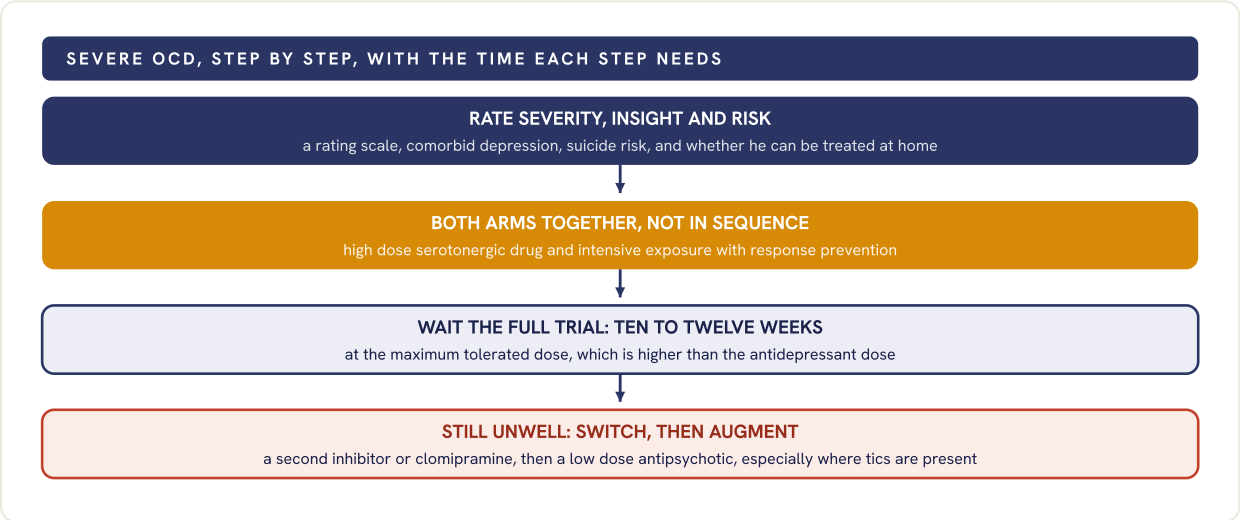

Fig 022.1 The ladder with the waiting time written into each step.

- **Dosing.** Serotonin reuptake inhibitors are used at the top of their range in this disorder, well above the usual antidepressant dose, and the response is slower. Clomipramine remains the reference agent, with an electrocardiogram before and during treatment.
- **Before calling it resistant.** Confirm the drug was taken at that dose for that long, that exposure was actually delivered with response prevention, and that the family have stopped providing reassurance. Most apparent resistance is one of these three.

WHAT EARNS THE MARKS

- **Obsession and compulsion defined separately** before the disorder is defined.
- **Drug and exposure started together,** the amber step, not therapy held back as a second line.
- **The ten to twelve week trial at high dose,** written as a number, and the three checks before resistance is declared.

SEE ALSO Slot II - 25 treatment resistant OCD Slot II - 19 non-pharmacological management of OCD **SPRINT** Day 14, Anxiety, OCD and trauma-related disorders

II - 23

Describe the clinical management of generalised anxiety disorder. [10]

10 | ANXIETY, OCD AND RELATED DISORDERS | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Confirm the diagnosis**	Six months of uncontrollable worry, and what mimics it
2	**Step one and two**	Education, monitoring, guided self help
4	**Step three**	Therapy or drug, both first line, patient's choice
2	**Step four**	The refractory options, and what is avoided throughout

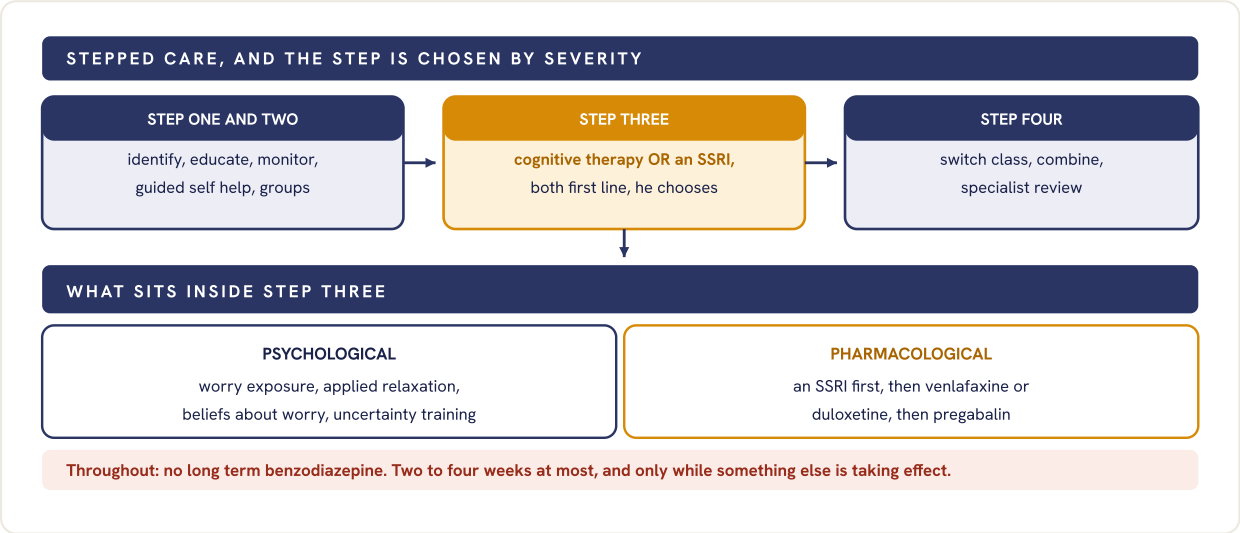

Fig 023.1 Four steps across the top, then the contents of the step that matters opened out below it.

- **Confirm first.** Six months of excessive, uncontrollable worry across several domains with restlessness, fatigue, poor concentration, irritability, muscle tension or disturbed sleep. Exclude thyroid disease, caffeine, withdrawal states and a primary depressive episode.
- **The models worth naming.** Worry as avoidance of worse imagery, positive and negative beliefs about worrying itself, and intolerance of uncertainty. Each generates a technique, which is why naming them is not decoration.
- **Duration.** Continue the drug for at least twelve months after response and taper slowly. This is a chronic relapsing condition, and stopping at three months is why so many return.

WHAT EARNS THE MARKS

- **Stepped care named as a structure,** so the answer is organised by severity rather than by treatment type.
- **Therapy and drug both marked first line,** the amber box.
- **The benzodiazepine limit stated in weeks,** the red strip.
- **Twelve months of continuation,** which most answers omit entirely.

SEE ALSO Slot II - 20 panic disorder Slot II - 26 agoraphobia **SPRINT**
Day 14, Anxiety, OCD and trauma-related disorders

II - 24

Discuss different psychological theories of depression and OCD. Describe how these theories can help in their management. [5+5]

5 + 5 | ANXIETY, OCD AND RELATED DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Depression theories	Cognitive, behavioural, interpersonal, psychodynamic
2 OCD theories	Learning and appraisal, from normal intrusions
3 Theory into technique	Each model names the intervention it generates
2 Choosing	By formulation, not by habit or availability

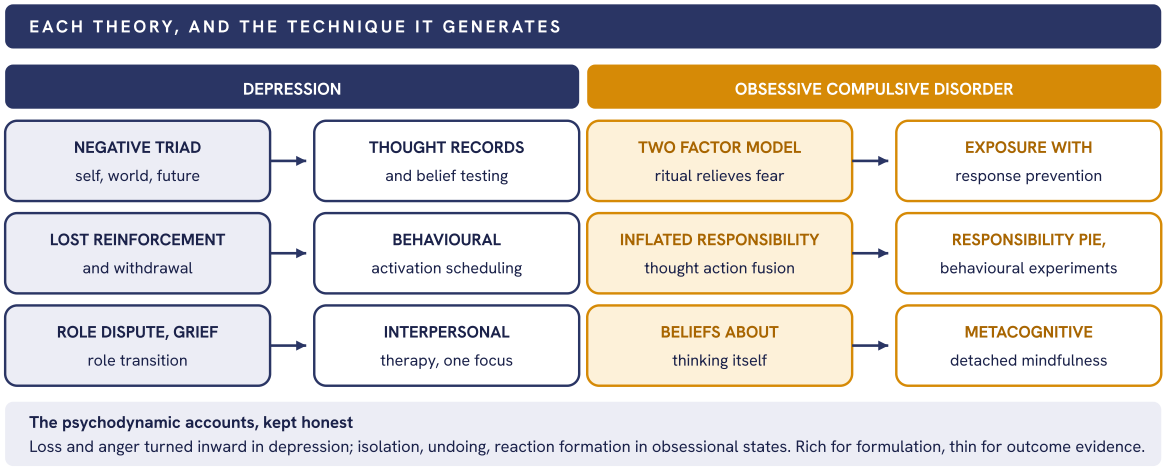

Fig 024.1 Each row reads left to right as one claim and the technique it licenses, which is exactly what the second half of the question asks for.

- **Choosing between them.** Take the model that fits the formulation. Withdrawal and inactivity call for activation, a dispute or a bereavement for interpersonal work, ritual maintained by relief for exposure, and a patient tormented by the meaning of his thoughts for the appraisal route.

WHAT EARNS THE MARKS

- **Every theory paired with its technique on the same line,** as each row of the figure does.
- **Both disorders answered,** because half the marks sit in the column most answers neglect.
- **Normal intrusions as the starting point of the OCD models,** which is what makes them treatments and not descriptions.
- **The psychodynamic strip kept honest,** useful for formulation, weak on outcome evidence.

SEE ALSO Slot II - 21 etiopathology of OCD Slot II - 19 non-pharmacological management of OCD

II - 25

What is treatment resistant OCD? What are the proposed neurobiology of such resistance? Discuss the treatment options for such a case. [2+3+5]

2 + 3 + 5 | ANXIETY, OCD AND RELATED DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The definition	What counts as adequate, and the staging above it
3 Proposed neurobiology	Circuit, glutamate, and the phenotypes that resist
5 Treatment options	Optimise, augment, then the neuromodulation lane

THE DEFINITION

Failure to achieve a meaningful fall in symptom score, conventionally under twenty five to thirty five per cent on a standard rating scale, after two adequate trials of a serotonin reuptake inhibitor at maximum tolerated dose for ten to twelve weeks each, together with an adequate trial of exposure with response prevention. Staged from non-response through refractory to intractable.

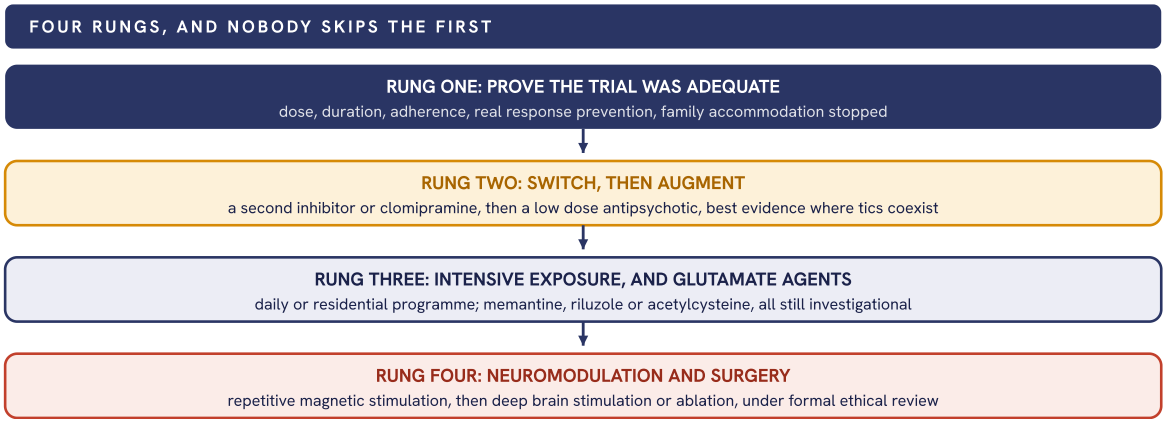

Fig 025.1 The ladder for resistant disease, with the audit of the first trial drawn as a rung rather than assumed, because most resistance is found there.

- **The proposed neurobiology.** Persistent overactivity in the orbitofrontal to caudate loop that does not normalise with treatment; raised glutamate in the caudate and cerebrospinal fluid with reduced inhibitory tone; and dopaminergic involvement, which is why antipsychotic augmentation helps where there are tics.
- **The phenotypes that resist.** Early onset, poor or absent insight, hoarding symptoms, prominent tics, and long duration before any treatment. Naming these is what turns a list of drugs into a clinical answer.

WHAT EARNS THE MARKS

- **The definition with its numbers,** two trials, ten to twelve weeks, a stated response threshold.
- **The first rung, auditing the trial,** before any new drug is proposed.
- **Glutamate named alongside serotonin,** which is the current part of the answer.
- **Surgery placed last and gated by ethical review,** not offered as one option among many.

SEE ALSO Slot II - 22 severe OCD Slot II - 21 etiopathology of OCD SPRINT
Day 37, Psychopharmacology II: resistance and neurostimulation

II - 26

What is agoraphobia? Discuss diagnostic features, differential diagnosis and management of agoraphobia. [2+3+2+3]

2 + 3 + 2 + 3 | ANXIETY, OCD AND RELATED DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	The fear is of escape being blocked, not of the place
3	Diagnostic features	Two of five situations, avoidance, six months
2	Differential diagnosis	Sorted by what he is actually afraid of
3	Management	Graded exposure, the drug, and the family rule

CONDITION	WHAT HE IS AFRAID OF	WHAT SETTLES IT
Agoraphobia	Being trapped or helpless if symptoms come	Fear of the escape route, not of the place
Social anxiety disorder	Being watched and judged	The fear goes when nobody can observe
Specific phobia	The situation itself	One circumscribed trigger, no panic elsewhere
Psychotic disorder	Being followed or harmed	A delusional reason for staying in

Ask what he expects to happen if he goes out. That one question does most of this table's work, and it also separates depression, where there is simply no drive to go.

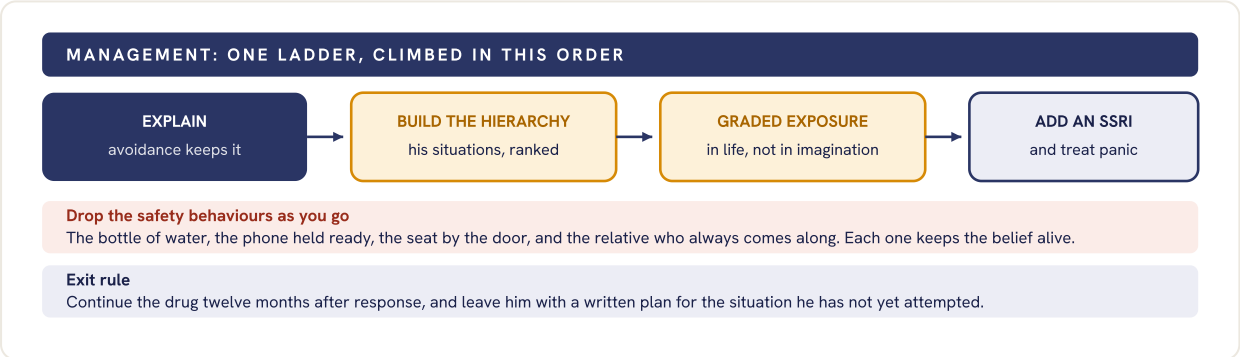

Fig 026.1 Exposure drawn as the spine of treatment with the drug alongside it, and the safety behaviours carried as a red strip because they are what quietly defeats the ladder.

- **Diagnostic features.** Marked fear in at least two of five settings: public transport, open spaces, enclosed places, queues or crowds, and being outside the home alone. Avoided, endured with dread, or requiring a companion; out of proportion; six months.

WHAT EARNS THE MARKS

- **The definition built on escape and help,** not on a list of feared places.
- **Two of five situations and the six month rule,** stated as criteria.
- **The differential sorted by the feared outcome,** which is the column that does the work.
- **The companion named as a safety behaviour,** the line most answers miss.

SEE ALSO Slot II - 20 panic disorder Slot II - 23 generalised anxiety disorder SPRINT

Day 14, Anxiety, OCD and trauma-related disorders

II - 27

Discuss the clinical features and differential diagnosis of hoarding disorder. [5+5]

5 + 5 | ANXIETY, OCD AND RELATED DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	Difficulty discarding, and the clutter that follows
3	Clinical features	Acquisition, insight, course, and the safety risk
3	The differential	Sorted by why the objects are kept
2	Comorbidity	What travels with it and changes the plan

THE DEFINITION

Persistent difficulty parting with possessions regardless of their actual value, driven by a perceived need to save them and by distress on discarding. The accumulation congests living areas so they cannot be used as intended, and it causes impairment or an unsafe home.

CONDITION	WHY THE OBJECTS ARE KEPT	WHAT SETTLES IT
Hoarding disorder	Attachment and a felt need to save	**Distress on discarding, comfort in keeping**
Obsessive compulsive disorder	An obsession, often contamination or harm	**Keeping is unwanted and brings no pleasure**
Depression	Nothing is kept deliberately; nothing is cleared	Accumulation is passive, follows low mood
Schizophrenia	A delusional reason for saving	The stated reason is bizarre
Frontal or degenerative disease	Loss of judgement and disinhibition	Late onset, with squalor and self neglect
Normal collecting	Interest and pride in the collection	Organised, displayed, no impairment

Two questions separate almost the whole table: why is it kept, and how does he feel while keeping it.

- **Clinical features.** Excessive acquisition in most cases, by buying or by collecting free things. Insight ranges from fair to absent, and the specifier is recorded. Onset is in adolescence, severity grows each decade, and the course is chronic without treatment.
- **The risk that must be assessed.** Fire load, blocked exits, falls, infestation, and the safety of children or dependent elders in the home. Public health or fire services may already be involved, and their involvement changes the treatment plan.

WHAT EARNS THE MARKS

- **Difficulty discarding named as the core,** not the clutter, which is only its consequence.
- **Acquisition and insight recorded as specifiers,** which shows the current classification is known.
- **The differential organised by motive,** the amber pair separating this from obsessive compulsive disorder.
- **The safety assessment,** which is what makes it a clinical answer rather than a description.

SEE ALSO Slot II - 21 etiopathology of OCD Slot II - 22 severe OCD SPRINT

Day 14, Anxiety, OCD and trauma-related disorders

AREA 04 OF 14 · P2-A01

Schizophrenia: aetiology, phenomenology and course

8	8.4%	II-28	II-35
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 · HIGH 1 · RECURRENT 1 · SINGLE 4

REVISION ORDER

Course and outcome first, and negative symptoms with it: those two carry the anchor weight here. Psychosocial management and the chronic-care slot next, they share their content. Early psychosis after that. Endophenotype and expressed emotion last, both short and both critical-evaluation shapes.

WHAT IS IN THIS AREA

Course and outcome, negative symptoms and their management, early psychosis and its prevention, psychosocial management, childhood psychosis, the chronic-care slot that asks what India actually provides and how family functioning is assessed, endophenotypes and biomarkers, and expressed emotion as a critical evaluation.

II - 28

Course and outcome of schizophrenia. [10]

10 SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB, KNRUHS, RGUHS, TN-MGRMU - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2 **Two questions**	Course is the shape over time, outcome the state at follow-up
2 **Course patterns**	Prodrome, episode structure, the ICD-11 course specifiers
3 **Outcome at follow-up**	The long cohorts, and what they actually measured
3 **Predictors and mortality**	Prognostic factors, the mortality gap, what is modifiable

SEPARATE THE TWO WORDS FIRST

Course is the longitudinal shape: prodrome, episodes, remissions, residual state. Outcome is the position at a stated follow-up point, on three separable axes, symptomatic, functional and personal. ICD-11 asks for both, an episode pattern plus a remission qualifier.

DOMAIN	POINTS TO BETTER OUTCOME	POINTS TO WORSE OUTCOME
Onset	Acute, later, clear precipitant	Insidious, before twenty, no precipitant
Premorbid	Good work and social record, married	Schizoid traits, poor school record
Symptoms	Positive and affective symptoms lead	Negative symptoms, early cognitive loss
Modifiable	Short untreated psychosis, adherence kept	Long untreated psychosis, substance use

Only the last row can be moved by a clinician. High expressed emotion and absent carer support belong with the right-hand column.

- **Course.** A premorbid phase, a prodrome of one to five years with attenuated symptoms and functional slippage, then the first episode. Deterioration is steepest in the first five years and then largely plateaus.
- **Outcome.** Roughly a third do well, a third relapse with residual disability, a third deteriorate. The WHO cohorts, IPSS, DOSMeD and ISoS, reported better outcome in Indian and other low-income centres, and Indian long-term follow-up supports it.
- **Qualify it.** That advantage is contested: differential attrition, milder acute-onset cases entering the cohorts, and outcome scored as social role rather than symptom count.
- **Mortality.** Life expectancy is fifteen to twenty years shorter, mostly cardiovascular, not suicide. Lifetime suicide is about five per cent, concentrated early and after discharge.

WHAT EARNS THE MARKS

- **Course and outcome separated in the first two lines.** Most answers merge them and then repeat themselves.
- **The prognostic grid, both columns,** with the modifiable row identified as modifiable.
- **The named cohorts with their caveat,** and the mortality gap named as cardiovascular.

SEE ALSO Slot II - 29 negative symptoms Slot II - 35 expressed emotion **SPRINT**

Day 8, Schizophrenia: aetiology, phenomenology, classification and course

II - 29

Describe negative symptoms in schizophrenia and discuss about the management. [5+5]

5 + 5 | SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB, KUHS, NTRUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2	The two factors	Diminished expression and avolition, five symptoms between them
3	Primary or secondary	Deficit syndrome, and the four causes that mimic it
3	Drug treatment	Lower the dose first, then switch, and monitor
2	Psychosocial	Skills, remediation and employment carry the function

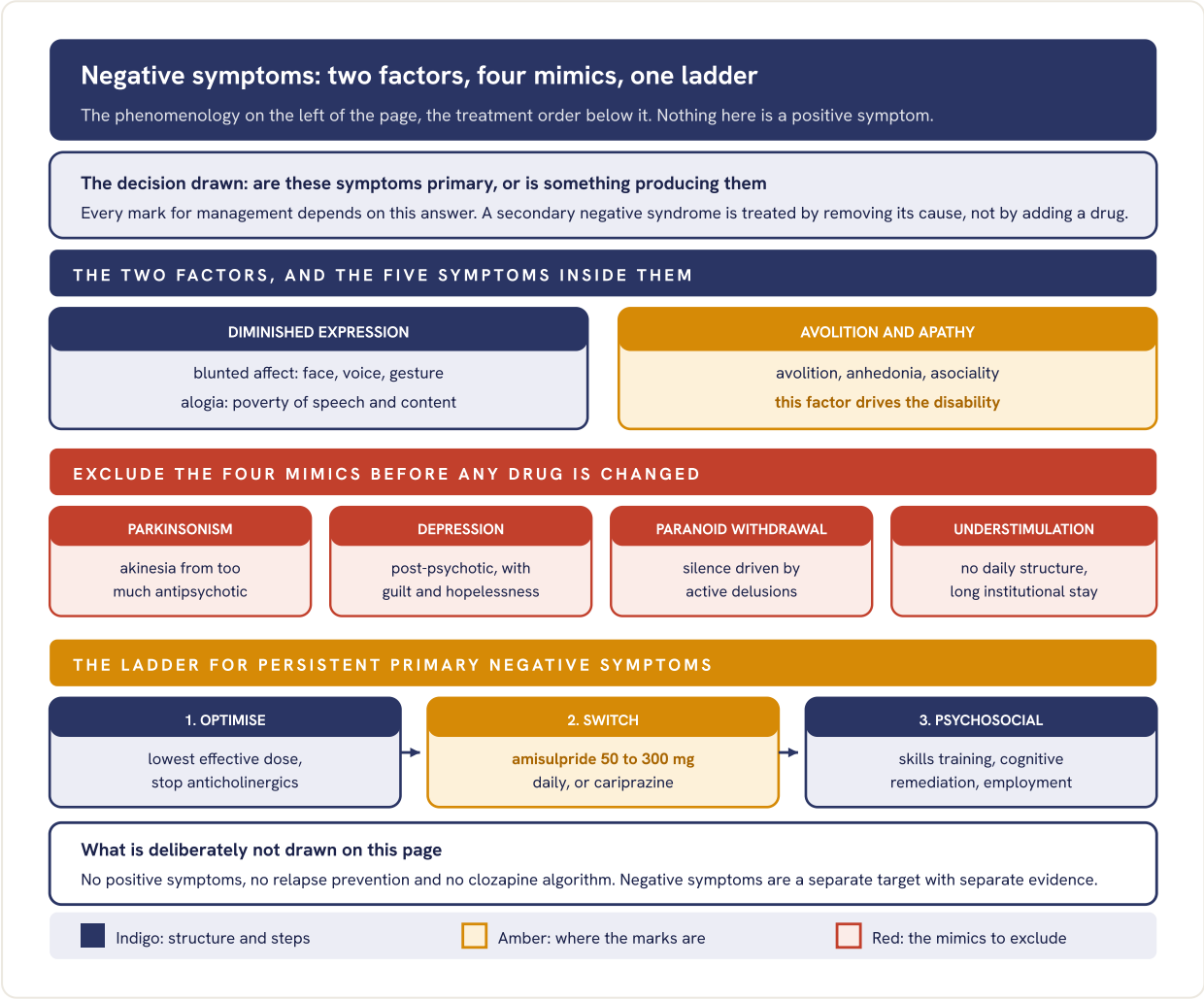

Fig 029.1 The two-factor structure of negative symptoms, the four secondary causes that must be excluded before the syndrome is called primary, and the three-step treatment ladder that follows.

- **The five symptoms.** Blunted affect and alogia make up diminished expression. Avolition, anhedonia and asociality make up the motivational factor. Attention impairment is described alongside them but loads separately.
- **Deficit syndrome.** Negative symptoms that are primary, enduring for at least a year, and present during clinical stability. It carries a poorer functional trajectory and is the population the drug trials target.

PITFALL

Clozapine is not the answer to this question. Its evidence is for positive symptom resistance; for primary negative symptoms it performs no better than other second generation agents. Low-dose amisulpride and cariprazine carry the dedicated trials.

WHAT EARNS THE MARKS

- **The two-factor structure named, with all five symptoms placed.** A flat list of six A words is a weaker answer.
- **The four mimics excluded before treatment is discussed.** This is the sequence the second half of the question is testing.
- **Dose reduction written as step one.** Adding a drug to treat drug-induced akinesia is the commonest error here.
- **Psychosocial treatment given its own line.** Function does not recover on pharmacology alone.

SEE ALSO Slot II - 28 course and outcome Slot II - 48 cognitive remediation **SPRINT**

Day 8, Schizophrenia: aetiology, phenomenology, classification and course

II - 30

What do you understand by the concept of early psychosis? Discuss the prevention and treatment of early psychosis. [4+3+3]

4 + 3 + 3 | SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

4 The concept	Clinical staging, the at-risk mental state, the critical period
3 Prevention	Indicated prevention only, and what is not recommended
3 Treatment	Shorten the untreated period, low dose, family and work

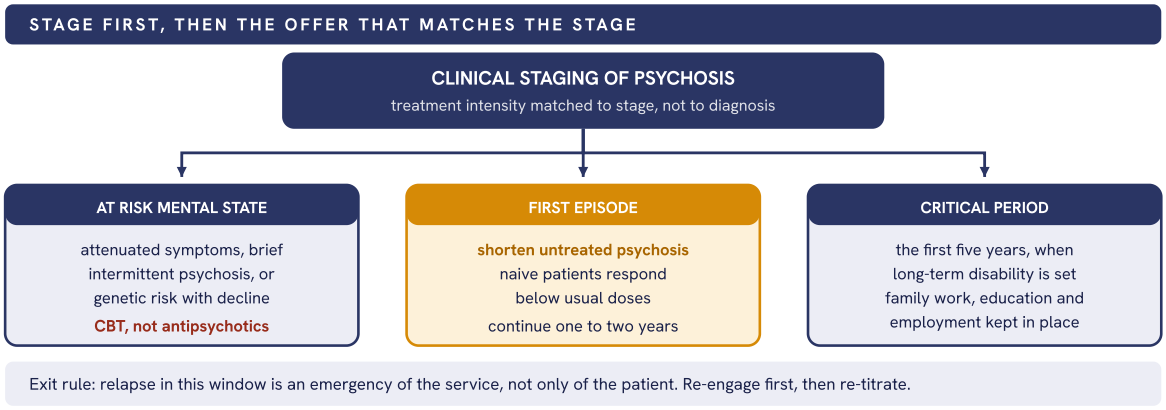

Fig 030.1 Early psychosis as three stages, each with the offer that belongs to it, and the first five years marked as the window where effort pays.

COLOUR CODE stage and structure | where treatment changes outcome | what is not recommended

- **The concept.** Psychosis is staged rather than only diagnosed: asymptomatic risk, undifferentiated distress, at-risk mental state, first episode, relapse, persistent illness.
- **Prevention.** Only indicated prevention is workable. Cognitive behavioural therapy reduces transition modestly. Universal screening is not justified: most at-risk cases never convert.
- **The Indian problem.** Untreated psychosis commonly runs beyond a year, through faith healing pathways and thin services. The district programme is the realistic vehicle.

PITFALL

Prescribing an antipsychotic for an at-risk mental state. Benefit is unproven, most of those treated would never have converted, and the metabolic and motor harms are certain.

WHAT EARNS THE MARKS

- **Staging stated as the organising idea,** not just the phrase "early intervention".
- **The three at-risk groups named.** They are the operational definition being asked for.
- **Untreated psychosis identified as the modifiable variable,** and the critical period given as five years.

SEE ALSO Slot II - 45 first episode pharmacology Slot II - 32 childhood psychosis

II - 31

Discuss psychosocial management in a case of schizophrenia. [10]

10 SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Principles	Alongside medication, phase matched, family as the unit
3 Family work	Psychoeducation, expressed emotion, a defined session count
3 Individual work	Adherence, cognitive therapy, skills, cognitive remediation
2 Rehabilitation	Supported employment, day care, disability entitlements

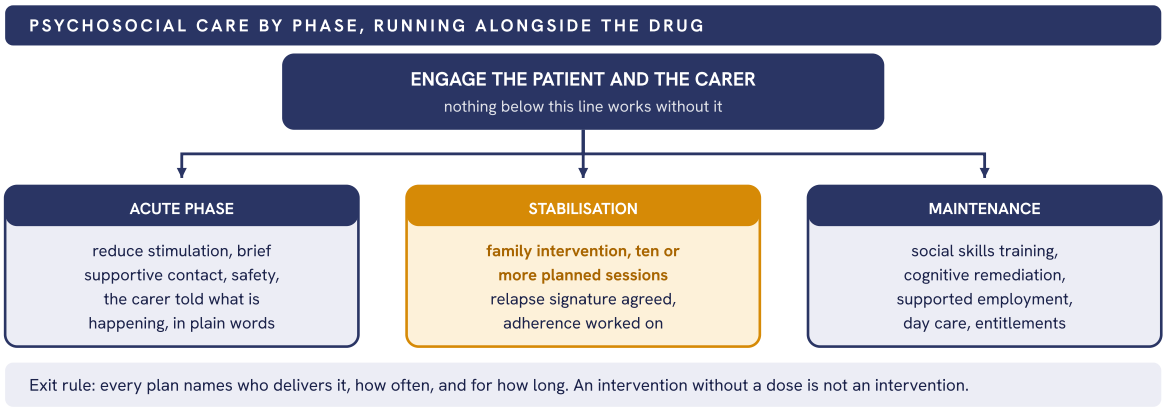

Fig 031.1 Psychosocial management by phase of illness, with the interventions that carry trial evidence placed where they are actually delivered.

- **Family intervention.** The strongest psychosocial effect on relapse: psychoeducation, communication and problem-solving training, and work on criticism and overinvolvement, over at least ten planned sessions.
- **Individual work.** Cognitive behavioural therapy for persisting positive symptoms, social skills training, cognitive remediation for attention and working memory, and adherence work in a motivational rather than a warning stance.
- **Work and entitlements.** Supported employment, placed first and trained on the job, outperforms prevocational training. Disability certification opens pension, concession and reservation.

PITFALL

Writing "counselling and rehabilitation" and stopping. An answer that cannot say how many sessions of what, delivered by whom, has described a sentiment rather than a plan.

WHAT EARNS THE MARKS

- **Phase-matched structure.** Acute, stabilisation and maintenance ask for different things.
- **Family intervention named with its session count.** It is the highest-yield line on the page.
- **Supported employment preferred to prevocational training,** with entitlements and carer burden included.

SEE ALSO Slot II - 33 chronic care in India Slot II - 35 expressed emotion

II - 32

Discuss the concept of childhood psychosis and its management. [10]

10 SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The concept	How it separated from autism, and the terms used now
3	What must be excluded	Organic causes, and the phenomena that only look psychotic
3	Drug treatment	Agent, low start, and the monitoring children need more of
1	Around the child	School, family and development kept moving

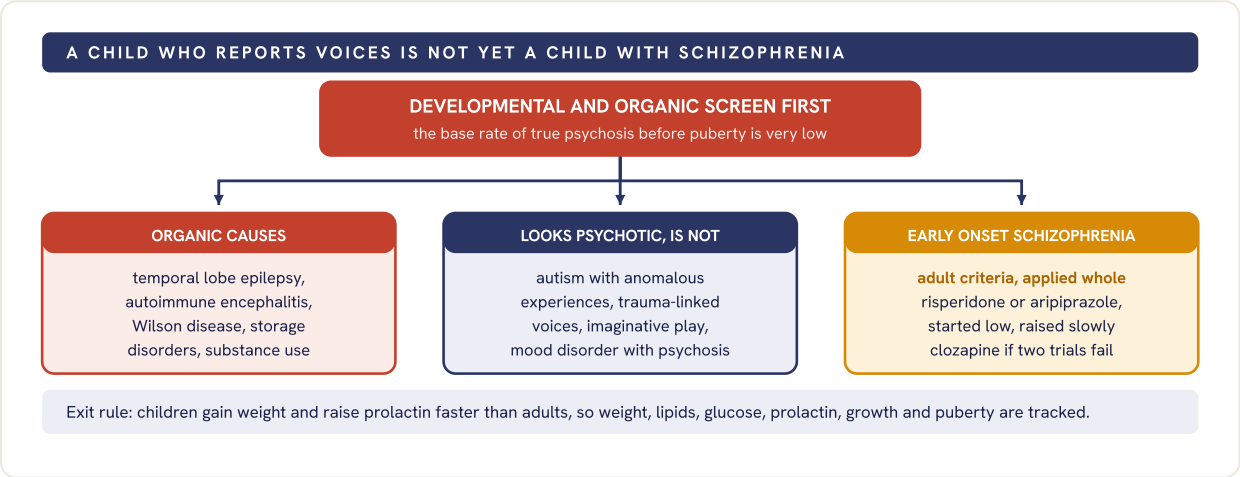

Fig 032.1 The order of work in a child with apparent psychosis: exclude the organic causes, separate the mimics, then treat as early onset schizophrenia.

- **The concept.** Until the 1970s every severe childhood disturbance was called childhood schizophrenia. Kolvin and Rutter separated it by age of onset, phenomenology, family history and course. The split has held.
- **The terms now.** Childhood onset means before thirteen and is rare, of the order of one in ten thousand. Early onset means before eighteen. Criteria are the adult criteria, unchanged.
- **What is different.** More premorbid language, motor and social delay, more insidious onset, more negative symptoms, a higher yield from genetic testing including the 22q11.2 deletion, poorer outcome.

PITFALL

Treating a reported voice as sufficient. Non-psychotic hallucinatory experiences are common in anxious, grieving and maltreated children and mostly remit.

WHAT EARNS THE MARKS

- **The historical separation from autism, with its basis.** The word "concept" in the stem asks for exactly this.
- **The organic screen written before any drug.** In this age group it separates a diagnosis from a mistake.
- **Monitoring described as heavier than in adults,** with the parameters named.

SEE ALSO Slot II - 30 early psychosis Slot II - 45 first episode pharmacology

II - 33

What are the options available in India for chronic care of longstanding schizophrenic patients? Discuss different government facilities a chronic schizophrenia patient can avail. How to assess family functioning in a case of schizophrenia? [2+4+4]

2 + 4 + 4 | SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What chronic care asks	Where the person lives, who supervises, what fills the day
4 Government routes	The programme, the Act, the certificate, the entitlements
2 Other options	Day care, halfway homes, long stay, carer-led groups
2 Family functioning	Four domains, and the instrument for each of them

WHAT CHRONIC CARE ACTUALLY DECIDES

Three questions, and every option below answers one of them: where the person lives, who supervises treatment, and what occupies the day. Cure is not on the list.

ROUTE	WHAT IT PROVIDES	HOW IT IS REACHED
District programme	Free outpatient care, medicines, outreach to the taluk	District hospital or outreach camp
Mental Healthcare Act 2017	Community living, less restrictive care, state duty to provide halfway homes	State authority, review board on appeal
Disability certificate	Benchmark disability at forty per cent on the notified scale	Medical board at the district hospital
What follows it	Pension, travel concession, employment reservation, carer tax relief	Certificate plus the disability identity card

Insurance for mental illness on the same terms as physical illness follows from section 21(4). Non-government day care, halfway homes and family associations sit alongside all of this.

- **Family functioning, four domains.** Burden, on the Family Burden Interview Schedule of Pai and Kapur, which separates objective from subjective. Expressed emotion, on the Camberwell Family Interview or the five minute speech sample. Knowledge and attitudes, on the Family Interview Schedule. General functioning, on the Family Assessment Device.
- **What it is for.** It answers whether this family can supervise medication and absorb a relapse. That decides home-based against residential care, and it is reassessed.
- **The line most answers miss.** The commonest crisis is the ageing or death of the parent carer. A named second carer, a nominated representative and a guardianship route belong in the plan.

WHAT EARNS THE MARKS

- **The three questions chronic care answers,** stated before any list of facilities.
- **Each government route with its access path.** Naming a scheme without saying how a patient reaches it is half a mark.
- **Named instruments for family functioning, including an Indian one,** with the domain each covers.

SEE ALSO Slot II - 31 psychosocial management Slot II - 35 expressed emotion

II - 34

What is endophenotype? Discuss schizophrenia endophenotype biomarkers. [3+7]

3 + 7 | SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 **Definition and criteria**	The five requirements, and that all five are needed
4 **The candidates**	Electrophysiology, eye movement, cognition, imaging
2 **Why they were wanted**	A simpler genetic target than the whole syndrome
1 **Where it stands**	One honest sentence on what the programme delivered

THE DEFINITION, WITH ITS FIVE CRITERIA

A measurable, heritable trait on the causal path between genotype and clinical syndrome. Gottesman and Gould require five things: association with the illness in the population, heritability, state independence, co-segregation within families, and presence in unaffected first degree relatives above the population rate.

CANDIDATE	WHAT IS MEASURED	STANDING
P50 gating	Suppression of the response to the second of two clicks	Deficient; linked to the alpha-7 nicotinic locus
Prepulse inhibition	Startle reduced by a weak preceding stimulus	Reduced in patients and relatives
Mismatch negativity	Automatic detection of an auditory change	Amplitude reduced; the most reproducible
Eye movement	Smooth pursuit tracking and antisaccade errors	Impaired in patients and many relatives
Neurocognition	Working memory, sustained attention, verbal memory	Impaired, heritable, state independent

Structural imaging findings, neurological soft signs, dermatoglyphic asymmetry and the blunted niacin skin flush are described alongside but meet fewer of the five criteria.

- **Why they were wanted.** The syndrome is heterogeneous and polygenic. A trait nearer gene action was expected to be genetically simpler, mappable in smaller samples.
- **What happened.** The candidates proved as polygenic as the syndrome, and none has shown larger genetic effect sizes. The promised shortcut did not appear.
- **Where it stands.** They survive as vulnerability markers, as a way of stratifying trial samples, and as objective endpoints. The ambition was absorbed into dimensional frameworks cutting across diagnoses.

WHAT EARNS THE MARKS

- **All five criteria, listed as criteria.** Three of five is the commonest way the first three marks are lost.
- **Candidates grouped by modality,** not offered as an undifferentiated list.
- **The relative finding stated,** since presence in unaffected relatives is what makes a trait an endophenotype at all.

SEE ALSO Slot II - 80 functional imaging Slot II - 81 electroencephalography

II - 35

Expressed emotion in psychiatry: critically evaluate. [10]

10 SCHIZOPHRENIA: AETIOLOGY, PHENOMENOLOGY AND COURSE

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it is	Five components, rated from the relative, with thresholds
3	The case for it	Relapse prediction, holding across several diagnoses
3	The case against	Culture, direction of causation, cost of measuring
2	The verdict	Modifiable, so worth measuring; never a family blame

THE CONSTRUCT, STATED PRECISELY

The emotional climate of the household toward the patient, rated from a semi-structured interview with the relative. A property of a relationship at a point in time, not a personality of the family.

COMPONENT	WHAT IS RATED	THRESHOLD FOR HIGH
Critical comments	Count of critical remarks about the patient	Six or more
Hostility	Generalised criticism or rejection of the person	Any
Overinvolvement	Overprotection, self-sacrifice, dramatic distress	Rating of three or more
Warmth	Sympathy, concern, interest in the person	Not part of the definition
Positive remarks	Praise and appreciation	Not part of the definition

Rated by the Camberwell Family Interview, which runs over an hour and needs training. The five minute speech sample and self-report questionnaires are shorter proxies with lower sensitivity.

- **The case for it.** High expressed emotion roughly doubles nine to twelve month relapse risk in schizophrenia, and the association holds in mood disorders, eating disorders and substance use.
- **The cultural objection.** Indian samples show lower rates, and overinvolvement means something different where multigenerational co-residence and close supervision are normative.
- **The causal objection.** Criticism rises with the patient's disturbance, so part of the association runs from illness to relative. The relationship is bidirectional.
- **The verdict.** Worth measuring because it is modifiable: family intervention that lowers criticism lowers relapse. That is also why it is never presented to a family as a fault.

WHAT EARNS THE MARKS

- **All five components, with the three that define high expressed emotion marked.** Warmth and positive remarks are rated but excluded.
- **Both objections argued rather than named.** The stem says critically evaluate.
- **A verdict at the end.** A critical evaluation that never lands on a position is an essay, not an answer.

SEE ALSO Slot II - 28 course and outcome Slot II - 31 psychosocial management

AREA 05 OF 14 · P2-A09

Trauma, dissociative and somatic symptom disorders

8	8.4%	II-36	II-43
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 3 · HIGH 1 · SINGLE 4

REVISION ORDER

The two dissociative slots together: one asks management and one asks phenomenology, and they are one answer split. PTSD and pathological grief next. The somatoform and abnormal illness behaviour pair after that. The rape slot and the depression scales slot are independent.

WHAT IS IN THIS AREA

Dissociative and conversion disorder in two slots, post-traumatic stress disorder, pathological grief, the somatoform slot with chronic pain, abnormal illness behaviour, and the rape slot with marital rape and survivor coping. One slot here asks for the rating scales of depression with one described in full. That is an assessment question the boards set inside a trauma area, and it is answered on its page as it was set.

II - 36

Management of dissociative disorders. [10]

10 TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

KNRUHS, RGUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

3 **Diagnose positively**	Positive signs, not a diagnosis of exclusion
4 **The core treatment**	Explanation, rehabilitation, then the psychological work
3 **Traps and prognosis**	What harms, and what predicts recovery

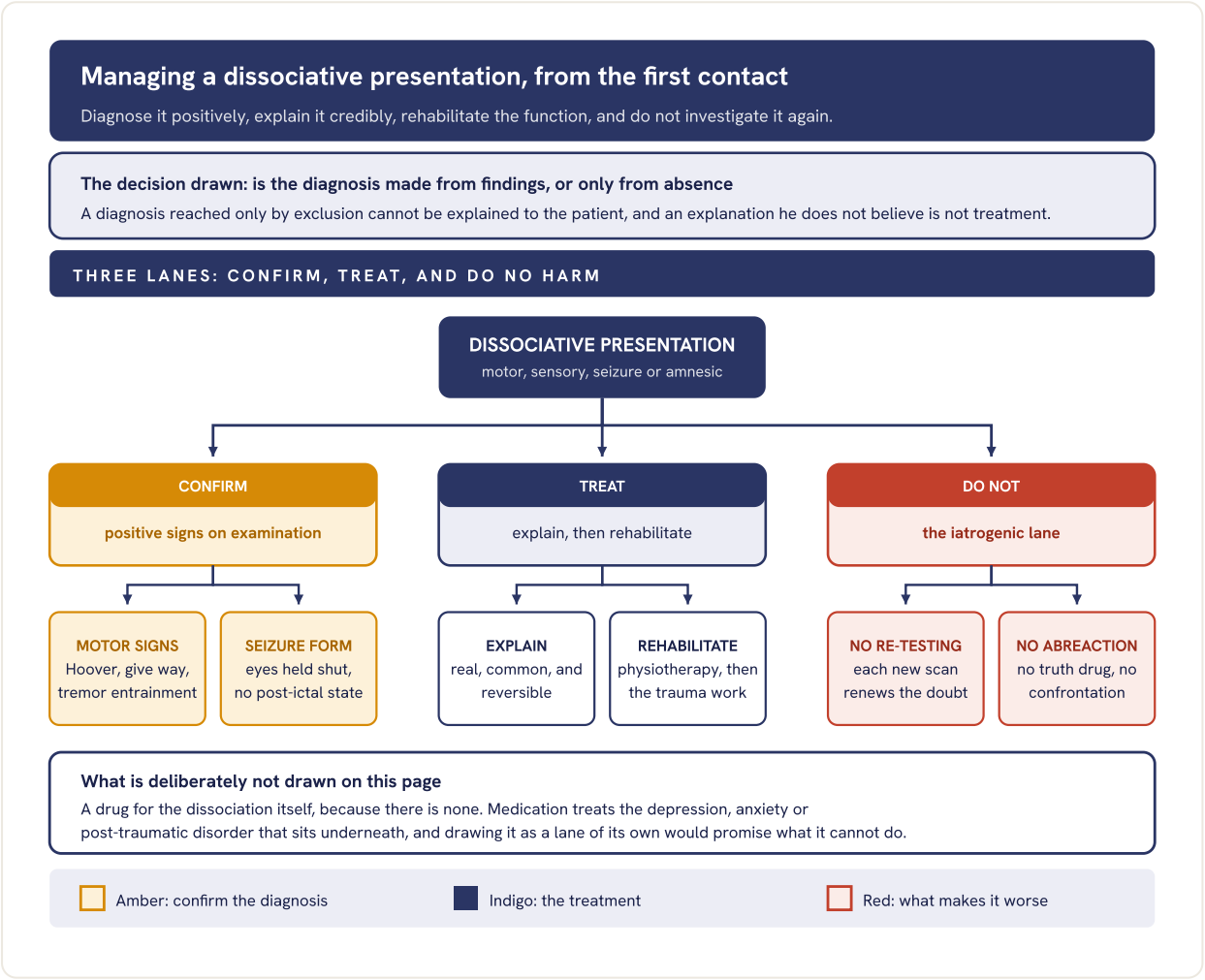

Fig 036.1 Confirmation drawn as a lane of its own, because the explanation given to the patient can only be as convincing as the findings it rests on.

- **How the explanation is given.** Name the diagnosis, show him the positive sign on his own body, say the symptom is genuine and the pathway is reversible, and give the reason the nervous system can produce it. Then hand over to rehabilitation with the same message.

PROGNOSIS, HONESTLY STATED

Short duration, acute onset, a clear precipitant, early diagnosis and the absence of ongoing gain all predict recovery, which in acute presentations is often complete. Long duration before diagnosis, chronic pain, unresolved litigation and prominent comorbidity predict a poorer outcome.

WHAT EARNS THE MARKS

- **A positive sign named,** the first amber leaf, rather than the phrase diagnosis of exclusion.
- **The explanation written as a treatment step,** with the words that are used.
- **Physiotherapy before insight work** in the motor presentations.
- **The red lane,** repeated investigation and abreaction named as harms, not as options.

SEE ALSO Slot II - 43 features and signs of conversion disorder Slot II - 41 somatoform disorder and chronic pain

SPRINT Day 15, Dissociative, conversion and anxiolytic pharmacology

II - 37

Mention the rating scales of depression and describe one of them. [10]

10 TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 The axis	Who rates it, and what the scale is for
4 The scales	Clinician rated, self rated, special populations
3 One described	Structure, scoring, bands, and its use
1 Its limitations	The criticism an examiner is listening for

STATE THE AXIS BEFORE THE LIST

Sort by who rates it and why it is being used. Severity scales measure change and do not make a diagnosis; screening scales find cases and do not grade them. A candidate who states this before listing anything has already earned the first two marks.

GROUP	SCALES	FORMAT	CHIEFLY USED FOR
Clinician rated	Hamilton depression rating scale; Montgomery and Asberg scale	Semi-structured interview	Severity and change in trials
Self rated	Beck depression inventory; Zung self rating scale	Questionnaire	Severity as the patient sees it
Screening	Patient health questionnaire, nine item	Nine items, criterion mapped	Case finding in general settings
Perinatal	Edinburgh postnatal depression scale	Ten items, no somatic items	Screening after childbirth
Elderly	Geriatric depression scale; Cornell scale in dementia	Yes or no; informant based	Avoiding somatic confounding
Medically ill	Hospital anxiety and depression scale	Fourteen items, two subscales	Use where illness mimics somatic items

The amber cells mark the two purposes that are most often confused: measuring change, and finding a case.

- **The Hamilton scale, described.** A clinician administered severity scale from 1960, most often used in its seventeen item form. Items are scored zero to four or zero to two and summed, with conventional bands running from normal through mild, moderate and severe. It takes about twenty minutes and remains the commonest primary outcome measure in antidepressant trials.
- **Its limitations.** It is weighted towards somatic and sleep items, so it inflates in the physically ill and can improve on sedation alone. It is multidimensional rather than a single construct, and it measures severity, never diagnosis.

WHAT EARNS THE MARKS

- **The axis stated first,** clinician against self rated, severity against screening.
- **Special population scales included,** perinatal, elderly and medically ill, which most answers omit.
- **One scale described with its structure and scoring,** as the question asks, not merely named again.
- **The somatic weighting criticism,** which is the line that separates a top answer.

SEE ALSO Slot II - 39 pathological grief Slot II - 38 post-traumatic stress disorder **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 38

Write clinical features, pharmacological management and prognosis of post-traumatic stress disorder. [4+3+3]

4 + 3 + 3 | TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

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

SKELETON

4 Clinical features	The four clusters, the time rule, the subtypes
3 Pharmacological	First line, the nightmare drug, and what is avoided
3 Prognosis	Recovery rates, and what predicts the chronic course

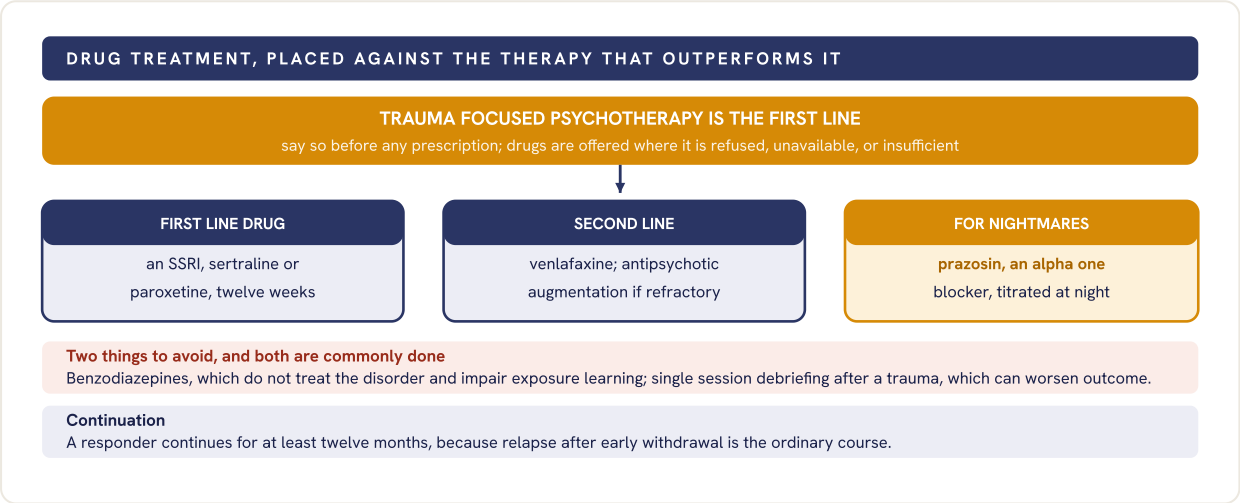

Fig 038.1 The drug lanes drawn beneath the therapy that outranks them, with the two avoidable harms carried as a red strip.

- **Clinical features.** After exposure to death, serious injury or sexual violence: intrusion as memories, nightmares and flashbacks; avoidance of reminders inside and outside; negative alterations in thinking and mood, including amnesia for part of the event and distorted self blame; and hyperarousal with startle, irritability and poor sleep. Longer than one month.
- **The variants to name.** A dissociative subtype with depersonalisation or derealisation, and delayed expression where the picture completes only months later. The newer international classification adds complex post-traumatic disorder, with affect dysregulation, a negative self concept and relationship difficulty.
- **Prognosis.** About half recover within a few months and roughly a third run a chronic course. Interpersonal and intentional trauma, childhood adversity, peritraumatic dissociation, poor social support, ongoing threat and comorbid depression or substance use all predict chronicity.

WHAT EARNS THE MARKS

- **Four symptom clusters named as clusters,** with the one month rule.
- **Psychotherapy placed above drugs,** the amber band, even though only drugs were asked for.
- **Prazosin for nightmares,** the specific answer most candidates never give.
- **Prognosis given with predictors,** not the single word variable.

SEE ALSO Slot II - 39 pathological grief Slot II - 40 coping among survivors of sexual assault

II - 39

Describe the clinical features, risk factors and management of pathological grief. [10]

10 TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

DNB, NTRUHS, RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 **Where the line falls**	Grief against disorder, by time, intensity and culture
3 **Clinical features**	Yearning, identity disruption, avoidance, impairment
2 **Risk factors**	The death, the relationship, and the person
3 **Management**	The grief specific therapy, and what drugs do and do not do

WHERE THE LINE FALLS

Not the presence of grief but its persistence, its intensity and its cost. Both current classifications require intense longing or preoccupation with the person who died, lasting well beyond the expected period for the culture, with impairment. Six months is the earlier threshold, twelve months the later and more conservative one.

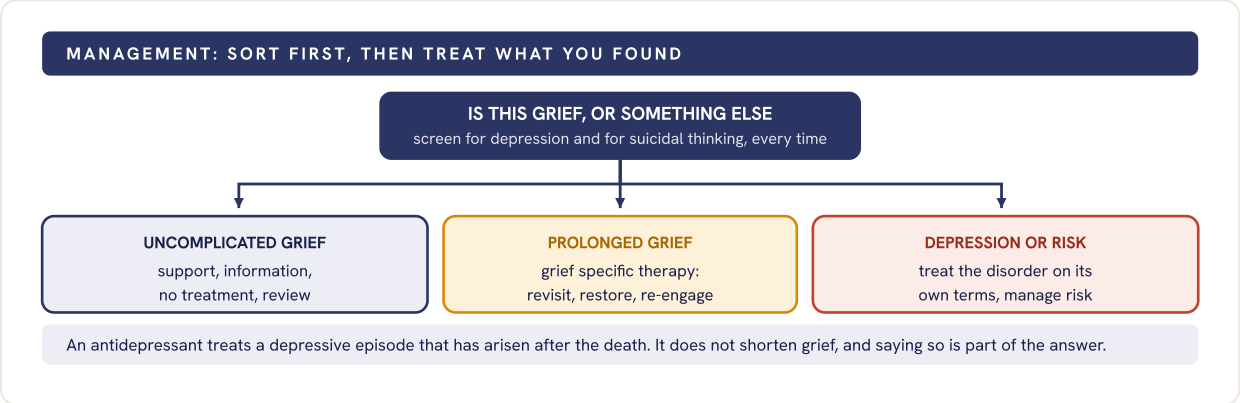

Fig 039.1 One sorting question with three destinations, drawn because the commonest error is treating all three the same way.

- **Clinical features and risk factors.** Intense yearning, preoccupation, disbelief, emotional pain, identity disruption, numbness, avoidance of reminders and a sense that life is meaningless. Risk rises with sudden, violent or self inflicted death, the loss of a spouse or a child, dependent relationships, prior depression, poor support and unresolved conflict with the person who died.
- **Culture is part of the criterion.** Both systems set the threshold against what is expected in the person's own culture and religion. Structured mourning practice with a fixed period is not pathology, and reading it as pathology is a real diagnostic error in Indian practice.

WHAT EARNS THE MARKS

- **The time threshold given as a number,** with both classifications acknowledged.
- **Identity disruption named,** which is the feature separating this from a depressive episode.
- **Grief specific therapy named as the treatment,** the amber box, rather than counselling in general.
- **Culture written into the threshold,** not added as a closing remark.

SEE ALSO Slot II - 38 post-traumatic stress disorder Slot II - 37 rating scales of depression

II - 40

Define rape. What is marital rape? Write in brief about coping skills and quality of life among rape survivors. [2+5+3]

2 + 5 + 3 | TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	The statutory definition, and the seven circumstances
2	Marital rape	The exception, its limits, and the remedies that remain
3	Coping	The phases, and which coping styles predict outcome
3	Quality of life	What is impaired, and what the clinician can change

THE DEFINITION, AND THE EXCEPTION

Indian criminal law defines rape as specified sexual acts by a man with a woman against her will or without valid consent, including consent obtained by fear or fraud, consent she cannot understand, and any act with a girl under eighteen. It was widened in 2013 beyond penetrative intercourse and carried into the criminal code that replaced the older statute in 2024. An exception excludes a husband where the wife is not below eighteen, so marital rape is a distinct offence only where she is a minor. The exception has been challenged on constitutional grounds; check its current status.

PHASE AFTER ASSAULT	WHAT IS SEEN	WHAT THE CLINICIAN DOES
Acute, days to weeks	Shock, disorganisation, numbness or agitation	**Safety, medical care, a believing response**
Outward adjustment	Apparent return to routine, suppression, avoidance	Keep contact open; do not read calm as recovery
Reorganisation	Intrusions, fear, sexual and relational difficulty	**Trauma focused therapy when it is wanted**
If it does not resolve	Post-traumatic disorder, depression, self harm, substance use	Treat the disorder that has formed

A common sequence, not a schedule: any phase may be absent, and returning to an earlier one is not deterioration.

- **Coping, and what predicts outcome.** Avoidance coping, self blame directed at her character rather than at a choice, and social withdrawal all predict worse outcome. Disclosure met with belief, practical support, problem focused coping and re-established routine predict better outcome. A disbelieving or blaming response from family, police or hospital is itself a risk factor.
- **Quality of life, and the clinician's part.** Sleep, sexual function, work, study, mobility and trust are all affected, often for years. What a clinician can change: prompt medical care with emergency contraception and infection prophylaxis, an examination conducted with consent and dignity, no discredited virginity testing, careful documentation for the record, and referral to a support service.

WHAT EARNS THE MARKS

- **The statutory definition with the consent circumstances,** and the age rule.
- **The exception stated accurately,** including that it has been challenged, without asserting an outcome.
- **Coping described by style rather than by stage alone,** since style is what predicts outcome.
- **Secondary victimisation named,** which is the part of the answer a clinician can actually act on.

SEE ALSO Slot II - 38 post-traumatic stress disorder Slot II - 39 pathological grief

II - 41

Discuss the concept of somatoform disorder. Describe the management of a case of a middle aged woman presenting with chronic pain syndrome. [6+4]

6 + 4 | TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The concept	What the older category grouped, and on what basis
3 The shift	From unexplained symptoms to a positive criterion
4 The case	Engage, one doctor, function as the goal

THE CONCEPT, AND HOW IT CHANGED

The older grouping collected physical symptoms suggesting disease that were not fully explained by it, not intentionally produced, and causing distress: somatisation, hypochondriacal, autonomic and persistent pain disorders. The current systems drop the unexplained criterion and define the disorder positively, by disproportionate thoughts, high health anxiety and excessive time given to symptoms, whether or not a physical disease is also present.

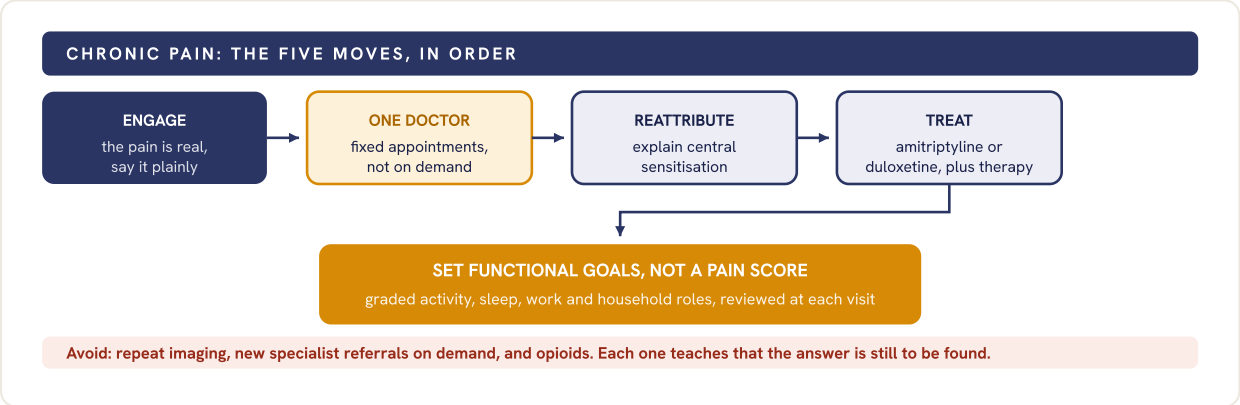

Fig 041.1 Five moves in sequence, ending in the goal that replaces cure, with the three iatrogenic traps carried along the foot.

- **The two errors to avoid in her case.** Investigating again to reassure her, which reassures nobody for long; and telling her the pain is psychological, which she hears as an accusation and which is also not what the current criterion says.

WHAT EARNS THE MARKS

- **The change in the criterion stated,** from medically unexplained to a positive psychological feature.
- **A single coordinating doctor with scheduled visits,** the amber box, which is the intervention with the best evidence here.
- **Function named as the goal instead of pain relief.**
- **The three iatrogenic traps,** repeat imaging, referral on demand, and opioids.

SEE ALSO Slot II - 42 abnormal illness behaviour Slot II - 36 management of dissociative disorders

II - 42

Abnormal illness behaviour. [10]

10 TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	The definition, and the two ideas it was built on
2	The axes	Affirming or denying, conscious or not
4	The four cells	Each with the conditions that sit in it
2	Why it is useful	What it changes in assessment and management

THE DEFINITION

The persistence of a maladaptive way of experiencing, perceiving, evaluating and responding to one's own health, despite an adequate and accurate explanation having been given. It extends the older ideas of the sick role, with its rights and its obligations, and of illness behaviour, which is how anyone responds to bodily change.

AXIS	NOT CONSCIOUSLY MOTIVATED	CONSCIOUSLY MOTIVATED
Illness affirming	Somatic symptom and illness anxiety disorders, conversion presentations	Malingering for an external gain; factitious disorder for the sick role itself
Illness denying	Unawareness of deficit after brain injury; apparent unconcern about a real disability	Deliberate concealment, to keep a job, a licence or a marriage
What decides the cell	Absence of any intended gain on careful history	An identifiable external gain, or evidence of deliberate production
What it changes	Treat, engage, reattribute	Do not confront alone; document, protect, and manage the risk

The amber row is the one that carries the marks, because separating malingering from a somatic symptom disorder is the judgement the concept was created to discipline.

- **Why the concept is useful.** It describes behaviour rather than assigning blame, it holds denial and affirmation in one frame, and it forces the question of motivation to be asked explicitly instead of assumed. A structured questionnaire exists for research use.
- **The honest limit.** Conscious motivation cannot be measured, only inferred, and inference here carries real cost to the patient. Malingering is not a psychiatric diagnosis, and it should be recorded only on evidence that would stand outside the consulting room.

WHAT EARNS THE MARKS

- **The definition quoted, with despite an adequate explanation** in it, which is the operative clause.
- **The sick role named as its ancestor,** with the obligations as well as the rights.
- **Both axes drawn, giving four cells,** not a list of somatoform conditions.
- **The limit on inferring motivation,** which is the ethically serious part of the answer.

SEE ALSO Slot II - 41 somatoform disorder and chronic pain Slot II - 43 features and signs of conversion disorder

II - 43

What are the main clinical features of dissociative (conversion) disorders? Write distinctive physical examination findings in conversion disorder. [3+7]

3 + 7 | TRAUMA, DISSOCIATIVE AND SOMATIC SYMPTOM DISORDERS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Clinical features	Loss of integration, the time relation, the varieties
5 The positive signs	By system, each with how it is elicited
2 How signs are used	What they prove, and what they do not

THE CLINICAL FEATURES

A partial or complete loss of the normal integration between memory, awareness of identity, sensation and control of movement. Onset is usually close in time to a stressful event or an insoluble difficulty, symptoms follow the patient's own idea of illness rather than anatomy, and no physical disorder accounts for them.

SIGN	HOW IT IS ELICITED	WHAT IT DEMONSTRATES
Hoover sign	Feel the weak leg's heel while he flexes the good hip against resistance	**Hip extension returns when attention is elsewhere**
Give way weakness	Test power steadily against resistance	Sudden ratchety collapse rather than a fixed weakness
Tremor entrainment	Ask him to tap a rhythm with the other hand	**The tremor takes up that rhythm, or stops**
Tubular visual field	Confrontation at two distances	The field does not widen with distance as it must
Seizure form	Observe the event, and the minutes after it	Eyes held shut, long duration, no post-ictal state
Preserved substrate	Reflexes, tone, plantars, and wasting	All normal in a limb said to be paralysed for weeks

Each sign shows the same thing: the function is present when it is not being attended to. That single sentence is worth writing before the list.

- **What the signs do not prove.** They support a positive diagnosis, they do not exclude coexisting disease, and none of them says anything about motivation. La belle indifference is unreliable and should not be offered as a sign.
- **How to use them.** Demonstrate one to the patient. A sign he can see for himself makes the explanation credible, which is the first step of treatment rather than a separate exercise in diagnosis.

WHAT EARNS THE MARKS

- **Loss of integration named as the core feature,** with the temporal relation to stress.
- **The common principle stated before the list:** function returns when attention moves.
- **At least four named signs with how each is elicited,** as the middle column does.
- **The limit,** that a positive sign does not exclude disease and says nothing about intent.

SEE ALSO Slot II - 36 management of dissociative disorders Slot II - 42 abnormal illness behaviour **SPRINT** Day 15, Dissociative, conversion and anxiolytic pharmacology

AREA 06 OF 14 · P2-A02

Schizophrenia: treatment and antipsychotics

8	8.3%	II-44	II-51
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 · HIGH 1 · RECURRENT 1 · SINGLE 5

REVISION ORDER

Treatment resistance first, it is the anchor. Then the side-effect group as one block: metabolic, tardive dyskinesia and neuroleptic malignant syndrome share most of their material. The haloperidol slot next. Cognitive remediation and the stigma slot last, and neither is long.

WHAT IS IN THIS AREA

Treatment-resistant schizophrenia, first-episode psychosis in an adolescent, the first-generation antipsychotics with haloperidol in detail, the metabolic side effects and their prevention, tardive dyskinesia, neuroleptic malignant syndrome, cognitive remediation in rehabilitation, and a stigma and discrimination slot that asks for a local policy.

II - 44

Treatment resistant schizophrenia. [10]

10 SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB, RGUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

3	The definition	Four conditions, all of them, before the label is used
2	Pseudo-resistance	What is excluded first, and how each is checked
4	Clozapine	Titration, target, plasma level, and the monitoring
1	If clozapine fails	Augmentation, with the one option that has evidence

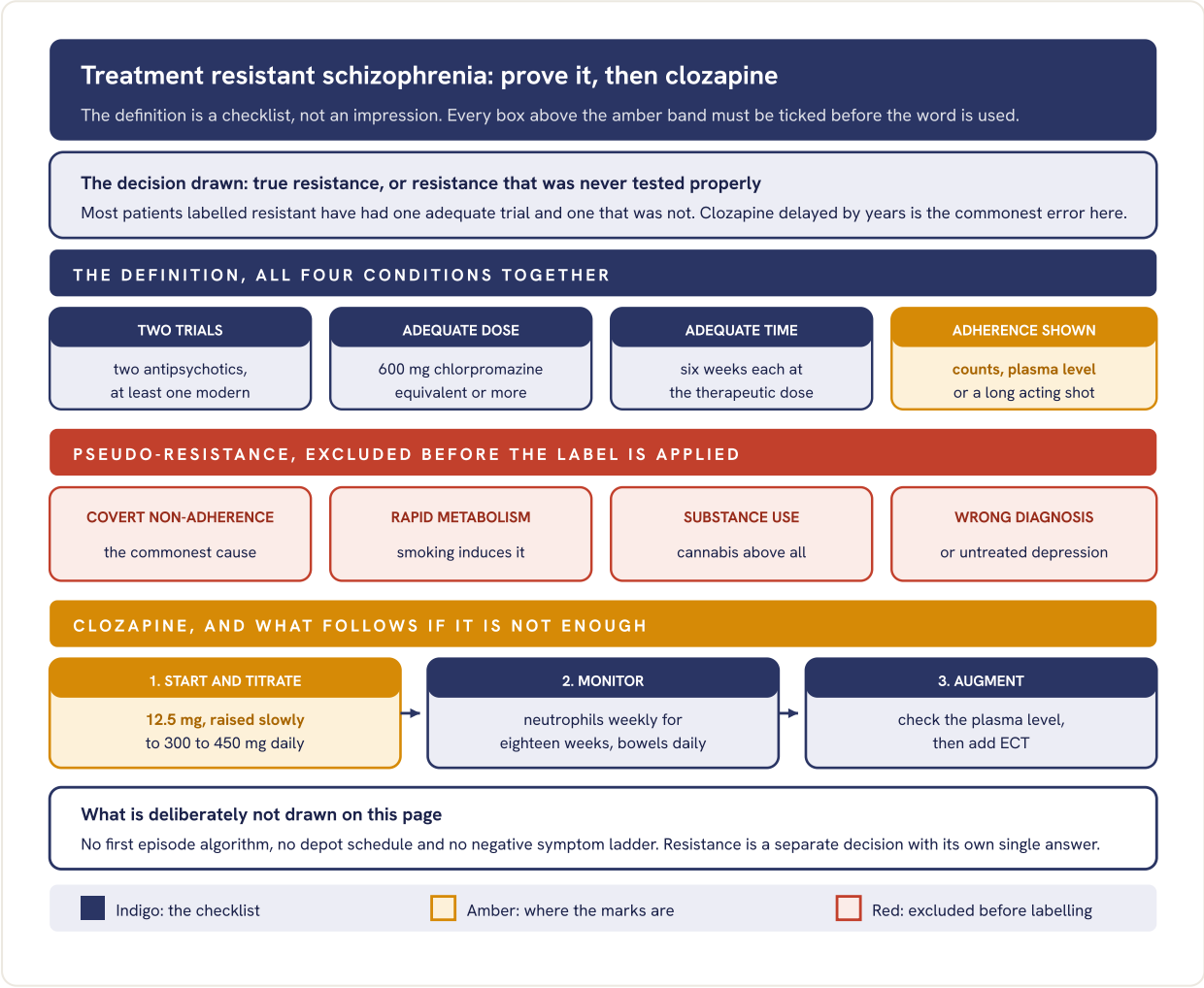

Fig 044.1 The four conditions that define resistance, the four causes of apparent resistance that are excluded first, and the clozapine sequence that follows once the label is earned.

- **Clozapine.** The only agent with established efficacy here, and roughly a third to a half respond. Non-response at a normal dose is checked against a plasma level, with about 350 nanograms per millilitre as the threshold worth reaching before it is called a failure.
- **Its dangers, in order of what actually kills.** Constipation progressing to ileus, then myocarditis in the first weeks, then agranulocytosis in about one per cent. Seizures rise with dose. Sedation, sialorrhoea and weight gain drive most discontinuation.

PITFALL

Adding a second antipsychotic instead of starting clozapine. Combination has weak evidence and certain added harm, and every month spent on it is a month of untreated illness in the period when function is still recoverable.

WHAT EARNS THE MARKS

- **All four parts of the definition, including documented adherence.** Most answers give two trials and stop.
- **Pseudo-resistance excluded before clozapine is named.**
- **Clozapine written with its titration, target range and monitoring schedule,** not just as a word.
- **Electroconvulsive therapy named as the augmentation with the best evidence.**

SEE ALSO Slot II - 29 negative symptoms Slot II - 50 metabolic effects Slot II - 52 treatment resistant depression

II - 45

Describe the pharmacological management of first episode psychosis in an adolescent. [10]

10 SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Before the first dose**	Organic and substance screen, baselines, consent and assent
4	**The first choice**	Which agent, what starting dose, what to avoid
2	**If it does not work**	Switch once, then clozapine after two adequate trials
2	**Duration and monitoring**	How long to continue, and what is tracked meanwhile

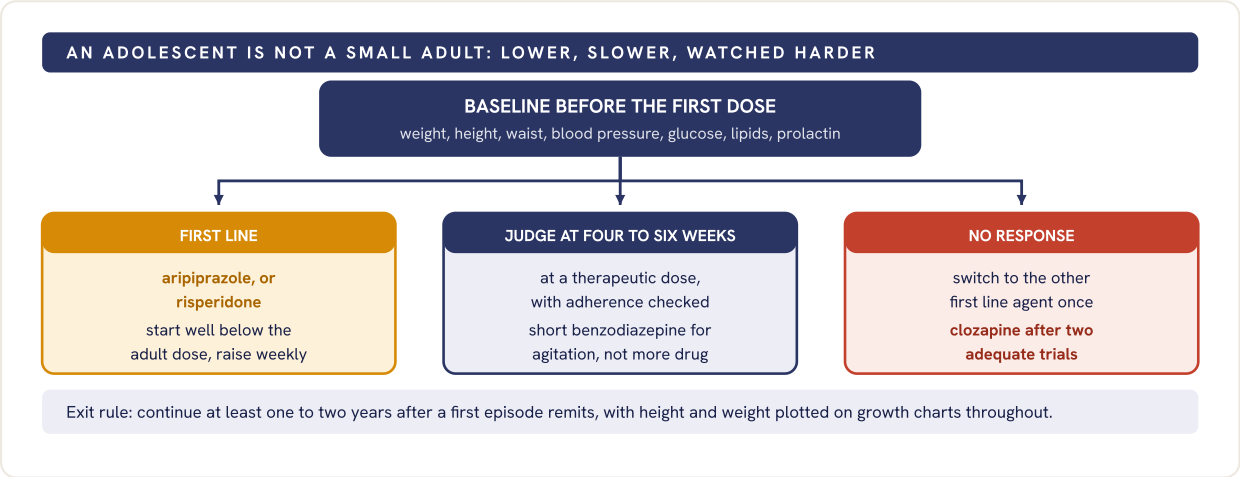

Fig 045.1 First episode pharmacology in an adolescent: the baseline that precedes the first dose, the two first line agents, and the point at which clozapine becomes the answer.

COLOUR CODE assessment and review · the drug choice · failure pathway

- **Before prescribing.** Confirm the diagnosis, exclude substance-induced and organic psychosis, record the metabolic baseline. A minor cannot consent alone: the guardian is the nominated representative, and assent is still sought.
- **Why these two agents.** Adolescents gain weight and raise prolactin more readily than adults. Aripiprazole is metabolically the least costly, risperidone raises prolactin most, olanzapine is avoided first line.
- **Pharmacology is half the plan.** Family psychoeducation and school liaison run in parallel from week one, and the answer should say so.

PITFALL

Raising the dose at two weeks for apparent non-response. Response in a drug-naive first episode builds over four to six weeks; early escalation buys side effects and a false diagnosis of resistance.

WHAT EARNS THE MARKS

- **The metabolic baseline written before the first dose.**
- **A named first line agent with a reason,** and olanzapine explicitly declined at this age.
- **An adequate trial defined, then the switch, then clozapine.** The sequence is the answer.

SEE ALSO Slot II - 30 early psychosis Slot II - 50 metabolic effects

II - 46

What is neuroleptic malignant syndrome? How would you confirm the diagnosis? Write the management of neuroleptic malignant syndrome. [2+4+4]

2 + 4 + 4 | SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **What it is**	Idiosyncratic reaction to dopamine blockade, and the tetrad
4 **Confirming it**	Criteria, the laboratory pattern, and what must be excluded
3 **Management**	Stop, support, then the specific agent by severity
1 **Rechallenge**	The rule that prevents a second episode

- **What it is.** An idiosyncratic, life threatening reaction to dopamine blockade. The tetrad is hyperthermia, lead pipe rigidity, altered sensorium and autonomic instability, usually within two weeks of a dose change.
- **Confirming it.** No test confirms it, so the diagnosis is criterion based: fever, rigidity and a creatine kinase commonly above a thousand units per litre, with leucocytosis, low serum iron and myoglobinuria. Imaging and cerebrospinal fluid exclude.
- **What must be excluded.** Serotonin syndrome, with clonus and hyperreflexia within hours. Malignant catatonia, where catatonic signs precede the drug. Malignant hyperthermia, anticholinergic toxicity, infection and heat stroke.

Fig 046.1 One irreversible action, then three layers: always, by severity, and if it does not turn.

PITFALL

Treating the fever as infection and continuing the antipsychotic. Rigidity with a rising creatine kinase after a dose change is this syndrome until proved otherwise.

WHAT EARNS THE MARKS

- **The tetrad named as a tetrad, with its time relation to the drug.**
- **Criterion-based diagnosis stated,** with the honest line that no test confirms it.
- **Stopping the drug written as the first line of management.**
- **The rechallenge rule.** Most answers stop at recovery and lose the last mark.

SEE ALSO Slot II - 51 tardive dyskinesia Slot II - 91 catatonia

II - 47

Classify first generation antipsychotics. Describe the mechanism of action, metabolism and side effect profile of haloperidol. [5+5]

5 + 5 | SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The axis	Chemical class first, then the potency axis that matters clinically
3	The classes	Four groups, each with its representative drugs
3	Haloperidol pharmacology	Receptor profile, occupancy window, handling by the liver
2	Its adverse effects	Grouped by the pathway that produces each of them

NAME THE AXIS BEFORE THE LIST

The classification is by chemical structure. The axis that predicts what a patient will feel is potency, and the two do not line up.

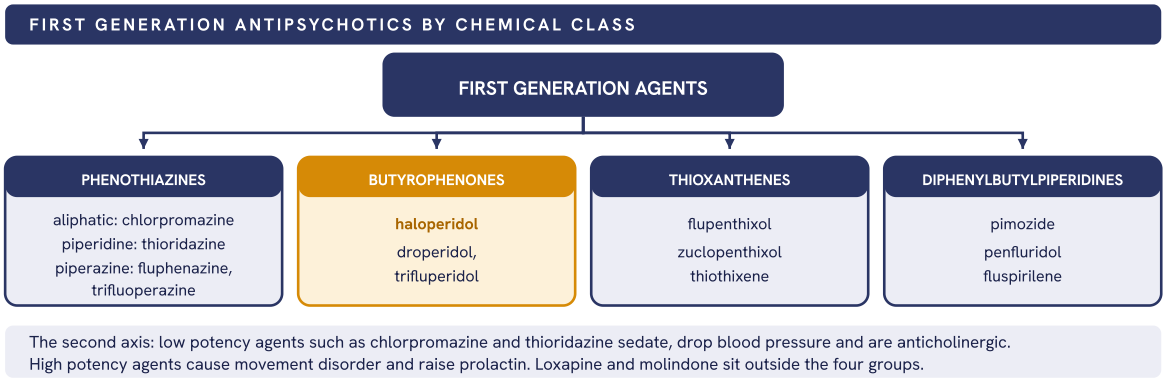

Fig 047.1 One axis, four branches, with the potency axis carried as a strip along the foot.

- **Mechanism.** A high potency D2 antagonist with little muscarinic, histamine or alpha-1 affinity. Antipsychotic effect appears near sixty five per cent striatal D2 occupancy; above eighty per cent, movement disorder is near certain.
- **Metabolism.** Well absorbed, heavily protein bound, lipophilic, with first pass loss that makes the oral dose exceed the parenteral. Cleared through CYP3A4, CYP2D6 and glucuronidation, half life roughly a day. The decanoate is given about every four weeks.
- **Adverse effects, by pathway.** Nigrostriatal blockade gives dystonia, akathisia, parkinsonism and later tardive dyskinesia; tuberoinfundibular blockade raises prolactin. Beyond them: the malignant syndrome, a lowered seizure threshold, and QT prolongation.

WHAT EARNS THE MARKS

- **The chemical axis stated, then the potency axis named as the clinically useful one.**
- **Four classes with named representatives,** not a flat list of drug names.
- **The occupancy window, with both numbers.** It explains the whole side effect profile in one line.
- **Adverse effects grouped by pathway,** which is what separates a top answer from a recalled list.

SEE ALSO Slot II - 46 neuroleptic malignant syndrome Slot II - 51 tardive dyskinesia

II - 48

What is cognitive remediation? Discuss its role in rehabilitation in schizophrenia. [2+8]

2 + 8 | SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	The consensus definition and its four active ingredients
3 What is trained	Neurocognition and social cognition, domain by domain
3 The evidence	Effect on cognition, smaller on function, and the condition
2 Place in rehabilitation	An adjunct, and what it must be paired with to pay

THE DEFINITION, AND ITS FOUR INGREDIENTS

Behavioural training that improves cognitive processes, aiming at durability and generalisation to daily function. Four ingredients define it: a trained therapist, repeated practice, explicit strategy development, and transfer procedures.

DOMAIN TRAINED	HOW IT IS TRAINED	WHAT IT SHOULD LOOK LIKE IN DAILY LIFE
Processing speed	Timed symbol and coding drills	Keeping pace with a conversation or task
Attention	Sustained detection tasks	Sitting through a shift or a class
Working memory	Span and running memory tasks	Holding an instruction long enough to act
Verbal learning	Structured list and story recall	Remembering a medication schedule
Social cognition	Emotion recognition, perspective taking	Reading intent, so fewer misfires

The third column is the point. Training that never leaves the third column has failed its own definition.

- **The evidence.** Meta-analyses show a small to moderate gain in global cognition and a smaller one in functioning, durable at follow-up. The functional gain is substantially larger when remediation runs alongside psychiatric rehabilitation than when it runs alone.
- **Why cognition is the target.** Cognitive impairment predicts work, independent living and social function better than positive symptoms do, and it persists through symptomatic remission. Antipsychotics do not treat it.
- **The honest limit.** It is an adjunct, not a stand-alone treatment. Paired with supported employment it improves job tenure; delivered as computer exercises with nothing attached, it improves test scores and little else.

WHAT EARNS THE MARKS

- **The four ingredients named,** since the two definition marks sit there.
- **Social cognition included as a target,** not only neurocognition.
- **The condition on the evidence:** functional benefit depends on pairing it with rehabilitation.
- **Cognition stated as a better predictor of function than symptoms.**

SEE ALSO Slot II - 29 negative symptoms Slot II - 31 psychosocial management **SPRINT**

Day 9, Schizophrenia: management, antipsychotics and adverse effects

II - 49

What do you understand by stigma and discrimination? Describe different types of discrimination of recent time in India. How can a policy be framed to improve stigma associated with mental illness in your locality? [3+4+3]

3 + 4 + 3 | SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The two constructs	Stigma as a process, discrimination as the behaviour it produces
4 Types in India now	Work, insurance, civil law, health care, the family
2 Framing a local policy	Target a behaviour, use contact, use platforms that exist
1 Measuring it	An attitude score and a service outcome, before and after

THE TWO WORDS, SEPARATED

Stigma is a process with five parts, needing a power difference to operate: labelling, stereotyping, separation into us and them, status loss, and discrimination. Discrimination is the last part made visible. Stigma is public, internalised, structural, or attached to the family.

WHERE	HOW IT APPEARS NOW	WHAT THE LAW SAYS
Employment	Not hired after disclosure, moved out after an episode	Prohibited; reservation in the public sector
Insurance	Cover refused or priced apart from physical illness	Parity required under section 21(4)
Civil law	Capacity assumed absent; illness cited in matrimonial cases	Capacity presumed, assessed decision by decision
Health care	Diagnostic overshadowing, poorer physical care	Chaining prohibited; least restrictive setting
Family	Concealment, restricted marriage prospects	Not reachable by law; contact works here

The last row matters most in Indian practice and is the only one a district level policy can move directly.

- **Framing the policy.** Name one audience and one behaviour, not awareness in general. Contact based intervention, direct or filmed, with a person who has lived experience, outperforms education alone.
- **Delivery.** Use platforms that already exist, the district programme, schools, primary care and local government, and add a complaint route so the protections above are enforceable.
- **Measurement.** One attitude scale before and after, plus one service outcome such as help-seeking delay. Single awareness days shift knowledge and leave behaviour untouched.

WHAT EARNS THE MARKS

- **Stigma given as a process with named parts,** and discrimination as its behavioural end.
- **Indian examples with the statutory position beside each.** The stem asks for recent India, not a general account.
- **Contact named as the active ingredient,** and a measurement plan attached. A policy with no outcome is an intention.

SEE ALSO Slot II - 33 chronic care in India Slot II - 31 psychosocial management

II - 50

Discuss metabolic side effects of the antipsychotics and strategies used to prevent such side effects. [3+7]

3 + 7 | SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 What the problem is	Five components, the excess risk, the mortality it drives
2 Why it happens	Receptor mechanisms, plus illness and circumstance
3 Prevention	Agent choice, monitoring with a threshold, lifestyle from day one
2 Once it has happened	Switch, metformin, and treating each component

STATE THE SYNDROME, THEN THE STAKE

Central obesity, raised triglycerides, low high density lipoprotein, raised blood pressure and raised fasting glucose. Around a third of patients meet the criteria, roughly twice the background rate, and this is the main reason life expectancy in schizophrenia is fifteen to twenty years shorter.

AGENT	METABOLIC RISK	WHAT FOLLOWS FROM IT
Clozapine, olanzapine	**Highest**	Used where efficacy justifies it, with the tightest monitoring
Quetiapine, risperidone, paliperidone	Intermediate	The common first choices, so the schedule must be real
Aripiprazole, lurasidone, ziprasidone	**Low**	Preferred at first episode and in young patients
Amisulpride, haloperidol	Low for weight	Cost is paid in prolactin and movement instead

Most weight is gained in the first months and is the hardest to reverse, so the first prescription matters more than any later switch.

- **Mechanism.** Histamine H1 and serotonin 5-HT2C blockade drive appetite, muscarinic M3 blockade blunts insulin release, sedation cuts activity. Diet, smoking and low income add to it independently.
- **Monitoring, with the numbers.** Weight, body mass index and waist at baseline, monthly for three months, then quarterly. Blood pressure, fasting glucose or glycated haemoglobin and lipids at baseline, at three months, then yearly. A gain above seven per cent of baseline weight is the trigger to act.
- **Prevention.** Choose a lower risk agent where efficacy allows, start diet and activity alongside the drug rather than after the weight arrives, and name who owns the physical health review.
- **Once it has happened.** Switch where the illness permits, add metformin started at 500 mg and titrated up, which is the best evidenced option, and treat each component to ordinary targets.

WHAT EARNS THE MARKS

- **The five components named,** then linked to the mortality gap rather than left as a list.
- **Agents ranked by risk,** which is what makes the prevention section actionable.
- **A monitoring schedule with intervals and an action threshold.**
- **Metformin named,** and physical health care assigned to a named person rather than left to nobody.

SEE ALSO Slot II - 44 treatment resistance Slot II - 45 first episode pharmacology

II - 51

Aetiology and management of tardive dyskinesia. [10]

10 SCHIZOPHRENIA: TREATMENT AND ANTIPSYCHOTICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Phenomenology, and the exposure rule that allows the diagnosis
3 Aetiology	Receptor supersensitivity, and what has been added to it
2 Who gets it	Risk factors, with the strongest one named first
3 Management	Remove, switch, then the specific drug class

- **What it is.** Involuntary repetitive choreoathetoid movements, classically buccolingual and masticatory, arising after months of dopamine antagonist exposure and persisting after the drug is reduced. Three months of exposure is conventionally required, one month above sixty.
- **Aetiology.** Classically postsynaptic D2 supersensitivity from chronic blockade. Added to it: maladaptive synaptic plasticity, loss of striatal inhibitory interneurons, oxidative injury, and genetic variation in the D2 and D3 receptors.
- **Risk factors.** Older age is the strongest, then cumulative dose and duration, first generation and high potency agents, early acute movement effects, diabetes and mood disorder. Incidence is roughly five per cent a year with older agents, lower but not zero with newer ones.

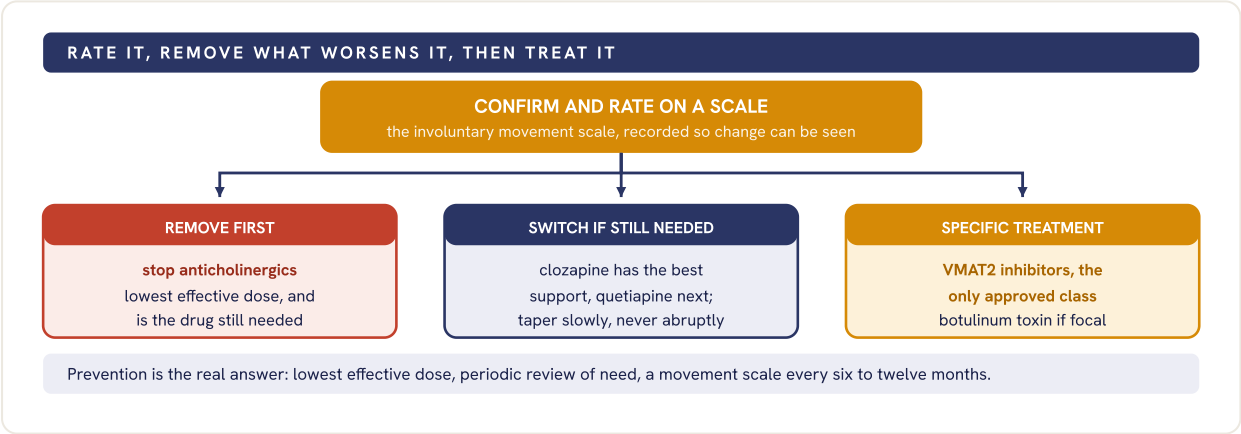

Fig 051.1 Management in order: rate the movements, remove what worsens them, switch if an antipsychotic is still required, then treat.

PITFALL

Adding trihexyphenidyl. It relieves acute dystonia and parkinsonism and makes tardive dyskinesia worse. Valbenazine and deutetrabenazine are the approved agents; in Indian practice tetrabenazine is the accessible member of the class.

WHAT EARNS THE MARKS

- **The exposure rule stated,** so the diagnosis is defined rather than described.
- **Supersensitivity given as the classical account with newer mechanisms added,** not instead of them.
- **Anticholinergic withdrawal written before any new drug,** and the VMAT2 class named.

SEE ALSO Slot II - 47 first generation antipsychotics Slot II - 46 malignant syndrome

AREA 07 OF 14 · P2-A05

Mood disorders: pharmacotherapy

7	7.5%	II-52	II-58
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 RECURRENT 1 SINGLE 5

REVISION ORDER

The two treatment-resistance slots together; one defines it and the other asks for five strategies, and they are the same answer twice. The two ECT slots next, likewise. Lithium and the MAOI slot are single-drug answers. The suicide neurobiology slot last, and it is the one that needs reading of its own.

WHAT IS IN THIS AREA

Treatment-resistant depression in two slots, the monoamine oxidase inhibitors, the long-term adverse effects of lithium, and a suicide slot that asks for the neurobiology and the immunometabolic depression link. Two of the seven slots are electroconvulsive therapy questions: mechanisms, preparation and monitoring in one, and the history, pre-treatment imaging and side effects in the other. The area is named for pharmacotherapy and the boards put the physical treatments in it.

II - 52

Treatment-resistant depression. [10]

10 MOOD DISORDERS: PHARMACOTHERAPY

DNB, KNRUHS, KUHS, MUHS, RGUHS - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2	The definition	Two adequate trials, and what adequate actually means
2	Pseudo-resistance	The four causes excluded before the label is used
4	The drug ladder	Optimise, switch, augment, combine, each given time
2	Beyond drugs	Added therapy, ECT, and the rapid-acting agents

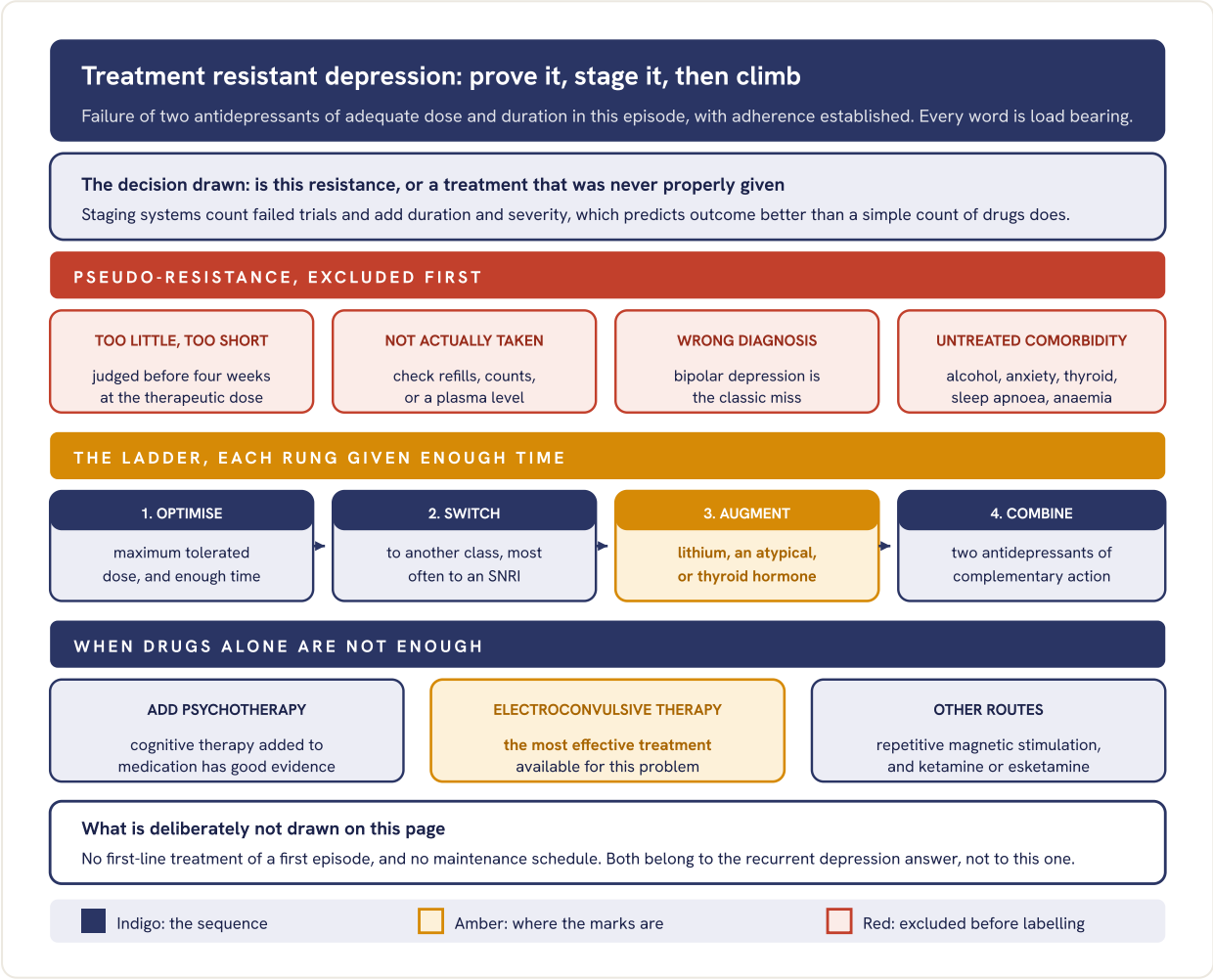

Fig 052.1 The four causes of apparent resistance excluded first, then the drug ladder, then what is done when the ladder runs out.

- **Augmentation, with the detail.** Lithium is the classical agent and carries the anti-suicide evidence. Atypical antipsychotics have the largest randomised evidence base. Thyroid hormone is the third established option, and each augmentation is judged over weeks rather than days.
- **The rapid-acting agents.** Ketamine and esketamine act within hours, including on suicidal thinking, which no oral antidepressant does. The benefit is short lived without repeated dosing, so they buy time rather than replace the ladder.

PITFALL

Counting trials the patient never really had. A drug stopped at two weeks, or at half the therapeutic dose, or never collected from the pharmacy, is not a failed trial, and an answer that does not say so has skipped the first two marks.

WHAT EARNS THE MARKS

- **The definition unpacked,** with adequate dose, adequate duration and confirmed adherence each spelled out.
- **Pseudo-resistance excluded before the ladder starts.**
- **The ladder in order, with augmentation agents named.**
- **Electroconvulsive therapy identified as the most effective option,** not held back as a last resort.

SEE ALSO Slot II - 57 true and pseudo resistance Slot II - 62 recurrent depressive disorder

II - 53

Classify monoamine oxidase inhibitors. What are the common side effects? What instructions would you give to your patient while prescribing one? [3+4+3]

3 + 4 + 3 | MOOD DISORDERS: PHARMACOTHERAPY | DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	The axis and the classes	Selectivity and reversibility, then the drugs in each box
2	Everyday side effects	What actually limits the dose in practice
2	The two emergencies	Hypertensive crisis and serotonin syndrome
3	What the patient is told	Food, drugs, warning signs, the card, the washout

CLASS	DRUGS	WHY THE CLASS MATTERS
Non-selective, irreversible	Phenelzine, isocarboxazid, tranylcypromine	**Full dietary restriction; the tyramine risk lives here**
Selective A, reversible	Moclobemide	**Displaced by tyramine, so the diet is far less restrictive**
Selective B, irreversible	Selegiline, rasagiline	Selective at low dose only; selectivity is lost as the dose rises

The classification is by selectivity and reversibility, and both axes exist only because they change what the patient may eat.

- **Everyday side effects.** Postural hypotension is the usual dose-limiting problem. Then insomnia, weight gain, sexual dysfunction and peripheral oedema. Hydrazines can be hepatotoxic; tranylcypromine is stimulant in character and can be misused.
- **The two emergencies.** Hypertensive crisis from tyramine in food or from a sympathomimetic, and serotonin syndrome from any serotonergic drug added on top. Both are avoidable and both are what the instructions exist to prevent.
- **What the patient is told.** Avoid aged cheese, cured and fermented meat, soy sauce, yeast extract, broad bean pods and draught beer; fresh food is safe. Take nothing new without asking, especially cough remedies, decongestants, pethidine, tramadol and other antidepressants. A severe throbbing headache with palpitations means hospital now. Carry a card, tell any dentist or anaesthetist, and allow a two week washout before a serotonergic drug is started.

WHAT EARNS THE MARKS

- **Both axes of the classification named,** selectivity and reversibility, before the drug list.
- **Moclobemide's lighter dietary restriction explained, not just stated.**
- **The instructions written as instructions,** in words a patient could repeat back, since that is what the last three marks reward.

SEE ALSO Slot II - 52 treatment resistant depression Slot II - 46 malignant syndrome

II - 54

What are the mechanisms of action of ECT? How would you prepare a patient for modified ECT? Write about monitoring of a patient undergoing modified ECT. [3+3+4]

3 + 3 + 4 | MOOD DISORDERS: PHARMACOTHERAPY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Mechanisms	Four hypotheses, and the honest line that none is settled
3	Preparation	Consent, workup, fasting, and the drugs to withhold
2	Monitoring during	Seizure adequacy, and the physiological parameters
2	Monitoring across the course	Response, cognition, threshold, and when to stop

MECHANISMS, AS FOUR HYPOTHESES

Neurotransmitter: enhanced serotonergic and dopaminergic transmission with altered receptor sensitivity. Anticonvulsant: seizure threshold rises across a course, and that rise tracks response. Neuroendocrine: hypothalamic release and normalisation of the stress axis. Neuroplastic: increased neurotrophic factor and hippocampal volume. None is established alone, and saying so is part of the answer.

STAGE	WHAT IS DONE	WHAT IS EASY TO FORGET
Consent	Informed consent, or the nominated representative where capacity is absent	**Unmodified ECT is prohibited; minors need Board permission**
Workup	Physical and cardiac review, haemogram, sugar, electrolytes, renal and liver function, ECG	Imaging only where clinically indicated, never routinely
The night before	Fasting six to eight hours, bladder emptied, dentures and ornaments removed	**Withhold benzodiazepines and anticonvulsants; review lithium**
In the suite	Anticholinergic, induction agent, muscle relaxant, preoxygenation, bite block	A cuffed limb, so the motor seizure can be timed

- **Monitoring during the treatment.** Continuous pulse oximetry, electrocardiogram and blood pressure, with electroencephalographic and motor seizure duration recorded. An adequate generalised seizure is expected, and a missed or very brief seizure is restimulated at a higher charge in the same session.
- **Monitoring across the course.** Symptom response on a rating scale before every second treatment, orientation and memory checked before each session, seizure threshold and charge tracked, and the course ended when improvement plateaus rather than at a fixed number.

WHAT EARNS THE MARKS

- **Four named mechanisms with the honest closing line,** rather than one confident theory.
- **Consent handled under the statute,** including the prohibition on unmodified treatment.
- **Seizure adequacy described as something measured,** with what is done when it is inadequate.
- **Cognitive monitoring given its own place,** because it is what decides whether the course continues.

SEE ALSO Slot II - 56 history and side effects of ECT Slot II - 52 treatment resistant depression

II - 55

Describe the long-term adverse effects of lithium therapy. [10]

10 | MOOD DISORDERS: PHARMACOTHERAPY | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Frame the answer	Long term effects, not toxicity, and why the distinction matters
4	System by system	Kidney, thyroid, parathyroid, then the rest
2	What makes it worse	Drugs, dehydration, and episodes of toxicity
2	Monitoring	What is checked, how often, and when it is checked sooner

SEPARATE TWO QUESTIONS BEFORE STARTING

Long-term adverse effects accumulate at therapeutic concentrations over years. Toxicity is an acute event at high concentrations. An answer that describes tremor, ataxia and confusion has answered the wrong question.

SYSTEM	WHAT HAPPENS	WHAT IS DONE ABOUT IT
Kidney	Nephrogenic diabetes insipidus with polyuria and thirst; slow interstitial nephropathy over decades	**Track the estimated filtration rate; amiloride for the polyuria**
Thyroid	Hypothyroidism and goitre, commoner in women	Thyroxine added; lithium is not stopped for this
Parathyroid	Raised parathyroid hormone with hypercalcaemia	**Check calcium; it is the effect most often missed**
Metabolic and skin	Weight gain, acne, psoriasis worsened, hair thinning	Weight tracked; dermatology for the skin, not withdrawal
Heart and nerve	Benign T-wave change, sinus node dysfunction in the elderly, fine tremor	Propranolol for tremor; electrocardiogram in older patients

- **Monitoring.** A twelve hour post-dose level every three to six months, renal function and thyroid function every six months, calcium once a year, and weight at each review. All of it sooner after a dose change, in the elderly, and in anyone with impaired renal function.
- **What drives the risk up.** Non-steroidal anti-inflammatory drugs, thiazides, drugs acting on the renin-angiotensin system, dehydration in hot weather, and every past episode of toxicity. Cerebellar damage after severe toxicity can be permanent.
- **The line that should end the answer.** None of these is a reason to stop a drug that prevents relapse and reduces suicide. They are reasons to monitor, and to make the decision with the patient.

WHAT EARNS THE MARKS

- **Long-term effects separated from acute toxicity in the first line.**
- **Parathyroid included,** which is the omission that separates a good answer from an average one.
- **A monitoring schedule with intervals,** not a general instruction to check levels.
- **The closing judgement,** that adverse effects are managed rather than used as a reason to stop.

SEE ALSO Slot II - 63 bipolar depression Slot II - 59 course and outcome

II - 56

Briefly describe the history of development of electroconvulsive therapy in psychiatry. What are the indications of brain imaging before modified ECT? What are the side effects of modified ECT? [5+2+3]

5 + 2 + 3 | MOOD DISORDERS: PHARMACOTHERAPY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

5	The history	A wrong idea, a right treatment, then a century of refinement
2	Imaging before ECT	Never routine; the findings that make it necessary
2	Side effects	Immediate, cognitive, cardiovascular, anaesthetic
1	The safety statement	Mortality, put in a comparison a reader can use

STEP	WHAT CHANGED	WHY IT MATTERED
The premise	Meduna induced seizures on the belief that epilepsy and schizophrenia were antagonistic	**The premise was wrong, the treatment worked anyway**
Chemical to electrical	Camphor, then pentylenetetrazol, then Cerletti and Bini in 1938	Electricity made the seizure predictable and timed
Modification	Curare, then succinylcholine with general anaesthesia	Fractures and dislocations effectively disappeared
Refinement	Unilateral placement; sine wave replaced by brief and ultrabrief pulse	**Same efficacy at a much lower cognitive cost**
Regulation	Anaesthesia and muscle relaxant made a legal requirement in India	Unmodified treatment is prohibited outright

- **Imaging is not routine.** It is indicated by clinical findings: focal neurological signs, papilloedema or other evidence of raised intracranial pressure, a new or atypical headache, recent head injury or stroke, unexplained seizures, known intracranial disease, and late onset illness with unexplained cognitive decline.
- **Side effects.** Immediate: headache, jaw and muscle ache, nausea, post-ictal confusion. Cognitive: anterograde amnesia that recovers over weeks, and retrograde loss of autobiographical memory that can persist, worst with bilateral placement, sine wave and high charge. Cardiovascular: a brief bradycardia then tachycardia and hypertension. Rarely prolonged seizure, dental injury or a switch into mania.
- **The safety statement.** Mortality is of the order of a few deaths per hundred thousand treatments, comparable to minor surgery under general anaesthesia, and most of that risk is anaesthetic rather than the seizure.

WHAT EARNS THE MARKS

- **The history told as a sequence with what each step fixed,** not as a list of names and years.
- **Imaging stated as not routine,** then justified by named clinical findings.
- **Cognitive effects split into anterograde and retrograde,** with what makes each worse.
- **A mortality figure with a comparison,** which is what makes the safety claim usable.

SEE ALSO Slot II - 54 mechanisms and monitoring Slot II - 91 catatonia

II - 57

Differentiate between true and pseudo resistance in psychopharmacotherapy. How do we define treatment resistant depression? Enumerate five strategies for managing treatment resistant depression. [3+2+5]

3 + 2 + 5 | MOOD DISORDERS: PHARMACOTHERAPY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The grid	Domain by domain, with what settles each one marked
2 The definition	Two adequate trials, spelled out word by word
5 Five strategies	Exactly five, numbered, in the order they are used

DOMAIN	PSEUDO-RESISTANCE	TRUE RESISTANCE	WHAT SETTLES IT
Dose	Never reached the therapeutic range	Maximum tolerated dose reached	**The prescription record, not the recollection**
Duration	Judged before four weeks at that dose	Four to six weeks at that dose	**Date the dose was reached, not the date started**
Adherence	Partial or covert non-adherence	Adherence established	**Pharmacy refills, counts, a plasma level**
Diagnosis	Bipolar depression, dysthymia, substance use	The diagnosis holds on review	**A life chart and collateral history**
Comorbidity	Untreated anxiety, alcohol, thyroid, sleep apnoea	Comorbidity identified and treated	Screening bloods and a sleep history

The definition applies only after every left-hand cell has been excluded, which is the point of putting the grid before the definition.

- **The definition.** Failure to respond to two or more antidepressants, each of adequate dose and adequate duration, in the current episode, with adherence established. Staging systems refine it by adding episode duration and symptom severity, which predict outcome better than a count of failed drugs alone.
- **Five strategies.** One, optimise to the maximum tolerated dose for long enough. Two, switch to another class. Three, augment with lithium, an atypical antipsychotic or thyroid hormone. Four, combine two antidepressants of complementary action. Five, neurostimulation, where electroconvulsive therapy is the most effective option available.
- **Two things that are not on that list of five but belong in the plan.** Cognitive therapy added to the medication, and treating the comorbidity that is holding the depression up.

WHAT EARNS THE MARKS

- **A grid, not two paragraphs,** with the settling test named in each row.
- **The definition given word by word,** since adequate dose, adequate duration and adherence each carry weight.
- **Exactly five strategies, numbered.** The stem asks for five, and six is as wrong as four.

SEE ALSO Slot II - 52 treatment resistant depression Slot II - 44 treatment resistant schizophrenia

II - 58

What are the evidence based risk and protective factors of suicide? Describe the neurobiology of suicide. Discuss the relationship between immunometabolic depression and antidepressants. [4+3+3]

4 + 3 + 3 | MOOD DISORDERS: PHARMACOTHERAPY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	Risk and protection	Distal and proximal risk, protection, and the Indian pattern
3	Neurobiology	The stress-diathesis model, and the findings under each half
3	Immunometabolic depression	What the construct is, and what it predicts about treatment

LAYER	RAISES RISK	PROTECTS
History	**A previous attempt, the strongest single predictor**	Engagement with treatment that is continuing
Illness	Mood disorder, substance use, psychosis, personality disorder	Treatment of the disorder itself
State	Hopelessness, agitation, insomnia, recent discharge	Stated reasons for living, responsibility to children
Situation	Access to means, isolation, debt, chronic pain, a humiliating loss	**Restricting access to means, which changes population rates**

In India the leading methods are hanging and pesticide poisoning, rates are highest between fifteen and twenty nine, and the law now treats an attempt as evidence of severe stress rather than as a crime.

- **Neurobiology.** A stress-diathesis model: the diathesis is partly independent of the psychiatric diagnosis. Reduced serotonergic function is the most replicated finding, with low cerebrospinal 5-hydroxyindoleacetic acid in violent attempters and altered receptor binding in ventral prefrontal cortex, the region that restrains action on impulse. Add stress axis dysregulation, reduced neurotrophic signalling, and inflammatory changes through the kynurenine pathway.
- **Immunometabolic depression.** A proposed dimension in which energy-related atypical symptoms, hypersomnia, increased appetite, weight gain and leaden fatigue, cluster with obesity, insulin resistance, dyslipidaemia and low grade inflammation on markers such as C-reactive protein.
- **Why it matters for antidepressants.** This profile predicts poorer response to conventional antidepressants, and several of them, notably mirtazapine, paroxetine and the tricyclics, worsen the metabolic picture further. The relationship runs both ways, so agent choice and metabolic monitoring belong together in these patients.

WHAT EARNS THE MARKS

- **Risk layered into history, illness, state and situation,** with protection answered as its own column.
- **Means restriction named,** since it is the intervention with population-level evidence.
- **The stress-diathesis frame stated before the findings,** so the neurobiology reads as a model rather than a list.
- **The bidirectional relationship in the last part,** which is what the word discuss is asking for.

SEE ALSO Slot II - 52 treatment resistant depression Slot II - 59 course and outcome

AREA 08 OF 14 · P2-A04

Mood disorders: syndromes and nosology

7	7.5%	II-59	II-65
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 6

REVISION ORDER

The three bipolar slots first and in that order, because they build on one another: course and outcome, then the depressive episode, then the spectrum. Recurrent depressive disorder next. Genetics, the seasonal slot and the postpartum slot are independent of all of it.

WHAT IS IN THIS AREA

The course and outcome of bipolar affective disorder, bipolar depression, the bipolar spectrum with bipolar II in detail, the genetics of bipolar disorder with genetic counselling, recurrent depressive disorder, the seasonal affective disorder slot that opens on climate and mental health, and the postpartum slot.

II - 59

Course and outcome of bipolar affective disorder. [10]

10 MOOD DISORDERS: SYNDROMES AND NOSOLOGY

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Onset	Age, the depressive first episode, and the diagnostic delay
3	The pattern over time	Kindling, shortening cycles, predominant polarity
3	Between episodes	Subsyndromal symptoms, cognition, functional recovery
2	Outcome and mortality	Suicide, physical mortality, what predicts a worse course

THE KINDLING MODEL, STATED FIRST

Early episodes follow identifiable stressors; later ones become autonomous, and the interval between them shortens. Whatever its final status as a mechanism, it is the frame that makes early and continuous treatment the logical conclusion of the answer.

FEATURE	WHAT FOLLOW-UP SHOWS	WHAT FOLLOWS FROM IT
Onset	Late teens to mid twenties, usually a depressive episode	Years of delay before the diagnosis is corrected
Episodes	Frequency rises and cycle length shortens untreated	Prophylaxis is judged over years, not months
Between episodes	Subsyndromal symptoms in over half; depression dominates	**Residual symptoms are the strongest relapse predictor**
Rapid cycling	Develops in fifteen to twenty per cent	Review thyroid and any antidepressant exposure

Depressive weeks far outnumber manic weeks across follow-up, which is why an answer organised around mania describes the wrong illness.

- **Functional recovery lags symptomatic recovery.** Fewer than half return to premorbid work and social function even when the episode has resolved, and cognitive impairment in attention, verbal memory and executive function persists into euthymia.
- **Mortality.** Lifetime suicide is of the order of one in twenty, many times the population rate and highest early in the illness and in mixed states. Cardiovascular death accounts for most of the shortened life expectancy.
- **What predicts a worse course.** Early onset, psychotic and mixed features, rapid cycling, comorbid substance use, poor adherence and a higher count of prior episodes.

WHAT EARNS THE MARKS

- **Kindling named, then used** to justify early and continuous treatment.
- **Depression identified as the dominant burden,** which most answers invert.
- **Functional and cognitive outcome separated from symptomatic outcome.**
- **Both mortality routes given,** suicide and cardiovascular disease.

SEE ALSO Slot II - 63 bipolar depression Slot II - 65 bipolar II disorder **SPRINT**

Day 11, Mood disorders: classification, depressive and bipolar syndromes

II - 60

Describe the genetics of bipolar disorder and enumerate principles of genetic counselling. [10]

10 MOOD DISORDERS: SYNDROMES AND NOSOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The size of the effect	Heritability, and which design that number comes from
4	The four designs	Family, twin, adoption, molecular, each with its finding
2	What molecular work found	Polygenic, shared across diagnoses, not yet predictive
2	Counselling	Empirical risk, non-directive stance, guilt, what is modifiable

NAME THE AXIS BEFORE THE LIST

The classification here is by study design, because each design answers a different question: family studies show aggregation, twin studies separate genes from shared environment, adoption studies break the two apart, and molecular work asks which genes.

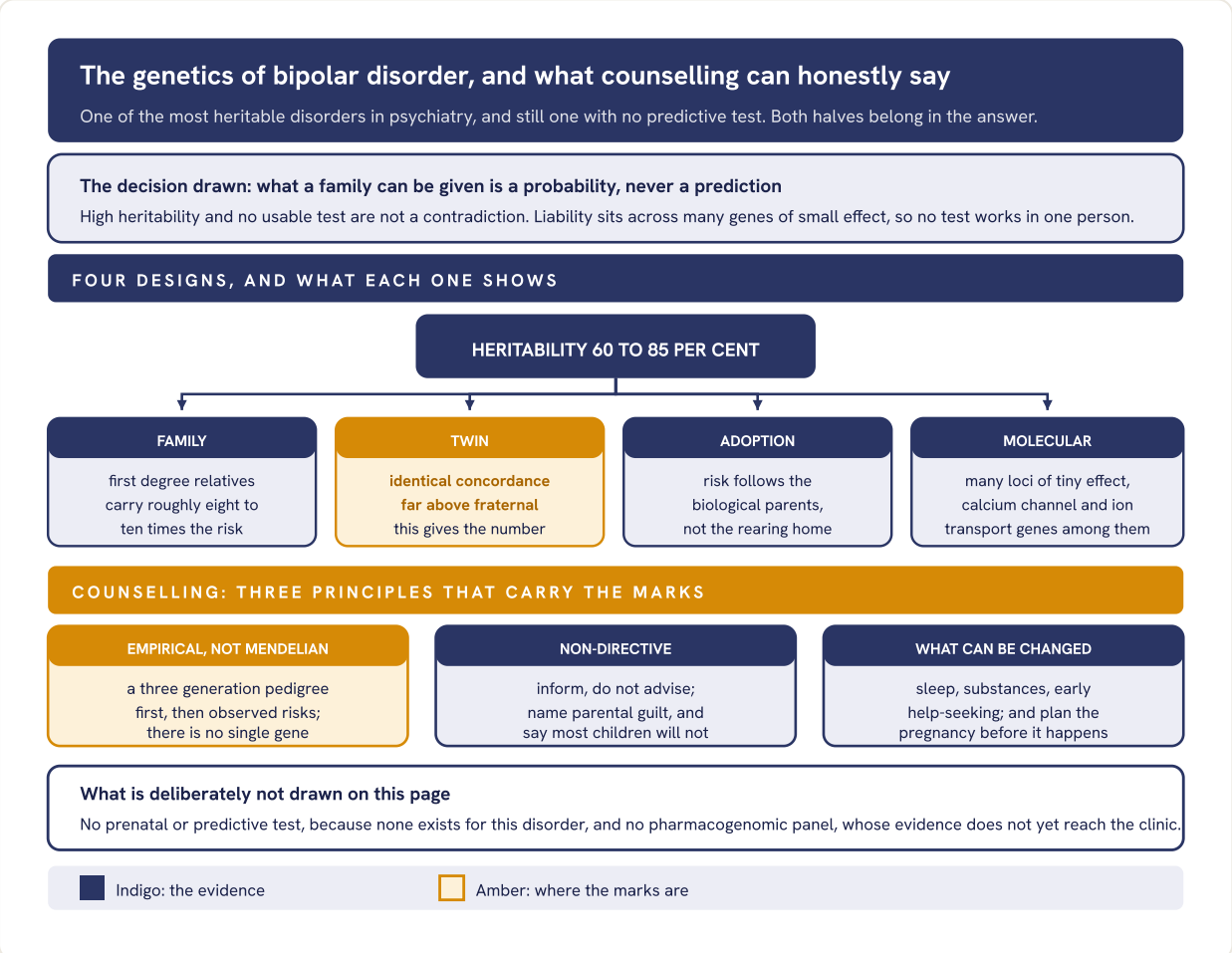

Fig 060.1 The four study designs as one tree, with the counselling principles carried below them because each principle follows from what the design above it can and cannot show.

- **What molecular work actually found.** Liability is polygenic: dozens of loci, each of very small effect, with substantial genetic overlap with schizophrenia and with depression. Polygenic risk scores explain a small fraction of variance and are not clinical instruments.
- **The counselling sentence that matters most.** High heritability is a statement about a population, not about one family, and it never entitles anyone to predict a particular child.

WHAT EARNS THE MARKS

- **The four designs distinguished by what each can show,** not merged into one paragraph on heredity.
- **Polygenicity stated,** with the honest line that no predictive test exists.
- **Counselling given as principles, not as a risk figure,** with guilt and non-directiveness both named.

SEE ALSO Slot II - 59 course and outcome Slot II - 34 endophenotypes

II - 61

Describe the effect of climate on mental health problems. Write the management of a case with seasonal affective disorder. How does light therapy work? [3+4+3]

3 + 4 + 3 | MOOD DISORDERS: SYNDROMES AND NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Climate and mental health**	Heat, disaster, drought, and the drug-specific hazards
2	**The seasonal disorder**	The specifier, the atypical picture, the latitude effect
3	**Management**	Light first, then drugs, then what prevents next winter
2	**How light works**	Retina to hypothalamus, phase advance, melatonin

- **Climate and mental health.** Rising temperature tracks with violence and with suicide rates; disasters bring post-traumatic stress and depression; drought and crop failure drive rural distress and suicide in India.
- **The drug-specific hazards.** Heat with dehydration pushes lithium toward toxicity, and anticholinergic and antipsychotic drugs impair sweating, so heatstroke is likelier in the people we treat.
- **The seasonal specifier.** A pattern, not a separate illness: episodes tied to a season across at least two consecutive years, seasonal clearly outnumbering non-seasonal. The winter type is atypical, with hypersomnia, carbohydrate craving and weight gain, and it rises with latitude.

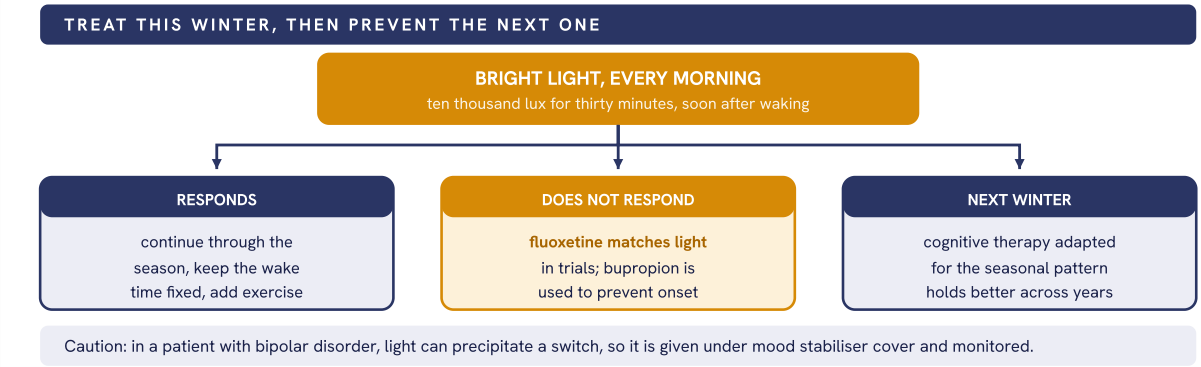

Fig 061.1 Management of the seasonal pattern, with prevention of the following winter treated as part of the same plan.

HOW LIGHT THERAPY WORKS

Light reaches melanopsin-containing retinal ganglion cells, which project through the retinohypothalamic tract to the suprachiasmatic nucleus. Morning light advances a delayed rhythm and suppresses melatonin. Side effects: headache, eye strain, switch.

WHAT EARNS THE MARKS

- **Climate answered as more than heat,** with the lithium and thermoregulation hazards named.
- **The seasonal specifier given with its two-year rule,** and the atypical symptom picture.
- **Light described with a dose, a timing and a mechanism,** which is where the last three marks are.

SEE ALSO Slot II - 62 recurrent depressive disorder Slot II - 63 bipolar depression

II - 62

Discuss the management of recurrent depressive disorder. [10]

10 MOOD DISORDERS: SYNDROMES AND NOSOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **What it commits you to**	Two episodes, no mania, so prophylaxis is the question
3 **Acute phase**	Drug choice, adequate dose and duration, measured response
2 **If it does not respond**	Check, optimise, switch, augment, then ECT
3 **Continuation and after**	How long, at what dose, who needs indefinite treatment

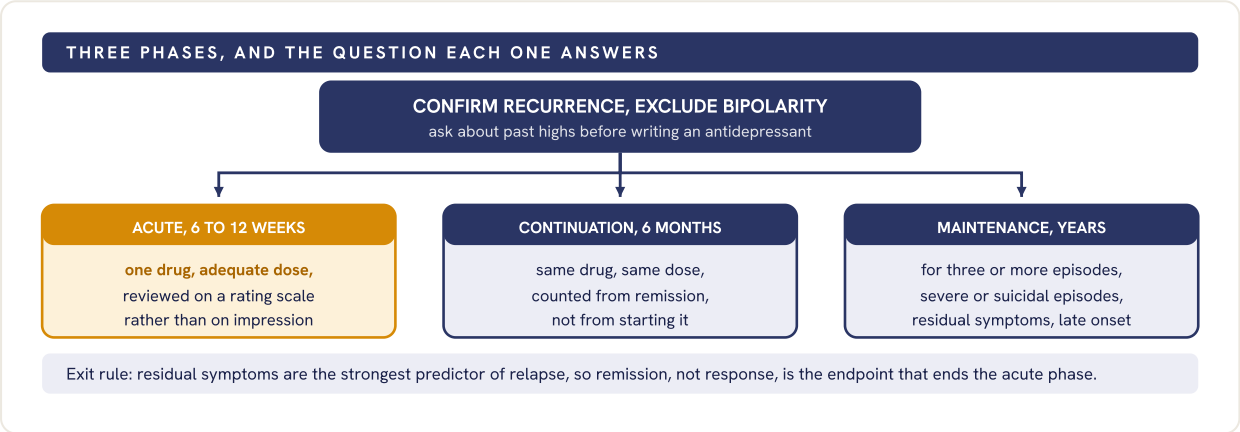

Fig 062.1 The three treatment phases, with the criteria that decide who moves from continuation into indefinite maintenance.

- **If it does not respond.** In order: check adherence and the dose actually taken, review the diagnosis, optimise to the maximum tolerated dose, then switch class or augment with lithium, an atypical or thyroid hormone. ECT for severe, psychotic or suicidal illness.
- **Psychological treatment.** Cognitive behavioural therapy or interpersonal therapy in the acute phase, and mindfulness-based cognitive therapy specifically for relapse prevention in those with three or more episodes.
- **The rest of the plan.** Treat alcohol use, sleep and thyroid disease, address the maintaining stressor, and write a relapse plan the patient keeps.

PITFALL

Stopping the antidepressant when the patient feels better. Continuation is counted from remission and at the dose that produced it; halving it is the commonest way an otherwise correct plan produces a relapse.

WHAT EARNS THE MARKS

- **Three phases named, each with its duration.** This is the structure the question is testing.
- **The maintenance criteria listed,** rather than a vague statement that some patients need long treatment.
- **Remission, not response, given as the endpoint,** with residual symptoms tied to relapse risk.

SEE ALSO Slot II - 52 treatment resistant depression Slot II - 63 bipolar depression

II - 63

Discuss management of a patient with bipolar affective disorder presenting in a depressive episode. [10]

10 MOOD DISORDERS: SYNDROMES AND NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Assess first	Mixed features, rapid cycling, suicide risk, current drugs
4 First line treatment	The agents with evidence, and what each is chosen for
2 The antidepressant	When it may be used, and when it must not be
2 After recovery	Continue what worked, and the anti-suicide argument

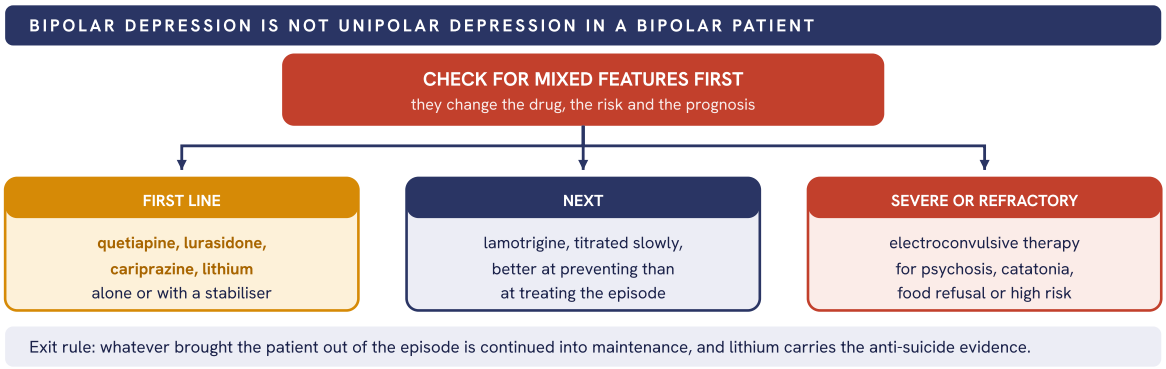

Fig 063.1 The treatment sequence for a depressive episode in bipolar disorder, entered through the mixed-features check because that answer changes everything below it.

- **The antidepressant question.** Monotherapy is not acceptable: efficacy is weak and the risks are a switch and accelerated cycling. If used at all it goes on top of an antimanic agent, and is avoided entirely in mixed features and rapid cycling.
- **Assessment that changes the plan.** Past highs, mixed features now, rapid cycling in the last year, any antidepressant already started, thyroid and renal function before lithium, and suicide risk recorded.
- **Psychological work.** Cognitive behavioural, family-focused and interpersonal and social rhythm therapy all have evidence, and routine and sleep stabilisation starts immediately.

PITFALL

Treating it as unipolar depression because that is what the patient presents with. An antidepressant alone here is the commonest prescribing error in this scenario.

WHAT EARNS THE MARKS

- **The mixed-features check placed before any prescription.**
- **Named first line agents,** rather than "a mood stabiliser" left unspecified.
- **The antidepressant answered explicitly,** including when it must not be used.
- **Continuation into maintenance,** with lithium's anti-suicide evidence stated.

SEE ALSO Slot II - 59 course and outcome Slot II - 65 bipolar II disorder

II - 64

What are the common psychiatric problems seen during the post-partum period? Write in detail about any one of these. What are the prognostic implications of post-partum psychiatric disorders for future mental health? [2+5+3]

2 + 5 + 3 | MOOD DISORDERS: SYNDROMES AND NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The three, separated	Blues, depression, psychosis, by timing and by frequency
3	Psychosis: the picture	Rapid onset, perplexity, and the two risks that define it
2	Psychosis: management	Admit, exclude the medical causes, treat, supervise
3	Prognostic implications	Recurrence, the bipolar link, and the effect on the child

CONDITION	HOW COMMON	ONSET	WHAT IT NEEDS
Postpartum blues	Most mothers	Days three to five	Explanation and support; it resolves
Postpartum depression	About one in ten	Weeks to months	Screening, therapy, an antidepressant if moderate
Postpartum psychosis	One or two per thousand	**First two weeks**	**Emergency admission and supervision of the infant**
Anxiety and obsessional states	Common, under-recognised	Any point	Distinguish intrusive thoughts from intent

Onset timing is the single most useful discriminator, which is what the amber cells mark.

POSTPARTUM PSYCHOSIS, IN DETAIL

Onset within days, with perplexity and a fluctuating, almost delirious quality, labile mood and delusions that often concern the baby. An emergency, because of suicide and harm to the infant. Infection, eclampsia and thyroiditis are excluded.

- **Management.** Admit, ideally with the baby where a mother and baby facility exists, and never leave the infant unsupervised until risk has been assessed. An antipsychotic with lithium is the usual combination, and electroconvulsive therapy works quickly and well when the illness is severe.
- **Prognosis for the mother.** Postpartum psychosis is usually the first appearance of a bipolar diathesis. Recurrence after a further delivery runs at roughly one in three to one in two, and more than half go on to have episodes unrelated to childbirth.
- **The child and the next pregnancy.** Untreated maternal depression affects infant cognitive, behavioural and attachment outcomes. Prophylaxis started immediately after the next delivery reduces recurrence.

WHAT EARNS THE MARKS

- **All three separated by timing and frequency** before any one is described.
- **Psychosis named as an emergency,** with both risks and the medical exclusions.
- **The bipolar link stated,** with the child and the next pregnancy included, since the stem asks about future mental health.

SEE ALSO Slot II - 59 course and outcome Slot II - 63 bipolar depression

II - 65

What is meant by bipolar spectrum? Discuss the clinical features, comparative nosology, course, outcome and management of bipolar II disorder. [10]

10 | ASKED AT 15 MARKS | MOOD DISORDERS: SYNDROMES AND NOSOLOGY

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The spectrum idea	What it claims, and the objection it has to answer
4 The grid	Domain by domain against bipolar I, discriminators marked
2 Course and outcome	Depression dominates, and what that costs
2 Management	What is treated, and the antidepressant caution

DOMAIN	BIPOLAR I	BIPOLAR II	WHAT SETTLES IT
The high	Mania, one week or admission	Hypomania, at least four days	Has there ever been a manic episode
Impairment in the high	Marked, work and relationships break	An unmistakable change, function largely held	Was function actually lost
Psychosis in the high	May be present	Never, by definition	Psychosis in a high makes it bipolar I
Depression	Present, but shares the burden	Dominates the course by a wide margin	Depressive weeks outnumber hypomanic ones heavily
How it is found	Usually diagnosed in the manic episode	Usually missed for years	Collateral history for past hypomania

Not a milder illness. It carries comparable suicide risk, more chronic depression and more comorbidity than bipolar I.

- **The spectrum idea.** That bipolarity is dimensional rather than categorical, running from full mania through hypomania and antidepressant-associated highs to hyperthymic temperament. It explains cases that fall between the categories.
- **The objection.** Widening the boundary risks recoding temperament and irritability as illness, and prescribing mood stabilisers to people the trials never studied. A good answer states both the case and the objection.
- **Management.** Quetiapine has the strongest evidence for the depressive phase, with lithium, lamotrigine and lurasidone as alternatives. Antidepressant monotherapy carries switch and cycling risk here too. Add psychoeducation, routine and sleep stabilisation, and continue what worked into maintenance.

WHAT EARNS THE MARKS

- **The grid, with the discriminators identified as discriminators.** A list of ten differences without ranking is a mid-band answer.
- **The spectrum argued from both sides,** because the stem asks for comparative nosology.
- **The explicit statement that it is not the milder illness,** which is the line most answers get wrong.

SEE ALSO Slot II - 59 course and outcome Slot II - 63 bipolar depression

AREA 09 OF 14 · P2-A13

Psychotherapies

7	6.9%	II-66	II-72
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 6

REVISION ORDER

The two cognitive slots together, they are one answer at two lengths. Brief psychodynamic and couple therapy next. The yoga and mobile therapy slots share a critical-appraisal shape and prepare as one. Mutism last, and separately.

WHAT IS IN THIS AREA

Cognitive therapy and the components of cognitive behaviour therapy in two slots, brief psychodynamic therapies, couple therapy with its types and contraindications, the evidence base for yoga in psychiatric practice with the research methodology it raises, and a critique of mobile-based psychotherapies. The seventh slot asks about mutism, its causes and the organic against psychological differential. That is a clinical question the boards set inside this area, and it is answered on its page as it was set.

II - 66

Write a note on cognitive therapy. [10]

10 PSYCHOTHERAPIES KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 The model	Three levels of cognition, and the maintenance cycle
3 Structure	Formulation, agenda, homework, time-limited course
3 Techniques	Cognitive and behavioural, named with what each is for
2 Evidence and limits	Where it works, and where it is not first choice

THE MODEL, IN ONE PARAGRAPH

Beck's proposition is that it is the appraisal of an event, not the event, that produces the emotion, and that appraisal runs at three levels: automatic thoughts at the surface, intermediate beliefs as rules and assumptions, and core beliefs about self, others and the world. Therapy makes the appraisals explicit, tests them against evidence, and changes the behaviour that keeps them untested.

TECHNIQUE	WHAT IT DOES	WHAT IT IS FOR
Thought record	Situation, thought, emotion, evidence, alternative	**Makes automatic thoughts visible**
Socratic questioning	Guided discovery rather than persuasion	The patient reaches the conclusion
Behavioural experiment	A prediction is written down, then tested in life	**The strongest belief-change tool**
Activity scheduling	Graded activity with mastery and pleasure ratings	Depression, early sessions
Downward arrow	Repeated "what would that mean" questioning	Reaches the core belief

Amber marks the two techniques an examiner expects by name and by mechanism. The rest support them.

- **Evidence and limits.** Strong support in depression, anxiety disorders, obsessive-compulsive disorder and post-traumatic stress disorder, and as an adjunct in psychosis and bipolar disorder. It asks for a degree of reflective capacity, and in acute severe illness it follows stabilisation rather than replacing it.

WHAT EARNS THE MARKS

- **The three levels of cognition,** named in order and distinguished from one another.
- **Structure described as structure:** shared formulation, set agenda, homework between sessions, a fixed number of sessions.
- **Techniques tied to their purpose,** which is what the grid does and a bare list does not.
- **A stated limit.** Naming where it is not first choice reads as command of the method.

SEE ALSO Slot II - 71 components of cognitive behaviour therapy Slot II - 70 brief psychodynamic therapies SPRINT Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 67

Give an account of the evidence based integration of yoga into psychiatric practice. What methodological issues arise in research in this field? [5+5]

5 + 5 | PSYCHOTHERAPIES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What is tested	Yoga is a package, so name its components first
3	The evidence	Condition by condition, with the study type
2	Mechanism	What is measured to change biologically
3	Methods	Why these trials are hard to read

- **What is being tested.** Not one intervention but a package: postures, regulated breathing, meditation and an ethical frame. Trials differ in which components they deliver, so two studies of "yoga" may share almost nothing.

CONDITION	WHAT THE EVIDENCE SHOWS	STUDY BASE	STATUS
Depression	Adjunctive benefit; some support as monotherapy in milder illness	Randomised trials, meta-analysed	**Adjunct, reasonable**
Schizophrenia	Add-on gains in negative symptoms and social cognition; little on positive symptoms	Indian randomised add-on trials	**Adjunct, promising**
Anxiety and stress	Reduction in symptom scores, weaker than for depression	Smaller trials, mixed controls	Supportive only
Insomnia	Modest gains in sleep quality	Small trials	Preliminary
Mechanism studies	Raised brain-derived neurotrophic factor, lowered cortisol, higher vagal tone	Biomarker substudies	Hypothesis generating

STATUS: WHY THE TRIALS ARE HARD TO READ

Participants and instructors cannot be blinded, so expectancy is unavoidable. Waitlist controls inflate the effect, and few studies use an active comparator matched for attention and contact time. The intervention is not standardised in content, dose or teacher training. Samples are small, follow up is short, and attrition is high. Allegiance and publication bias run in one direction in this literature. Outcome measures are not always validated in the population studied. The honest position is that yoga is a reasonable adjunct with a real but modest and imprecisely measured effect, not a replacement for established treatment.

WHAT EARNS THE MARKS

- **Naming the components first,** because the heterogeneity argument depends on it.
- **Each condition tied to its study type,** not a general claim that yoga helps.
- **Blinding and the choice of control named as the two central problems.**
- **The status line:** adjunct rather than replacement, stated in one sentence.

SEE ALSO Slot II - 69 mobile based psychotherapies **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 68

What is couple therapy? What are the different types of couple therapy? Write the contraindications of couple therapy. [2+6+2]

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

SKELETON

2 Definition	The relationship, not a partner, is the patient
2 The axis	Classify on what the model changes first
4 The schools	Four families, drawn, with the best evidenced named
2 Contraindications	Safety first, then agenda, then capacity

- **Definition.** A psychological treatment in which the couple relationship is the unit of intervention and both partners attend together, directed at the pattern between them rather than at one partner's symptoms.

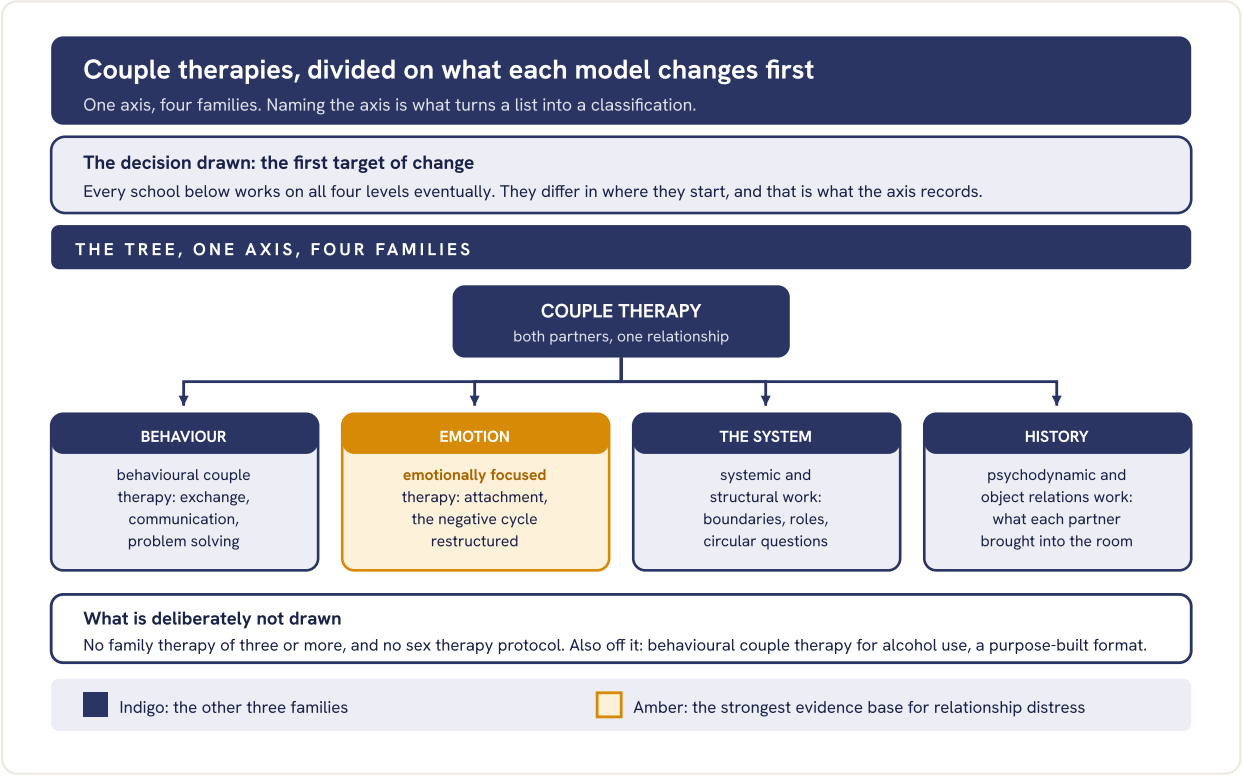

Fig 068.1 One axis, the first target of change, dividing the field into four families, with the emotionally focused branch marked because it carries the largest trial base.

CONTRAINDICATIONS

Ongoing intimate partner violence or coercive control, where joint sessions can raise risk and individual safety work comes first. One partner having privately decided to separate and using the sessions to soften the exit. A secret affair the therapist is asked to hold. Acute psychosis, intoxication or untreated severe illness in either partner. A partner attending under pressure rather than by choice.

WHAT EARNS THE MARKS

- **The axis stated before the list,** since a classification without an axis is only a list.
- **Every branch of the tree reproduced,** with one named model in each.
- **Violence named first among contraindications,** which is the safety mark.

SEE ALSO Slot II - 70 brief psychodynamic therapies **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 69

Write a critique of mobile based psychotherapies in mental disorders. [10]

10 PSYCHOTHERAPIES DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What they are	Guided, unguided, blended, and conversational agents
4	Claim against evidence	Take each promise and test it
2	Risks	Safety, privacy, and who is left out
2	Where it fits	A step in stepped care, not a replacement

- **What they are.** Structured psychological interventions delivered by smartphone: unguided self-help apps, guided programmes with brief clinician contact, blended care alongside face-to-face sessions, and conversational agents.

THE CLAIM	WHAT THE EVIDENCE ACTUALLY SHOWS	THE CRITIQUE
It works	Small to moderate effects in depression and anxiety, larger when guided	**Effects shrink sharply against active controls**
It scales	Reach is real where a therapist workforce does not exist	Language, literacy, shared devices and connectivity exclude many
It is used	Adherence in trials is supported by research contact	**Real-world engagement falls away within weeks**
It is safe	Trials exclude acute risk and severe illness	Most apps have no pathway when risk is disclosed
It is regulated	A minority of store apps carry any trial evidence	Claims are largely unpoliced, and data sharing is common

Amber marks the two lines that carry the critique: the comparator problem and the gap between trial adherence and real use.

PITFALL

Writing a critique that is only a list of benefits with a caveat at the end. A critique takes each claim in turn and says what supports it and what does not. The balanced position is that guided digital therapy has a defensible place at the low-intensity step of stepped care, with a named clinician attached, and no place as a substitute in severe illness or where risk is active.

WHAT EARNS THE MARKS

- **Guided separated from unguided** in the first two lines, since the effect sizes differ.
- **The comparator argument,** that waitlist controls flatter these trials.
- **The engagement gap between trial and real use,** stated as the central practical objection.
- **A placed conclusion,** naming the step of care where it belongs rather than approving or dismissing it.

SEE ALSO Slot II - 67 yoga in psychiatric practice SPRINT

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 70

Discuss brief psychodynamic therapies. [10]

10 PSYCHOTHERAPIES KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	What makes them brief	Focus, time limit, therapist activity, worked ending
4	The schools	Each with its signature move and its length
2	Selection	Who does well, and who should not be offered it
1	Evidence	Where it stands against cognitive therapy

WHAT MAKES A DYNAMIC THERAPY BRIEF

Four features, present in all of them: a single agreed focus rather than the whole personality; a time limit set at the outset and named to the patient; an active therapist who works within Malan's triangle of conflict and triangle of person; and termination treated as therapeutic material from the first session rather than as an administrative ending.

SCHOOL	THE SIGNATURE MOVE	TYPICAL LENGTH
Malan	Brief focal therapy; the two triangles made explicit	**About twenty to thirty sessions**
Sifneos	Anxiety-provoking therapy on an oedipal focus, with a high selection bar	Twelve to twenty sessions
Davanloo	Sustained active challenge to defences early in the work	Up to about forty sessions
Mann	A central issue about separation and time; the limit is itself the treatment	**Exactly twelve sessions**
Luborsky	Supportive-expressive work on the core conflictual relationship theme	Sixteen to twenty sessions

Amber marks the two most quotable pairings. Attaching a length to a name is the cheapest mark in this question.

- **Selection.** Psychological mindedness, capacity to form an alliance quickly, a circumscribed focus, motivation, tolerance of anxiety, and at least one good relationship in the history.
- **Not offered where** there is psychosis, active dependence, poor impulse control, acute risk, or personality pathology that needs a longer frame. Interpersonal psychotherapy grew from this tradition but is not itself psychodynamic.

WHAT EARNS THE MARKS

- **The four shared features named before any school,** which is the structure of the answer.
- **Malan's two triangles by name,** since they are the shared vocabulary of the field.
- **Schools with lengths attached,** read straight off the grid.
- **Selection criteria and exclusions given separately,** not merged into one paragraph.

SEE ALSO Slot II - 66 cognitive therapy Slot II - 68 couple therapy **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 71

What do you understand by the term cognitive behaviour therapy? Discuss in detail its main components. [3+7]

3 + 7 | PSYCHOTHERAPIES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The term	A family of treatments, not one therapy
4 The components	Formulation, behavioural, cognitive, relapse work
2 Session architecture	What every hour looks like, in order
1 The third wave	What was added, and what it changed

THE TERM

Not a single therapy but a family of structured, time-limited, problem-focused treatments sharing one proposition: thought, feeling, body and behaviour maintain one another, so changing any of them changes the rest. What makes a treatment a member of the family is the architecture, a shared formulation, a session structure and homework, rather than any particular technique.

COMPONENT	WHAT IS DONE	WHY IT IS IN THE PACKAGE
Formulation	A maintenance cycle drawn with the patient, plus the longitudinal account	**Everything else is chosen from it**
Psychoeducation	The model explained and the patient socialised to it	Consent and collaboration
Behavioural work	Activation, graded exposure with response prevention, experiments, skills	**Carries much of the effect**
Cognitive work	Thought records, evidence testing, downward arrow to core beliefs	Changes the appraisal
Homework	Agreed tasks between sessions, reviewed at the next	**Dose relates to outcome**
Relapse prevention	A written blueprint: warning signs, what worked, the plan	Protects the gain

Amber marks the three components that carry the marks: the formulation everything hangs on, the behavioural work, and the homework.

- **Every session, in order.** Mood check, agenda set together, homework reviewed, one or two agenda items worked, summary and feedback taken, new homework agreed. Naming this sequence is worth as much as naming a technique.

WHAT EARNS THE MARKS

- **The word family in the definition,** since the question asks what the term means.
- **Behavioural components given real weight,** not treated as an afterthought to the cognitive ones.
- **The session sequence written out,** which few answers do and every supervisor recognises.
- **The third wave named:** acceptance and commitment therapy, mindfulness-based cognitive therapy, dialectical behaviour therapy and metacognitive therapy.

SEE ALSO Slot II - 66 cognitive therapy, the model in depth **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 72

What is mutism? What are its causes? How will you differentiate organic from psychological mutism? Discuss the management of mutism. [1+2+4+3]

1 + 2 + 4 + 3 | PSYCHOTHERAPIES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1 Definition	And the four things it is not
2 Causes	Neurological, psychiatric, drug related
4 The differentiation	Domain by domain, with what settles each
3 Management	Support, the challenge test, then the cause

- **Definition and causes.** Absence or marked reduction of speech in a person who has language, with hearing and the speech apparatus intact. It is not aphasia, dysarthria, aphonia or alogia. Neurological causes include akinetic mutism, frontal and supplementary motor lesions, posterior fossa surgery in children, seizures and delirium. Psychiatric causes include catatonia, depressive stupor, dissociative mutism, selective mutism, and autism with intellectual disability.

DOMAIN	ORGANIC	PSYCHOLOGICAL	WHAT SETTLES IT
Onset	With a neurological event or illness	With a psychosocial precipitant	History from an informant
Consciousness	Often clouded or fluctuating	Clear	**Level of awareness**
Focal signs	Gaze palsy, pyramidal or cerebellar signs	Absent	**Neurological examination**
Catatonic signs	Usually absent	Posturing, negativism, waxy flexibility, echophenomena	Bedside catatonia rating
Lorazepam challenge	No change	Catatonic mutism often eases within minutes	**The single most useful test**
Investigations	Imaging or electroencephalogram abnormal	Normal	Imaging and electroencephalogram

Amber cells are the discriminators. The remaining rows are supporting evidence, and saying which is which is itself a mark.

- **Management.** Support first: airway, hydration, nutrition, pressure areas and thromboprophylaxis in stupor. Then the lorazepam challenge and, if it responds, a lorazepam course for catatonia. Electroconvulsive therapy where catatonia does not respond or where there are malignant features, with antipsychotics withheld in that situation. Otherwise treat the cause found: the lesion, the mood disorder, or behavioural and family work with a serotonergic agent in selective mutism.

WHAT EARNS THE MARKS

- **Mutism separated from aphasia, dysarthria and aphonia** in the opening line.
- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **The lorazepam challenge named as the decisive bedside test,** and not buried in management.
- **Withholding antipsychotics in malignant catatonia,** which is the safety line in this answer.

SEE ALSO Slot II - 66 cognitive therapy SPRINT Day 19, Psychotherapies, clinical assessment, MSE and nosology

AREA 10 OF 14 · P2-A10

Personality disorders

7	6.6%	II-73	II-79
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 SINGLE 5

REVISION ORDER

Borderline first and hardest: it carries the highest-scoring question in this book, and the caregiver slot reuses it. Cluster B next, which the borderline answer already half writes. The classification-change slot after that. Organic personality disorder, the avoidant slot and the law slot last.

WHAT IS IN THIS AREA

Borderline personality disorder in two slots, one on clinical features and management and one on caregiver burden, cluster B as a group, the anxious-avoidant presentation, organic personality disorder, the recent changes to the classification of personality and substance use disorders, and the slot on personality disorder at the interface with the law.

II - 73

Discuss clinical features and management of borderline personality disorder. [4+6]

4 + 6 PERSONALITY DISORDERS

DNB, KNRUHS, KUHS, NTRUHS, RGUHS, TN-MGRMU - 9 APPEARANCES, 9 OF 78 SESSIONS

SKELETON

4 **Clinical features**	Instability across four domains, with the criteria
3 **The frame**	What holds treatment before any therapy begins
3 **Therapy, drugs, crisis**	What works, what is targeted, what harms

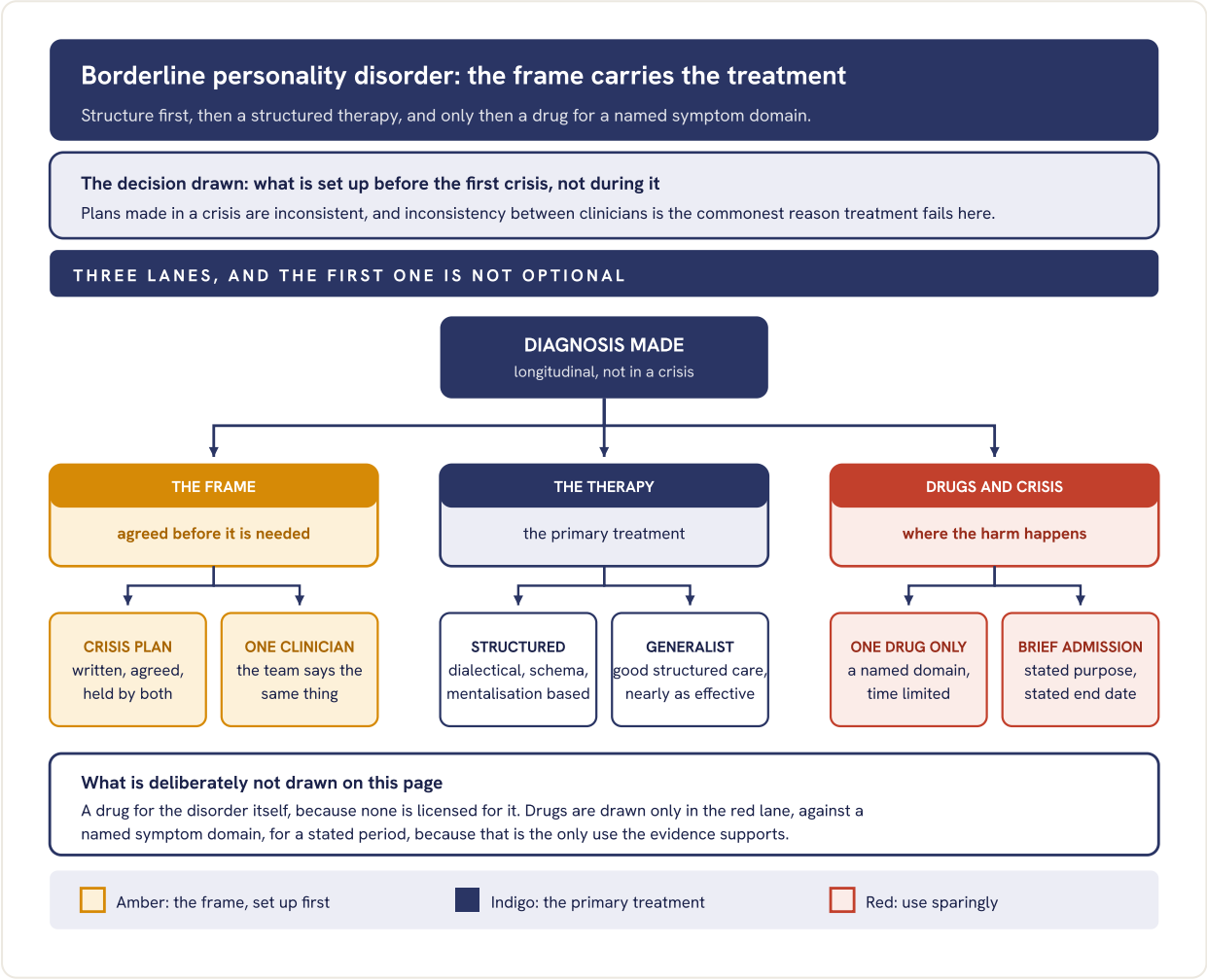

Fig 073.1 The frame drawn as a lane of its own and placed first, because it is what makes the other two safe rather than a preliminary to them.

- **Clinical features.** Instability across four domains: affect, with marked reactivity lasting hours; identity, with chronic emptiness; relationships, alternating idealisation and devaluation with frantic efforts to avoid abandonment; and behaviour, with impulsivity, intense anger, recurrent self harm and transient stress related paranoid or dissociative states.
- **Prognosis, which belongs in the answer.** Long follow up shows most patients no longer meet criteria after some years, with self harm and impulsivity remitting earliest. Social and occupational recovery lags well behind symptom remission, and saying both halves is what makes the statement useful.

WHAT EARNS THE MARKS

- **Features organised into domains,** not recited as nine criteria in order.
- **The frame written before any therapy,** the amber lane, with the crisis plan agreed in advance.
- **Psychotherapy named as the primary treatment,** with drugs confined to a named domain.
- **Prognosis given honestly,** symptom remission ahead of functional recovery.

SEE ALSO Slot II - 75 caregiver burden in borderline disorder Slot II - 76 cluster B personality disorders

II - 74

Discuss the recent changes in diagnostic classification systems related to personality disorders and substance use disorder. [5+5]

5 + 5 | PERSONALITY DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What changed and why	Categories failed on comorbidity and on reliability
3 Personality disorders	Severity and traits in one system, an alternative in the other
3 Substance use	One graded disorder, and a simplified dependence
2 Where it stands	What is actually coded in Indian practice today

AREA	THE CHANGE	WHY IT WAS MADE	STATUS
Personality, international system	**The ten types abolished; severity graded, with five trait domains**	Types overlapped heavily and were unreliable in practice	In force since 2022
Borderline pattern	Kept as a qualifier despite the abolition of types	Clinical and research continuity, and treatment selection	In force, pragmatic
Personality, American system	The ten types retained; a dimensional alternative placed alongside	Evidence supported change, the field was not ready	**Alternative model, for study**
Substance use, American system	Abuse and dependence merged into one disorder, graded by criteria met	The abuse category was unreliable and not a milder stage	In routine use
Substance use, international system	Harmful use kept; dependence simplified to three features	Simplicity and global usability, including non-specialist settings	In force since 2022
Behavioural addictions	Gambling, and then gaming, placed with the substance disorders	Shared course, comorbidity and neurobiology	Gambling routine, gaming newer

Craving was added as a criterion and legal problems removed; a graded severity replaced the two-category split in both fields at once.

STATUS: WHERE THIS SITS IN INDIAN PRACTICE

Indian records and certificates are coded on the international system, so the severity and trait model is the one that will be written down. The American categories remain the language of most teaching and of the trial literature, and the dimensional alternative there is still positioned for further study. Expect to read both, and to code one.

WHAT EARNS THE MARKS

- **The reason for the change, not only the change:** comorbidity, poor reliability, and arbitrary thresholds.
- **The borderline qualifier explained as a deliberate exception,** which shows the system is understood.
- **Merged substance disorder with graded severity,** and craving in, legal problems out.
- **The status strip,** separating what is coded here from what is taught.

SEE ALSO Slot II - 76 cluster B personality disorders Slot II - 90 dependence, abuse and harmful use

II - 75

What is caregiver burden? How to assess and manage caregiver burden of a patient with borderline personality disorder? [2+3+5]

2 + 3 + 5 | PERSONALITY DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	Objective and subjective burden, and expressed emotion
3 Assessment	Who to interview, what to ask, which instruments
3 What is specific here	Why this disorder burdens a family differently
2 Management	Educate, train, involve, relieve, and treat

THE DEFINITION

The physical, psychological, social and financial cost carried by the family of a person with illness. Objective burden is the measurable disruption to income, work, routine and health. Subjective burden is what it feels like: distress, shame, resentment and guilt. The two correlate poorly, so both are asked about.

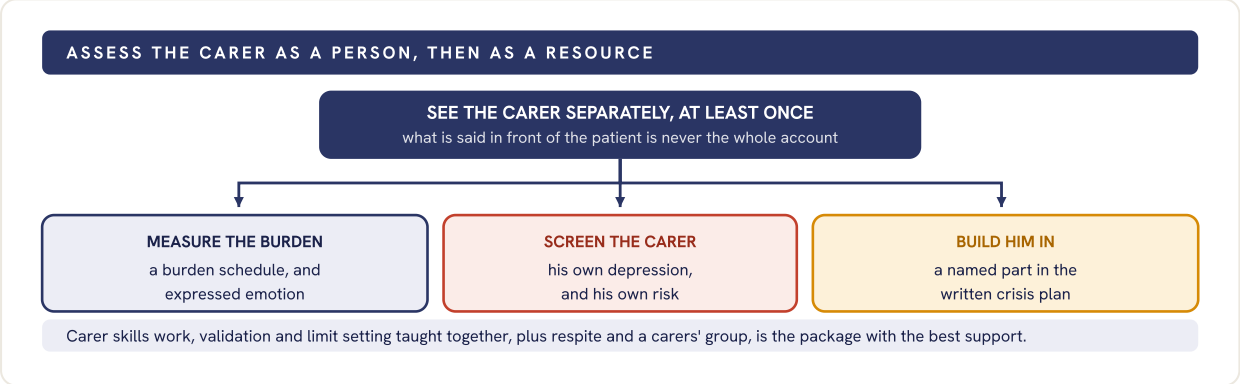

Fig 075.1 Three destinations from one interview, with the carer's own morbidity in red because it is the part most often left unasked.

- **What is specific to this disorder.** Repeated self harm and crisis calls, real fear that he will die, feeling blamed by services, and the pull to rescue that erodes the family's own limits. High expressed emotion is common and worsens the course, and it is usually exhaustion rather than hostility.
- **The guilt about limits.** Families are told to set limits and then feel responsible when distress follows. Say plainly that a limit held consistently is part of treatment, and give them the crisis plan that makes holding it safe.

WHAT EARNS THE MARKS

- **Objective and subjective burden distinguished,** with expressed emotion named alongside.
- **The carer interviewed separately,** the first box, and screened for his own illness.
- **A burden instrument named,** including one developed for Indian families.
- **The carer written into the crisis plan,** which converts assessment into management.

SEE ALSO Slot II - 73 borderline personality disorder Slot II - 77 organic personality disorders **SPRINT** Day 39, Community psychiatry and public health

II - 76

Cluster B personality disorders. [10]

10 PERSONALITY DISORDERS RGUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	The cluster	What the grouping is, and what holds it together
4	The four disorders	Each by its core motive, not by its symptom list
2	Overlap	Why they co-occur, and how they are separated
2	Where it is going	The dimensional reading of the same patients

THE CLUSTER

The dramatic, emotional and erratic group: antisocial, borderline, histrionic and narcissistic personality disorders. They share emotional dysregulation, impulsivity and difficulty sustaining relationships, and they co-occur so often that the cluster is better read as a region than as four separate boxes.

DISORDER	CORE MOTIVE	HOW IT SHOWS	WHAT SETTLES IT
Antisocial	Getting what he wants, others not counted	Deceit, aggression, recklessness, no remorse	**Conduct disorder before fifteen**
Borderline	Not being abandoned	Affective storms, self harm, idealise then devalue	**Self directed harm, abandonment fear**
Histrionic	Being attended to	Theatrical, suggestible, shifting shallow emotion	Distress at not being the centre
Narcissistic	Being admired	Grandiosity, entitlement, envy, poor empathy	**Rage on a blow to self esteem**

The motive column is what separates them under pressure. All four can look impulsive and angry in a crisis; only the reason differs.

- **Overlap and comorbidity.** Criteria sets overlap by design, and most patients meeting one set meet part of another. Substance use, depression, eating disorder and post-traumatic disorder are common, and childhood adversity is over-represented across the whole cluster.
- **Where classification is going.** The current international system does not use clusters at all. The same patients are described by severity plus trait domains, with dissociality and disinhibition carrying most of what this cluster used to name, and a borderline qualifier retained.
- **The caution worth writing.** These labels attract judgement. Antisocial personality disorder is not the same as psychopathy, most people carrying these diagnoses never offend, and all four are treatable.

WHAT EARNS THE MARKS

- **Each disorder given by its motive,** as the second column does, rather than four recited criteria lists.
- **The three amber discriminators,** which is what separates them in an examination vignette.
- **Overlap acknowledged,** since a cluster that never co-occurs would not be a cluster.
- **The dimensional reading,** which shows the classification is current.

SEE ALSO Slot II - 73 borderline personality disorder Slot II - 74 recent changes in classification SPRINT
Day 16, Personality disorders: framework, types and treatment

II - 77

Clinical features and management of organic personality disorders. [10]

10 PERSONALITY DISORDERS KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	A change after brain insult, in a previously different person
3 Clinical features	The six domains, and the syndromes by site
2 Causes and workup	What must be found before it is called a personality
3 Management	Environment first, behaviour second, drugs by target

THE DEFINITION, AND THE WORD THAT CARRIES IT

A persistent change in the habitual pattern of behaviour, following brain disease, damage or dysfunction, in someone whose premorbid personality was different. Change is the operative word: without an informant who knew him before, this diagnosis cannot honestly be made.

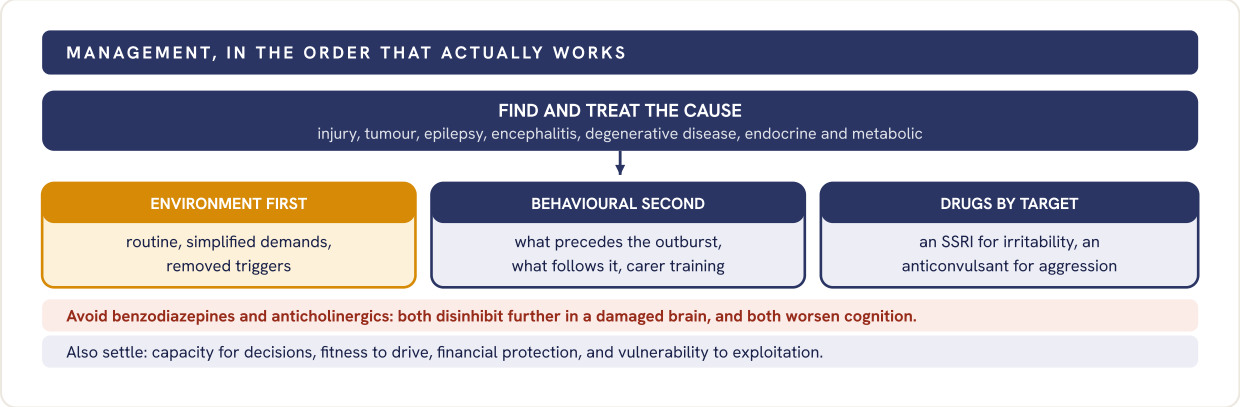

Fig 077.1 Cause, then environment, then behaviour, then drugs, with the two classes that make it worse carried as a red strip.

- **Clinical features.** Reduced persistence with goal directed activity; emotional change with lability, shallow euphoria or apathy; disinhibited expression of impulses; suspiciousness or preoccupation with a single theme; altered rate and flow of speech with circumstantiality and stickiness; and altered sexual behaviour.
- **By site.** Orbitofrontal damage disinhibits, dorsolateral damage produces apathy with executive failure, and medial frontal damage gives loss of drive. Long standing temporal lobe epilepsy is associated with a described pattern of viscosity, hyperreligiosity and circumstantial writing.

WHAT EARNS THE MARKS

- **Change from a known premorbid personality,** with the informant stated as necessary.
- **The features given as domains,** and at least one syndrome tied to its site.
- **Environment and behaviour before drugs,** the order drawn on the figure.
- **The two drug classes to avoid,** and the capacity and driving questions.

SEE ALSO Slot II - 75 caregiver burden Slot II - 76 cluster B personality disorders

II - 78

Personality disorders at the interface of psychiatry and the law: the legal use and the clinical clarification. [5+5]

5 + 5 | PERSONALITY DISORDERS | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Where the question arises	Criminal, civil and mental health law, separately
3	The legal use	What each forum wants the diagnosis to decide
3	The clarification	What the diagnosis can and cannot support
2	The clinician's role	Inform the court; do not answer its question

THE ONE DISTINCTION THE WHOLE ANSWER RESTS ON

A diagnosis is a clinical statement about a pattern of functioning. Responsibility, capacity and dangerousness are legal findings. The diagnosis is evidence towards them and is never equivalent to any of them, and most errors at this interface come from treating the two as the same statement.

FORUM	WHAT IT WANTS DECIDED	WHAT A PERSONALITY DISORDER ACTUALLY SUPPORTS
Criminal responsibility	Did he know the nature of the act, or that it was wrong	**Almost never a defence: reasoning and knowledge are preserved**
Fitness for trial	Can he understand and instruct counsel	Rarely impaired by personality alone
Mental health legislation	Is there a mental illness, and has capacity been lost	**Admission turns on capacity, not on the diagnosis**
Sentencing and risk	How dangerous is he, and for how long	A risk factor within a structured assessment, never the whole judgement
Civil matters	Testamentary capacity, contracts, custody, service fitness	Decided function by function, at a stated time

The statutory definition of mental illness turns on gross impairment of judgement, behaviour or the ability to meet ordinary demands of life, which is a functional test rather than a list of diagnoses.

- **The clinical clarification.** Diagnose longitudinally, never from one interview in custody. Separate the enduring pattern from state effects of intoxication, withdrawal, depression and detention itself. Antisocial personality disorder is not psychopathy, most people carrying these diagnoses never offend, and the disorders are treatable, which is itself relevant to disposal.
- **The ethical hazard.** A diagnosis can be used to extend detention on grounds of risk. The clinician's obligation is to state what the evidence supports and its uncertainty, in the court's language, and to leave the legal finding to the court.

WHAT EARNS THE MARKS

- **Diagnosis separated from the legal finding** in the first two lines.
- **The insanity test quoted in substance,** with the reason personality disorder rarely meets it.
- **Capacity, not diagnosis, named as the trigger** under mental health legislation.
- **The role boundary,** informing the court rather than answering it.

SEE ALSO Slot II - 76 cluster B personality disorders Slot II - 73 borderline personality disorder

II - 79

Write a short note on anxious-avoidant personality disorder. [10]

10 | PERSONALITY DISORDERS | WBUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Definition**	Wants closeness, avoids it, for one specific reason
3 **Clinical features**	The criteria, and how the life is narrowed
3 **Differential**	Sorted by what is feared and whether closeness is wanted
2 **Course and treatment**	What helps, and the order it is offered in

THE DEFINITION

A pervasive pattern of social inhibition, feelings of inadequacy and hypersensitivity to criticism, from early adulthood and across situations. He wants relationships and avoids them, restricting work and social life to stay safe from rejection, and will not become involved unless he is sure of being liked.

CONDITION	WHAT IS FEARED	WHAT SETTLES IT
Anxious avoidant personality	Being judged inadequate, then rejected	**Wants closeness, believes he is inferior**
Social anxiety disorder	Scrutiny in specific situations	Later onset, situation bound, less pervasive self view
Schizoid personality	Nothing; closeness is not wanted	**No desire for relationships at all**
Dependent personality	Being left to manage alone	Clings once attached, rather than avoiding attachment
Autism spectrum	Not fear; social communication itself differs	Lifelong, with restricted interests and sensory features

The two amber rows carry the note: wanting closeness separates this from schizoid, and a pervasive belief of inferiority separates it from situational social anxiety.

- **How the life narrows.** Jobs below ability are chosen for their low contact, promotions are declined, friendships are few and slow to form, and criticism is anticipated before it occurs. Depression, social anxiety and alcohol use are the usual comorbidities.
- **Treatment.** Cognitive behavioural work with graded social exposure and skills practice, and schema focused therapy where the inferiority belief is entrenched. A serotonergic drug treats comorbid social anxiety or depression and makes the exposure possible; it does not treat the personality pattern.
- **In the current international system.** Coded as personality disorder with prominent detachment and negative affectivity, graded by severity, rather than as a named type.

WHAT EARNS THE MARKS

- **Wanting contact while avoiding it,** stated in the definition, which is the whole of the disorder.
- **The schizoid comparison,** the second amber cell, which is the commonest examination discriminator.
- **Pervasive and early, against situational,** when separating it from social anxiety disorder.
- **Drugs placed as an enabler of therapy,** not as treatment of the personality.

SEE ALSO Slot II - 76 cluster B personality disorders Slot II - 74 recent changes in classification

AREA 11 OF 14 · P2-A14

Clinical assessment and cross-cutting nosology

6	5.5%	II-80	II-85
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 2 SINGLE 4

REVISION ORDER

EEG first, it is the one most often set. The nosology slot next, and give it time: it is three questions in one. Culture-bound syndromes after that. Functional MRI, functional impairment and the compliance slot are all short.

WHAT IS IN THIS AREA

EEG and functional MRI in psychiatry, the history of nosology with the Indian concepts and the conceptual differences between DSM-5 and ICD-11, the culture-bound syndromes with two Indian ones in detail, functional impairment and the tools that measure it, and treatment compliance set against adherence.

II - 80

Discuss briefly the role of functional magnetic resonance imaging in psychiatry. [10]

10 CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it measures	An indirect signal, and the two designs
4	Disorder findings	The replicated ones, disorder by disorder
2	Clinical uses	Where it touches a patient today
2	Limits	Why it is not a diagnostic test

WHAT IT ACTUALLY MEASURES

The blood oxygen level dependent signal, a haemodynamic proxy for neuronal activity rather than the activity itself. Good spatial resolution, poor temporal resolution. Two designs: task-based, comparing conditions, and resting state, mapping intrinsic networks such as the default mode, salience and central executive networks.

DISORDER	THE FINDING MOST OFTEN REPLICATED	WHAT IT IS TAKEN TO MEAN
Schizophrenia	Inefficient prefrontal recruitment on working memory; salience network disruption	**Dysconnectivity rather than one lesion**
Depression	Amygdala hyperreactivity to negative material with weak prefrontal regulation	A control-and-reactivity imbalance
Obsessive-compulsive	Hyperactivity in the cortico-striato-thalamo-cortical loop	**A circuit target for neurosurgery**
Post-traumatic stress	Amygdala overactivity, reduced ventromedial prefrontal control	Failure of extinction learning
Addiction	Ventral striatal cue reactivity with prefrontal control deficits	Reward learning gone wrong

- **Where it reaches a patient.** Presurgical language and memory mapping; individualised targeting of transcranial magnetic stimulation using functional connectivity; real-time neurofeedback in trials; and target identification for deep brain stimulation.
- **The limits.** Findings are group averages with overlapping distributions, so they do not diagnose an individual. Individual measures are of limited test-retest reliability, motion corrupts data, and cost and access confine it largely to research.

WHAT EARNS THE MARKS

- **Saying the signal is indirect** in the first line, which frames everything after it.
- **Task-based and resting-state separated,** with a named network.
- **Findings tied to what they are taken to mean,** which is what the grid does.
- **The clear statement that it is not a diagnostic test,** which most answers avoid saying outright.

SEE ALSO Slot II - 81 electroencephalography **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 81

Write a note on electroencephalography in psychiatry. [10]

10 CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What it records	Cortical postsynaptic potentials, and the rhythms
4 Patterns that decide	The findings that change the diagnosis
2 Other uses	Drugs, electroconvulsive therapy, sleep, research
2 Limits	What a normal and an abnormal record each fail to prove

WHAT IT RECORDS

Summated postsynaptic potentials of cortical pyramidal neurons, with excellent temporal and poor spatial resolution. Rhythms by frequency: delta below four hertz, theta four to eight, alpha eight to thirteen, beta thirteen to thirty, gamma above that. In psychiatry it is used mainly to answer one question: is this presentation driven by something other than a primary psychiatric disorder.

PATTERN	POINTS TO	WHY IT MATTERS HERE
Diffuse slowing	Delirium, and it tracks with severity	**Separates delirium from a functional psychosis**
Triphasic waves	Metabolic, classically hepatic encephalopathy	A treatable cause found
Periodic sharp wave complexes	Creutzfeldt-Jakob disease	A rapidly progressive dementia explained
Extreme delta brush	Anti-NMDA receptor encephalitis	**Immunotherapy, not antipsychotics**
Ictal or continuous discharges	Non-convulsive status epilepticus	**A psychiatric presentation that is a seizure**
Normal during an event	Dissociative non-epileptic seizures	Video recording is the standard

Amber marks the three patterns that change management the same day they are read.

- **Other uses.** Drug effects: benzodiazepines raise beta, clozapine causes dose-related slowing and spikes, lithium slows the record. Seizure adequacy and post-ictal suppression during electroconvulsive therapy. Polysomnography for sleep disorders. In research, event-related potentials such as the reduced P300 and mismatch negativity in schizophrenia.
- **The limits.** No electroencephalographic pattern establishes a primary psychiatric diagnosis. A normal record does not exclude epilepsy, and minor abnormalities occur in well people. Sleep deprivation and repeat recording raise the yield.

WHAT EARNS THE MARKS

- **The purpose stated up front:** exclusion of a non-psychiatric cause.
- **Named patterns tied to named conditions,** which is what the grid delivers.
- **Non-convulsive status epilepticus mentioned by name,** the highest-value line in the answer.
- **Both limits given,** that a normal record proves little and an abnormal one is not specific.

SEE ALSO Slot II - 80 functional imaging SPRINT Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 82

Describe the history of nosology in psychiatry. What are the Indian concepts in it? Discuss the conceptual differences between DSM-5 and ICD-11. [3+2+5]

3 + 2 + 5 | CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The lineage	Five moments, each with what it settled
2	The Indian strand	Unmada, and what survives in current practice
5	The two systems	Purpose, structure, personality, new categories

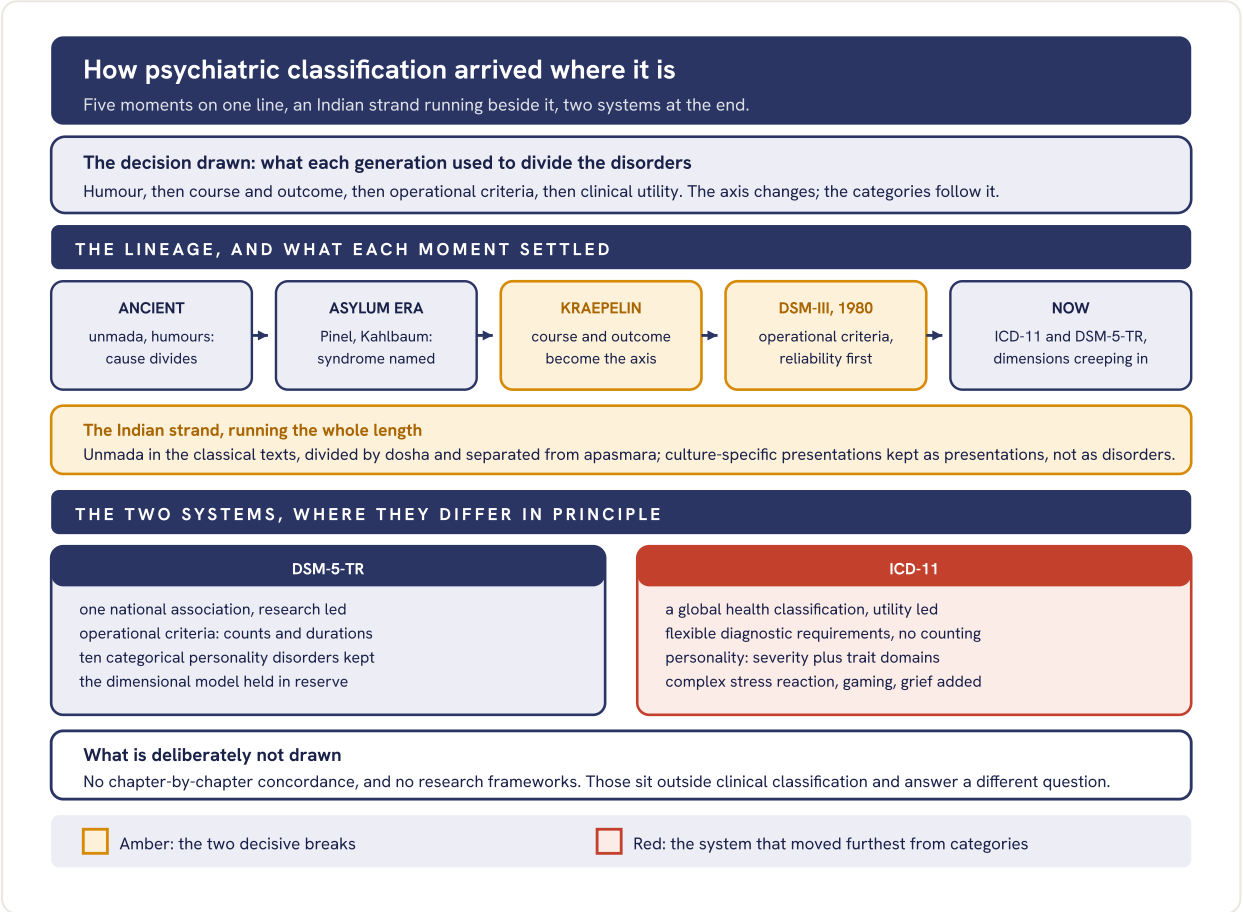

Fig 082.1 One axis, what each generation used to divide the disorders, carrying the field from cause to course to criteria to clinical utility, with the Indian strand shown as continuous rather than as an appendix.

- **The deepest difference.** Purpose. A manual written for research reliability counts symptoms; a classification written for global health service use describes a prototype and trusts the clinician. Almost every structural difference follows from that.

WHAT EARNS THE MARKS

- **The axis named at each stage,** cause, then course, then criteria, then utility.
- **Unmada given with its own internal division,** not mentioned as a passing courtesy.
- **Personality disorder used as the worked example** of the conceptual gap between the systems.

SEE ALSO Slot II - 83 culture bound syndromes **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 83

What are culture bound syndromes? Name those prevalent in India. Describe any two Indian examples in detail. [2+2+6]

2 + 2 + 6 | CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

DNB, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	And the three terms that replaced it
2 The Indian list	Named, with where each is seen
6 Two in detail	Picture, belief, differential, management

THE DEFINITION, AND WHY THE TERM CHANGED

Locality-specific patterns of distress and behaviour, recurrent within a culture, which may or may not map onto a diagnostic category. DSM-5 retired the single term and replaced it with three: the cultural syndrome, the cultural idiom of distress and the cultural explanation of cause. ICD-11 treats them as culturally specific presentations of recognised disorders. The Cultural Formulation Interview is the tool for eliciting them.

PRESENTATION	THE CORE COMPLAINT	WHERE REPORTED
Dhat syndrome	Fatigue and weakness attributed to loss of semen in urine	**Across India, chiefly young men**
Possession states	Identity taken over by a spirit or deity, time limited	**Nationwide, often at shrines**
Koro-like episodes	Fear of genital retraction, sometimes in local epidemics	Eastern and north-eastern India
Suchi-bai	Overwhelming preoccupation with ritual pollution and washing	Eastern India, chiefly widows
Ascetic syndrome	Withdrawal into religious austerity with social renunciation	Young men, described in India

- **Dhat syndrome.** A young man, often unmarried and from a rural background, reports whitish discharge in urine believed to be semen, with weakness, poor concentration, anxiety and sexual dysfunction. The belief rests on the classical idea of semen as the most refined bodily essence. Differential: depressive and anxiety disorders, urinary infection, sexual dysfunction. Management: examine, then explain the physiology, correct the myth, treat the comorbid disorder, and stop the cycle of tonics and repeat tests.
- **Possession states.** Altered identity and consciousness with a changed voice, most often in young women with little social power, usually brief and socially sanctioned. Separate sanctioned trance from dissociative trance disorder, from psychosis and from temporal lobe seizures. Engage the family and the faith healer rather than confronting them, and treat what is treatable.

WHAT EARNS THE MARKS

- **The three replacement terms named,** which shows the current position rather than the older one.
- **A list with locations attached,** not just names.
- **Each detailed example carried through four beats:** picture, belief, differential, management.
- **Collaboration rather than confrontation with traditional healers,** which is the practice mark.

SEE ALSO Slot II - 82 nosology and the Indian strand **SPRINT**

Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 84

Define functional impairment. What are its manifestations? What scales and assessment tools are used? [2+2+6]

2 + 2 + 6 | CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	The three-level framework, named properly
2 Manifestations	Domain by domain, not a list of symptoms
4 The instruments	Generic, disorder specific, and the Indian statutory one
2 Choosing one	What the answer depends on

THE DEFINITION

Reduction in the ability to carry out activities and fulfil life roles expected for a person's age, sex and social context, arising from a health condition. The international classification of functioning separates three levels: impairment of body function or structure, activity limitation, and participation restriction, each modified by environmental and personal context. Impairment is not the same as disability, and neither equals symptom severity.

INSTRUMENT	WHAT IT MEASURES	WHERE IT IS USED
WHO Disability Assessment Schedule	Six domains: cognition, mobility, self-care, getting along, life activities, participation	**The generic standard, all diagnoses**
Indian Disability Evaluation and Assessment Scale	Self-care, interpersonal activity, communication, work, plus duration of illness	**Statutory certification in India**
Sheehan Disability Scale	Three items: work, social life, family life	Brief, common in trials
Personal and Social Performance Scale	Four areas, clinician rated	Schizophrenia
Functioning Assessment Short Test	Six domains including autonomy and cognition	Bipolar disorder
Global Assessment of Functioning	A single number for symptoms and function together	Withdrawn for conflating the two

Amber marks the two that must appear: the generic instrument the classification recommends, and the one Indian certification actually runs on.

- **Manifestations by domain.** Self-care and household tasks; work or study, through both absence and reduced output while present; family and social roles; leisure and community participation; independent living and finances; and the burden that falls on the carer.

WHAT EARNS THE MARKS

- **The three-level framework named,** since it is what separates impairment from disability.
- **Manifestations given as domains,** which is what a functional assessment actually asks about.
- **The Indian statutory instrument named** and linked to certification under disability law.
- **A reason for withdrawing the global scale,** which shows why function is rated separately from symptoms.

SEE ALSO Slot II - 85 treatment compliance SPRINT Day 19, Psychotherapies, clinical assessment, MSE and nosology

II - 85

What is treatment compliance and why does it matter? How does it differ from adherence? Discuss the methods used to improve compliance in psychiatry. [1+1+2+6]

1 + 1 + 2 + 6 | CLINICAL ASSESSMENT AND CROSS-CUTTING NOSOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The three terms	What each assumes about who decides
2	Why it matters	Relapse, readmission, false treatment resistance
5	Improving it	Patient, treatment, service, each with real measures
1	Measuring it	How you would know whether any of it worked

TERM	WHAT IT DESCRIBES	WHO SETS THE PLAN	WHAT SETTLES THE DIFFERENCE
Compliance	Behaviour matching the prescriber's instruction	The clinician	**The patient is passive in the word itself**
Adherence	Behaviour matching a recommendation the patient agreed to	Both, by agreement	**Autonomy is preserved; the preferred term**
Concordance	The quality of the consultation, not the behaviour after it	A negotiation of beliefs	It describes the meeting, not the pills
Persistence	How long treatment is continued at all	Neither, it is a duration	A different measure, often confused

Amber cells carry the distinction the question is asking for. The fourth row is included because persistence is routinely reported as if it were adherence.

- **Why it matters.** Partial or complete non-adherence is the commonest identifiable cause of relapse in schizophrenia and bipolar disorder, and it drives readmission, suicide risk, and the mislabelling of an untried treatment as treatment resistant.
- **Improving it, at three levels.** Patient: psychoeducation with the family, motivational interviewing, working with the person's own explanatory model. Treatment: simplify to once daily, actively treat side effects, offer a long-acting injectable, monitor plasma levels where available. Service: continuity with one clinician, shared decision making, reminders, assertive follow up, and involving the family who in practice supervise medication at home.

PITFALL

Treating non-adherence as a fault in the patient. It is usually a modifiable problem in the treatment or the service, and side effects are the commonest single reason. Ask about them before assuming anything else.

WHAT EARNS THE MARKS

- **The terms distinguished on who sets the plan,** which is the real difference and not a matter of style.
- **Measures grouped at three levels,** rather than offered as one undifferentiated list.
- **The long-acting injectable named,** with family supervision noted as the Indian reality.
- **A method of measurement,** self-report, pill count, refill records or plasma level, which most answers never supply.

SEE ALSO Slot II - 84 functional impairment **SPRINT** Day 19, Psychotherapies, clinical assessment, MSE and nosology

AREA 12 OF 14 · P2-A06

Substance use: general concepts and alcohol

5	4.5%	II-86	II-90
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 2 SINGLE 3

REVISION ORDER

Delirium tremens first, it is short and it is set often. The neuropsychiatric slot next, it is the longest here. Early onset dependence and relapse prevention after that. The definitional slot last: it rewards precision rather than length.

WHAT IS IN THIS AREA

The neuropsychiatric complications of chronic alcohol use with their treatment and a locality reduction plan, delirium tremens, early onset alcohol dependence syndrome, non-pharmacological relapse prevention, and the definitional slot that separates use, abuse, harmful use and dependence and asks for the role of dopamine.

II - 86

Discuss in detail various neuropsychiatric disorders associated with chronic alcohol use/dependence. Write, in brief, about their treatment. How can you plan to reduce alcohol use in your area? [3+4+3]

3 + 4 + 3 | SUBSTANCE USE: GENERAL CONCEPTS AND ALCOHOL

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The syndromes	Deficiency, toxic and withdrawal groups, each named
4	Treatment	Thiamine before glucose, then the group specific step
3	The area plan	Screen, brief intervention, refer, district programme

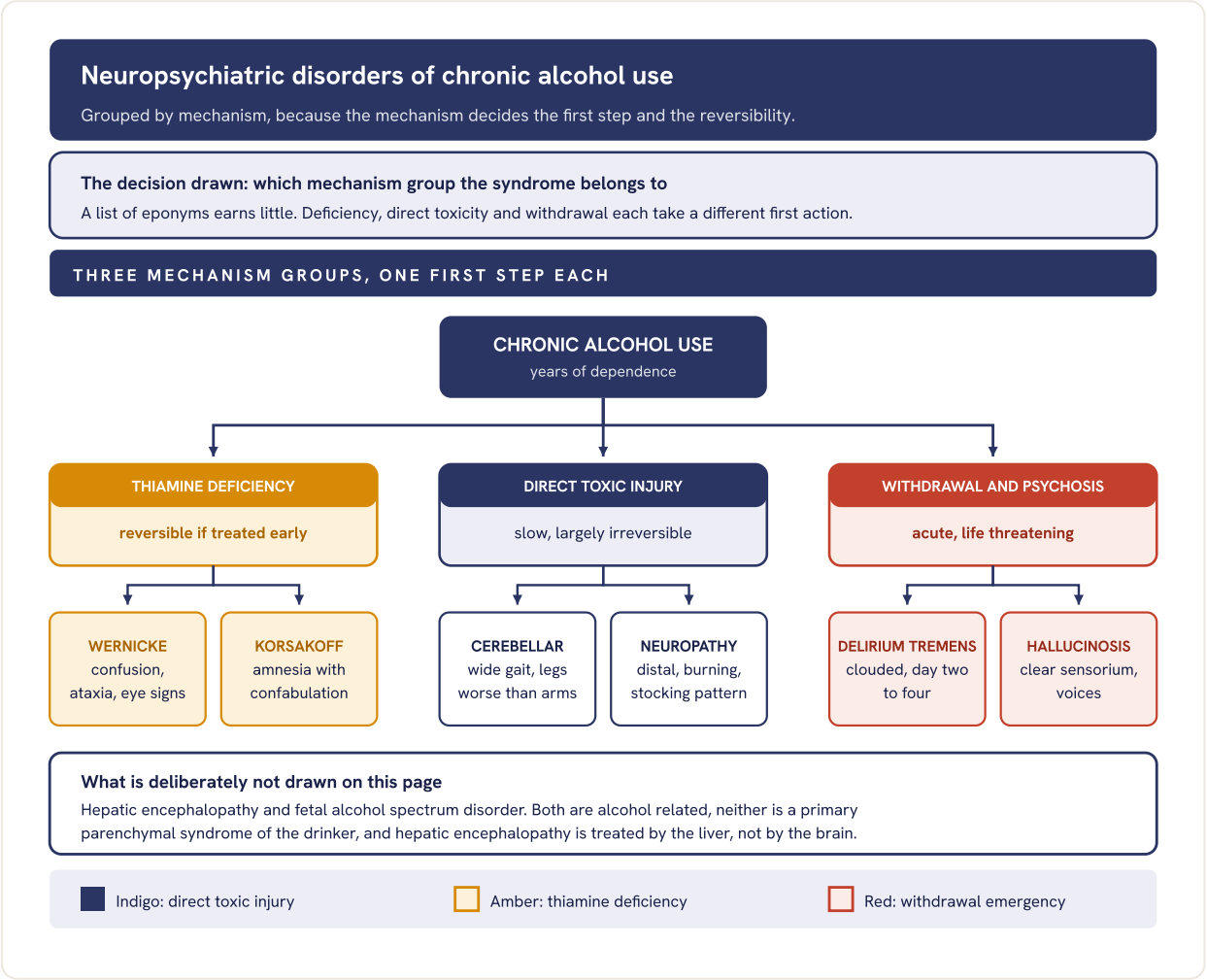

Fig 086.1 The syndromes sorted by mechanism, with the two reversible amber leaves kept together because they share one treatment.

- **Treatment.** Parenteral thiamine before any glucose load; 500 mg intravenously thrice daily for three days in suspected Wernicke encephalopathy, then oral. Benzodiazepine cover for withdrawal. Once dry, naltrexone, acamprosate or disulfiram with supervision.
- **The area plan.** Map the burden, screen every general clinic attender with AUDIT, deliver brief intervention on the spot, refer to the district de-addiction service under the national drug demand reduction plan, and link recovery to mutual help groups.

PITFALL

Korsakoff is not dementia. Selective anterograde amnesia with confabulation and intact procedural learning follows a thiamine emergency; global decline over years is alcohol related dementia, and thiamine will not reverse it.

WHAT EARNS THE MARKS

- **The three mechanism groups on the plate, named as groups.** Grouping is what turns a list of eponyms into an answer.
- **Thiamine written before glucose,** ahead of every other treatment line.
- **Korsakoff separated from alcohol related dementia,** as the pitfall box does.
- **An area plan built on screening and referral,** with a named national programme, not on exhortation.

SEE ALSO Slot II - 87 delirium tremens Slot II - 89 relapse prevention without drugs **SPRINT** Day 13, Substance use disorders

II - 87

Delirium tremens. [10]

10 SUBSTANCE USE: GENERAL CONCEPTS AND ALCOHOL

KUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	The withdrawal delirium, and when in the week it starts
2 Where it sits	The withdrawal timeline, so the diagnosis is dated
3 Clinical picture	Clouding, hallucinations, tremor, autonomic storm
3 Management	Benzodiazepine, thiamine, fluids, treat the precipitant

THE DEFINITION

An acute, transient, fluctuating organic brain syndrome occurring on withdrawal of alcohol in a physically dependent drinker, marked by clouding of consciousness, disorientation, vivid perceptual disturbance, coarse tremor and autonomic overactivity. It is a medical emergency, not a psychiatric one.

HOURS DRY	WHAT APPEARS	CONSCIOUSNESS	WHAT SETTLES IT
6 to 12	Tremor, sweating, nausea, anxiety	Clear	Simple withdrawal
12 to 24	Alcoholic hallucinosis, usually voices	Clear	**Clear sensorium excludes delirium**
24 to 48	Generalised withdrawal seizures	Post-ictal only	Fits precede, they do not define
48 to 96	Delirium tremens	Clouded and fluctuating	**Clouding plus autonomic storm**

Dating the picture from the last drink is the cheapest mark on this question, and the one most often left out.

- **Clinical picture.** Disorientation to time and place, vivid visual hallucinations that are often small animals, tactile misperception, marked tremor, fever, tachycardia, hypertension and profuse sweating, worse at night, with fear and psychomotor overactivity.
- **Management.** Monitored medical bed. Chlordiazepoxide by symptom triggered regimen, or lorazepam 2 to 4 mg when liver function is poor. Parenteral thiamine before glucose. Correct fluids, potassium and magnesium. Add haloperidol only under benzodiazepine cover.
- **Look for the precipitant.** Infection, head injury, pancreatitis, gastrointestinal bleed or hepatic decompensation is present in a large minority, and untreated it is what kills.

WHAT EARNS THE MARKS

- **The 48 to 96 hour window, stated as a number.** The table dates it; prose usually does not.
- **Clouded consciousness named as the discriminator** from alcoholic hallucinosis in the row above it.
- **Benzodiazepine as the treatment and antipsychotic as an adjunct,** never the reverse.
- **The search for a precipitant,** written as part of management rather than as an afterthought.

SEE ALSO Slot II - 86 neuropsychiatric disorders of alcohol Slot II - 88 early onset alcohol dependence SPRINT Day 13, Substance use disorders

II - 88

Early onset alcohol dependence syndrome. [10]

10 SUBSTANCE USE: GENERAL CONCEPTS AND ALCOHOL

RGUHS, WBUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	Dependence, and what the word early adds to it
3 The typologies	Cloninger and Babor, and the axis each divides on
3 Clinical picture	Temperament, family loading, comorbidity, course
2 Why it matters	What treatment changes because onset was early

THE DEFINITION

Alcohol dependence syndrome by the usual criteria, with onset of dependent drinking before about twenty five years. Age at onset is used as a marker for a distinct subtype rather than as a simple description, because it tracks heritability, temperament and treatment response.

DOMAIN	EARLY ONSET	LATE ONSET	WHAT SETTLES IT
Typology	Cloninger type II, Babor type B	Cloninger type I, Babor type A	Age at first dependence
Temperament	High novelty seeking, low harm avoidance	High harm avoidance, high reward dependence	Novelty seeking is the pivot
Heredity	Male limited, strong paternal loading	Milieu dependent, both sexes	Paternal alcohol use and criminality
Drinking	Spontaneous seeking, inability to abstain	Binges with loss of control, then guilt	Abstain versus control failure
Comorbidity	Antisocial traits, other drugs, violence	Anxiety and depression	Externalising versus internalising
Course	Faster progression, poorer outcome	Slower, better outcome	Prognosis, not severity alone

Amber cells are the discriminators. Age alone is descriptive; temperament and family loading are what make the subtype a claim.

- **Why it matters.** Early onset predicts poorer response to insight oriented work and a better response to structured coping skills training. Ondansetron has trial support in early onset drinkers specifically, while naltrexone suits the later onset, family history positive pattern.
- **The honest limit.** These are dimensional tendencies, not categories. A sizeable proportion of drinkers fit neither type cleanly, and no typology is diagnostic in either classificatory system.

WHAT EARNS THE MARKS

- **Both typologies named,** Cloninger and Babor, with the axis each divides on stated before the grid.
- **Temperament and paternal loading marked as the discriminators,** not age by itself.
- **A treatment consequence,** which is what turns a classification into an answer.
- **The honest limit line.** Naming it reads as command of the literature.

SEE ALSO Slot II - 87 delirium tremens Slot II - 90 dependence, abuse and harmful use SPRINT

Day 13, Substance use disorders

II - 89

Non pharmacological methods in the relapse prevention of alcohol use disorder. [10]

10 | ASKED AT 7 MARKS | SUBSTANCE USE: GENERAL CONCEPTS AND ALCOHOL

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Why non drug	Relapse is situational; medication does not teach coping
2	Engage first	Stage of change, then motivational enhancement
4	The three lanes	Individual, family and couple, community
2	Evidence and aftercare	What the trials showed, and the follow-up rule

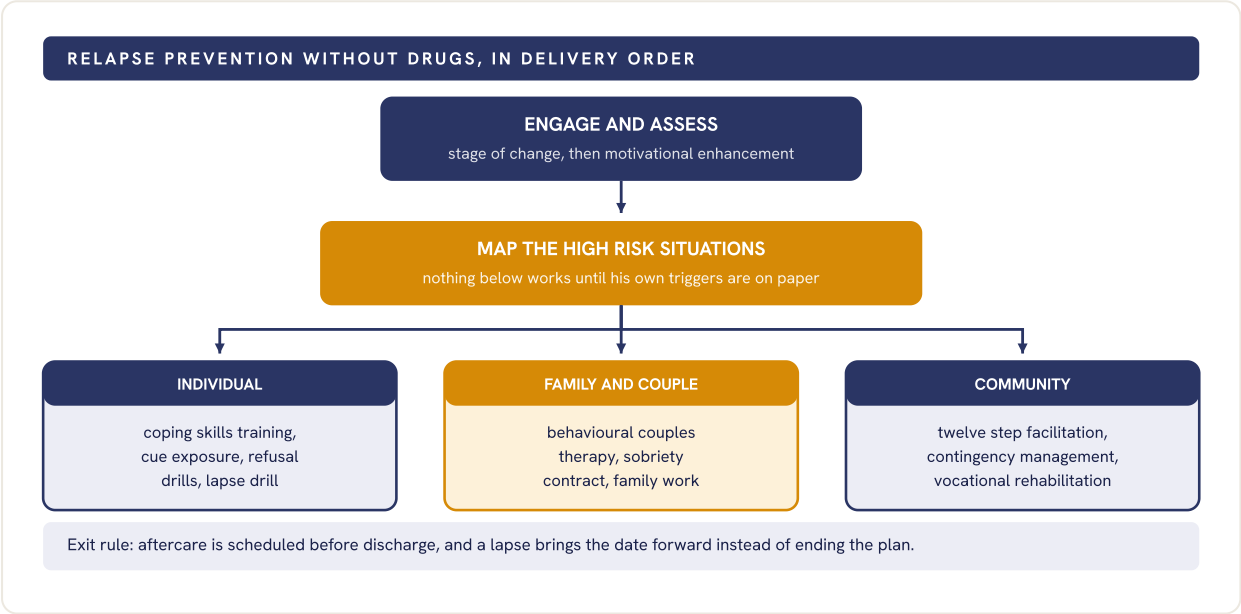

Fig 089.1 Engagement first, the trigger map as the gate, then three lanes that run together rather than in sequence.

- **The cognitive behavioural core.** Identify high risk situations, train coping responses, rehearse drink refusal, correct outcome expectancies, and plan for the seemingly irrelevant decisions that walk a man past his old bar.
- **The abstinence violation effect.** A lapse read as proof of failure becomes a relapse. Reframing it as a high risk situation that was mishandled is itself a technique.
- **Match the method to the stage.** For an ambivalent drinker motivational enhancement is the intervention, not a warm up; coping skills training delivered in precontemplation wastes both.

WHAT EARNS THE MARKS

- **The trigger map placed before the lanes,** as the amber gate on the flowchart.
- **Three lanes kept apart,** individual, family and community, rather than one list of therapy names.
- **The abstinence violation effect, named.** It is the concept the question is testing.
- **Aftercare scheduled before discharge,** the exit rule on the foot of the figure.

SEE ALSO Slot II - 86 neuropsychiatric disorders of alcohol Slot II - 13 motivational enhancement therapy **SPRINT** Day 13, Substance use disorders

II - 90

Define dependence of psychoactive substance. How is it different from use, abuse and harmful use? Discuss the role of dopamine in context of psychoactive substance use. [3+3+4]

3 + 3 + 4 | SUBSTANCE USE: GENERAL CONCEPTS AND ALCOHOL

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The definition	The dependence syndrome, quoted, with its time rule
3	The grid	Use, harmful use and dependence, domain by domain
2	The circuit	Where dopamine sits and what all these drugs share
2	What dopamine does	Wanting, not liking, and the shift to habit

THE DEFINITION

A cluster of physiological, behavioural and cognitive phenomena in which use of a substance takes on a much higher priority than behaviours that once had greater value. Three of six features, together, at some time in the previous twelve months.

DOMAIN	HARMFUL USE	DEPENDENCE	WHAT SETTLES IT
Core question	Has damage occurred	Has control been lost	Loss of control, not quantity
Physiology	Tolerance and withdrawal not required	Tolerance and withdrawal usual	Relief drinking or dosing
Salience	Use is one activity among several	Use displaces work and family	Priority over former interests
Craving	Not part of the definition	Strong desire or compulsion	The reported compulsion
Persistence	May stop once harm is shown	Continues with the harm known	Use despite known harm

Use is consumption without damage or loss of control. Abuse is the older American term and maps closely onto harmful use.

- **The circuit.** Every drug of dependence raises dopamine in the nucleus accumbens, directly as cocaine and amphetamine do, or by disinhibiting ventral tegmental neurons as opioids, alcohol and nicotine do.
- **What dopamine actually does.** It is not the pleasure signal. Phasic dopamine encodes reward prediction error and attaches incentive salience to cues, so wanting grows and outlasts liking.
- **The shift.** With repetition control migrates from ventral to dorsal striatum and use becomes habit. Striatal D2 availability falls, which is why abstinence alone does not restore free choice.

WHAT EARNS THE MARKS

- **The definition written out with the three of six rule** and the twelve month window.
- **Loss of control named as the discriminator,** as the first amber cell does, rather than a list of ten differences.
- **Dopamine described as wanting rather than pleasure,** and the ventral to dorsal shift that links it to relapse.

SEE ALSO Slot II - 88 early onset alcohol dependence Slot II - 11 neurobiology of opioid use disorder SPRINT Day 13, Substance use disorders

AREA 13 OF 14 · P2-A03

Other psychoses and catatonia

5	4.3%	II-91	II-95
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 RECURRENT 1 SINGLE 3

REVISION ORDER

The two delusional disorder slots together, because they are the same answer asked at two widths, and the shared-delusion slot sits directly on top of them. Catatonia next, and it is the anchor here. Schizoaffective disorder last, where the nosology is the part that earns the marks.

WHAT IS IN THIS AREA

Catatonia, schizoaffective disorder and its nosological status, delusional disorder in two slots, and shared delusional disorder. Five slots, and four of them are one family.

II - 91

What is catatonia? Briefly discuss etiology, clinical features and management of catatonia. [10]

10 OTHER PSYCHOSES AND CATATONIA

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

SKELETON

2	What it is	A syndrome across diagnoses, not a subtype of schizophrenia
3	Aetiology	Psychiatric, neurological, medical and drug related causes
3	Clinical features	Three presentations, the signs, and the rating scale
2	Management	Lorazepam, then ECT, and stopping what worsens it

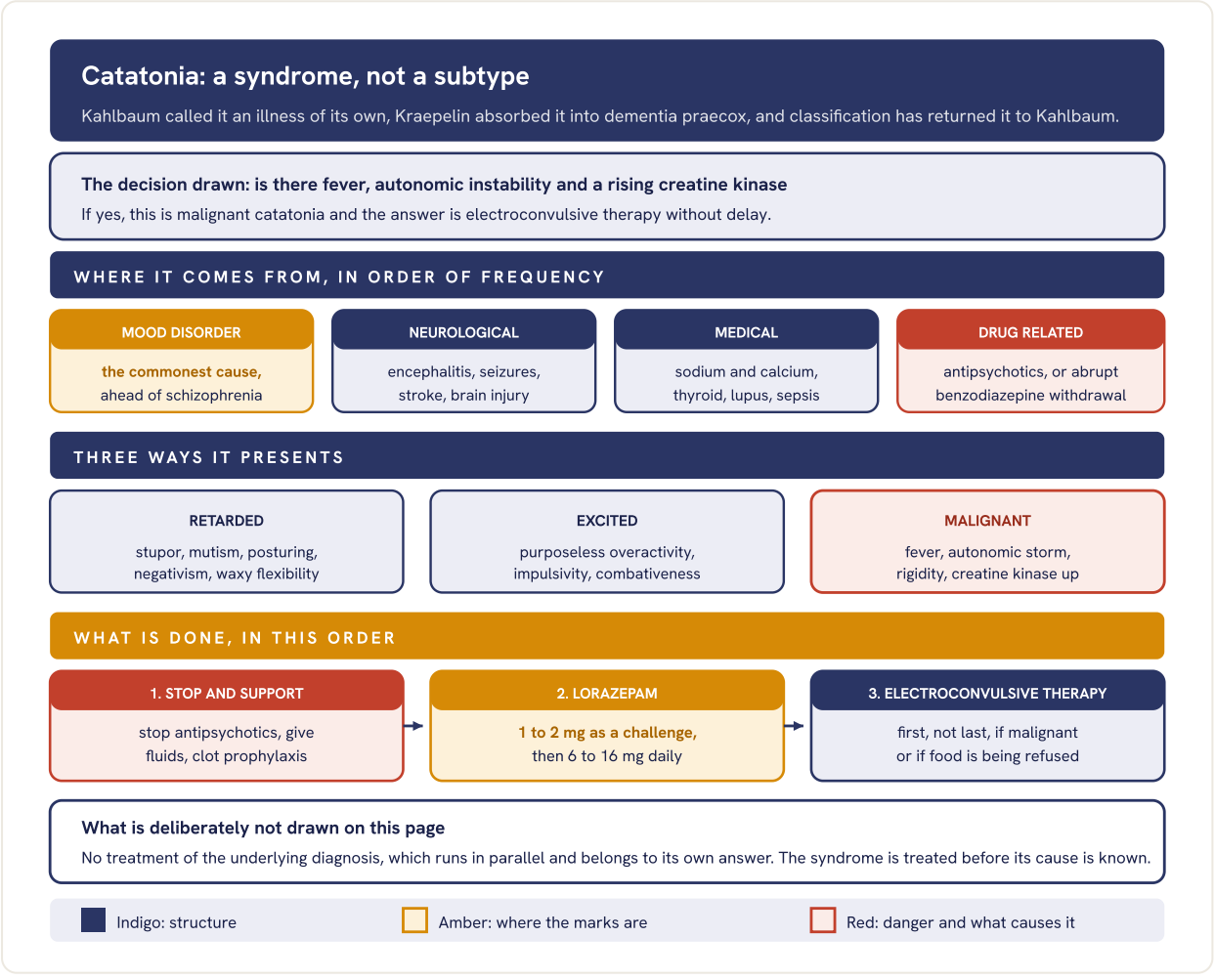

Fig 091.1 Catatonia as cause, presentation and treatment, with the malignant form separated because it changes the order.

- **Recognising it.** Fourteen signs make the bedside screen and twenty three the full scale: stupor, mutism, negativism, posturing, catalepsy, waxy flexibility, staring, grimacing, echolalia, echopraxia, stereotypy, automatic obedience, ambitendency, refusal of food.
- **The challenge test.** One to two milligrams of lorazepam given intravenously or intramuscularly, with the scale rated before and after. Improvement within ten minutes is both diagnostic and the start of treatment, and most non-malignant cases respond.

- **Investigations.** Creatine kinase, electrolytes, calcium, renal and liver function, thyroid, an electroencephalogram to exclude non-convulsive status, and an antibody screen if onset was rapid.

PITFALL

Giving an antipsychotic to a mute, immobile patient assumed to be psychotic. It can convert simple catatonia into the malignant form. The antipsychotic is stopped, not started, until the catatonia has cleared.

WHAT EARNS THE MARKS

- **Catatonia named as a transdiagnostic syndrome,** with mood disorder given as the commonest cause.
- **Causes grouped into four families,** not listed as one run of conditions.
- **The lorazepam challenge described as diagnostic and therapeutic at once.**
- **Electroconvulsive therapy placed first in the malignant form.**

SEE ALSO Slot II - 46 neuroleptic malignant syndrome Slot II - 92 schizoaffective disorder

II - 92

Discuss the nosological status, differential diagnosis, and biological and psychological treatment of schizoaffective disorder. [2+2+3+3]

2 + 2 + 3 + 3 | OTHER PSYCHOSES AND CATATONIA | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Nosological status**	Where it sits, and why the category stays contested
2	**The rules that decide it**	The two week window, and the majority of illness rule
3	**Biological treatment**	Antipsychotic backbone, then the mood agent, then ECT
3	**Psychological treatment**	Psychoeducation, relapse signature, family and work

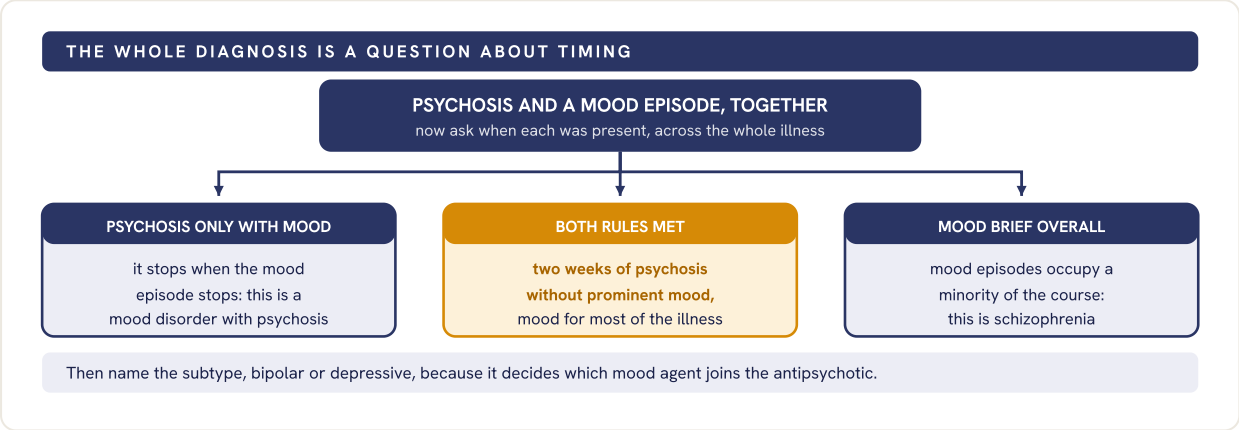

Fig 092.1 The differential drawn as a timing question, because both separating rules are rules about when.

- **Nosological status.** Described by Kasanin, it sits between the halves of the Kraepelinian dichotomy and is standing evidence against a clean split. Reliability and temporal stability are both low.
- **Where the systems differ.** One makes it a lifetime diagnosis, needing the two week window and mood for the majority of the illness. The other keeps it cross-sectional, a label for one episode.
- **Biological treatment.** An antipsychotic is the backbone, paliperidone the one agent with a specific licence. Lithium for the bipolar type, an antidepressant under cover for the depressive type, ECT when severe or refractory.
- **Psychological treatment.** Psychoeducation for a two-syndrome illness, cognitive therapy for psychosis, a written relapse signature, family work and vocational support.

PITFALL

Diagnosing it from one admission. Both rules are longitudinal, so the diagnosis needs a life chart; made cross-sectionally it is usually wrong.

WHAT EARNS THE MARKS

- **Both timing rules stated,** since together they are the whole differential.
- **The disagreement between classification systems named.**
- **Subtype used to select the mood agent,** and psychological treatment answered separately, since it carries its own three marks.

SEE ALSO Slot II - 91 catatonia Slot II - 63 bipolar depression

II - 93

Discuss diagnosis and treatment of delusional disorder. [10]

10 | OTHER PSYCHOSES AND CATATONIA | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Diagnosis**	The criteria, the duration, and what must be absent
2	**Assessment**	Corroboration, is the belief actually false, organic screen
3	**Treatment**	Engagement first, then the drug, then adapted therapy
2	**Risk and course**	Who is dangerous, how long to treat, what relapse looks like

- **Diagnosis.** One or more delusions for at least a month, with functioning outside the theme largely preserved. Hallucinations, if any, are not prominent and stay tied to the theme, and criterion A for schizophrenia is never met.
- **Assessment.** Corroborative history is not optional, because the belief is plausible and jealousy is sometimes true. Late onset demands an organic screen: dementia, stroke, substance use, untreated deafness or blindness.

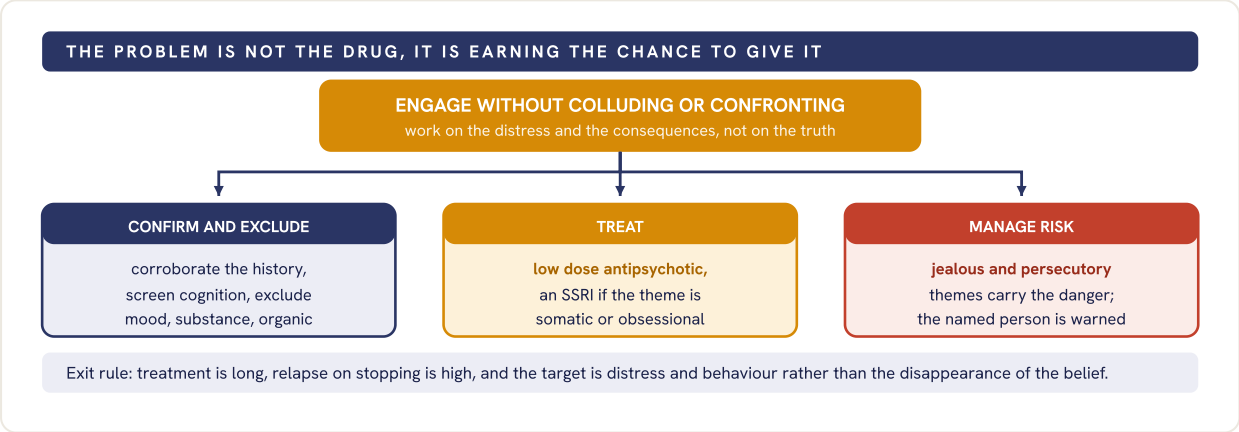

Fig 093.1 Management arranged around the real obstacle, engagement, with the three arms that follow it.

- **Drug treatment, honestly stated.** The evidence is thin, largely open studies, with roughly half improving. Second generation agents at low dose are the practical first choice; pimozide is not superior and carries cardiac risk.
- **Psychological treatment.** Cognitive therapy adapted for psychosis, starting with befriending and moving to reality testing that is offered, never imposed. Direct challenge hardens the belief.

PITFALL

Answering the truth question. Arguing that the belief is false, or agreeing it is true, both end treatment. The distress is real and worth treating either way.

WHAT EARNS THE MARKS

- **Duration and the preserved functioning criterion,** which is what separates this from schizophrenia.
- **Corroborative history and an organic screen in late onset.**
- **Engagement written as the first treatment step,** before any drug, with risk handled as its own task.

SEE ALSO Slot II - 94 the types of delusional disorder Slot II - 95 shared delusional disorder **SPRINT**

Day 10, Other psychotic disorders, delusional syndromes and catatonia

II - 94

Discuss the various types, clinical features, and management of delusional disorders. [10]

10 | ASKED AT 15 MARKS | OTHER PSYCHOSES AND CATATONIA

TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	The types	Five themes, each with the eponym it is known by
3	Clinical features	Encapsulated, systematised, personality preserved around it
3	Management by theme	Risk decides the plan more than the drug choice does
1	Where they present	Rarely to psychiatry, and never by self-referral

TYPE	THE BELIEF	EPONYM AND WHERE IT TURNS UP
Persecutory	Conspired against, followed, poisoned	Commonest; querulous variant reaches the courts
Jealous	The partner is being unfaithful	**Othello syndrome; carries the highest violence risk**
Erotomanic	A person of higher status is secretly in love	De Clerambault syndrome; presents by stalking
Somatic	Infestation, foul odour, a misshapen part	Ekbom syndrome; reaches dermatology first
Grandiose	A special talent, discovery or mission	Exclude mania and an organic cause first

Capgras and Fregoli are misidentification syndromes and usually sit inside another illness rather than here.

- **Clinical features.** The delusion is encapsulated and systematised, elaborated over years, and non-bizarre enough to be plausible. Around it, affect, speech, volition and personality stay intact, which is why work and family life can continue for a long time.
- **The give-away signs.** The specimen brought in a container in delusional infestation, the file of correspondence in the querulous type, and the partner interrogated about timings in the jealous type.

Fig 094.1 Management sorted by who the belief endangers, which is the axis that changes the plan.

WHAT EARNS THE MARKS

- **All five themes with their eponyms.** The stem asks for types first, so the grid is the answer to a third of the paper mark.
- **Encapsulation named as the defining feature,** with function preserved around it.
- **Management differentiated by theme,** rather than one antipsychotic for all five.

SEE ALSO Slot II - 93 diagnosis and treatment Slot II - 95 shared delusional disorder **SPRINT**

Day 10, Other psychotic disorders, delusional syndromes and catatonia

II - 95

Describe the clinical features and management of shared delusional disorder. [10]

10 | OTHER PSYCHOSES AND CATATONIA | KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	The pair, their roles, and where classification puts it now
3 Preconditions and forms	Closeness and isolation, and the four described forms
2 Clinical features	Shared persecutory content, an otherwise well recipient
3 Management	Separate, assess apart, treat the inducer, safeguard

- **What it is.** A delusion held by one person, the inducer, transmitted to a closely attached second person, the recipient. Described as folie a deux by Lasegue and Falret. Neither current system keeps it as a separate category: the recipient is now coded as having a delusional disorder in their own right.
- **Preconditions and forms.** A long, close, usually blood or marital tie, isolation from anyone who would contradict the belief, and a dominant partner who is genuinely ill. Four forms: imposed, where separation cures the recipient; simultaneous, both predisposed; communicated, the belief kept after separation; and induced.
- **Clinical features.** The content is usually persecutory and plausible, so it survives casual scrutiny. The recipient has few other psychotic features and intact premorbid function, so the pair reaches services late, usually through neglect.

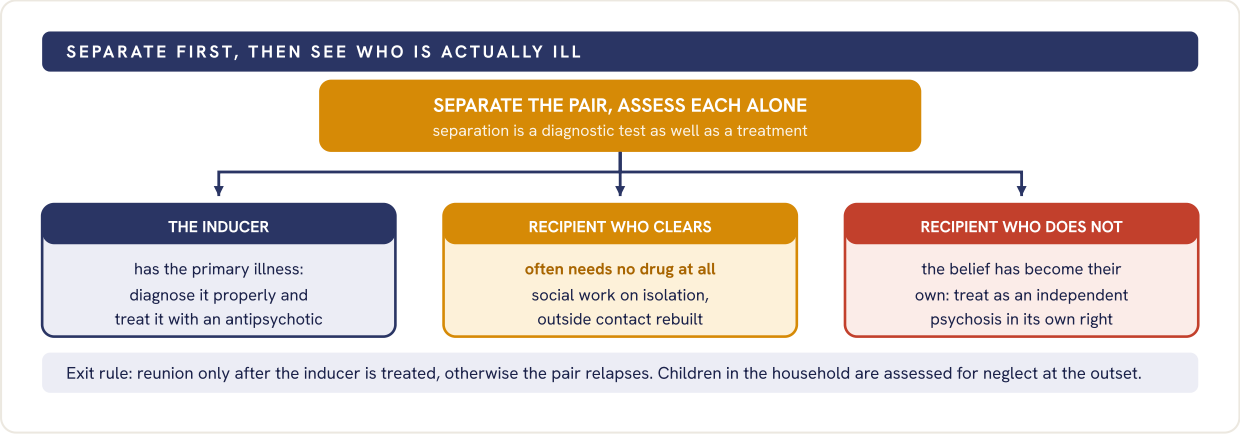

Fig 095.1 Separation used as both test and treatment, with the three outcomes it produces.

PITFALL

Admitting and medicating both. The recipient frequently needs neither, and treating them as equally ill misses that the real intervention is separation, safeguarding of any children, and proper treatment of the inducer.

WHAT EARNS THE MARKS

- **Inducer and recipient named as distinct roles,** with the current classification position said plainly.
- **The four described forms,** since they are what the outcome after separation depends on.
- **Separation written as the first management step,** and as a diagnostic test.
- **Safeguarding of children and the reunion condition,** which most answers never reach.

SEE ALSO Slot II - 93 diagnosis and treatment Slot II - 94 the types of delusional disorder

AREA 14 OF 14 · P2-A12

Suicide, self-harm and impulse control

5	4.0%	II-96	II-100
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 5

REVISION ORDER

Build one suicide answer that covers assessment, biology and prevention, and three of the five slots open. Then the policy limb, which only the last slot asks for. Aggression and behavioural addiction are independent and both are short.

WHAT IS IN THIS AREA

Suicide in three slots: the enquiry with its genetic disposition, the biological aspects with prevention and post-attempt management, and the biopsychosocial slot that asks for the Indian prevention strategy by name. Aggression and behavioural addiction carry the impulse-control limb. Every slot in this area was seen once in the record, which is the whole of what its chips claim.

II - 96

Describe the suicidal enquiry and the diagnostic category to which suicide belongs. Write about genetic disposition in suicide. [4+3+3]

4 + 3 + 3 | SUICIDE, SELF-HARM AND IMPULSE CONTROL

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	The enquiry	Graded questions, collateral, and what gets written down
3	Where it sits	Neither system carries suicide as a mental disorder
3	Genetics	Family, twin, adoption, molecular, endophenotype

THE ENQUIRY, IN THE ORDER IT IS ASKED

Ask unhurriedly and in plain words, moving from the general to the specific: distress and hopelessness, then wish to be dead, then thoughts of ending life, then plan, method, access, preparatory acts, intent and timing, then deterrents and reasons for living. Cover the current episode, the most severe past episode and the most recent one. Take collateral history. Asking directly has not been found to raise risk, and it is what allows the rest of the assessment to happen.

- **Where it sits.** Suicidal behaviour is not a diagnosis in either system. DSM-5-TR carries suicidal behaviour and non-suicidal self-injury as conditions that may be a focus of clinical attention. ICD-11 codes intentional self-harm as an event, not as a mental disorder, so the diagnosis recorded is the underlying condition.

LINE OF EVIDENCE	THE FINDING	WHAT IT SUPPORTS
Family	Aggregation in first-degree relatives beyond the transmitted psychiatric disorder	**Something specific is transmitted**
Twin	Higher concordance in monozygotic than dizygotic pairs	A genetic contribution to attempt
Adoption	Excess in biological, not adoptive, relatives	**Genes rather than shared home**
Molecular	Serotonergic and stress-axis candidates; few loci replicate at genome-wide level	Polygenic, small individual effects
Endophenotype	Impulsive aggression tracks through families	The trait is inherited, not the act

Amber marks the two rows that answer the question actually asked: what is transmitted, and whether it is genetic rather than environmental.

WHAT EARNS THE MARKS

- **A graded enquiry written as a sequence,** not the single question "are you suicidal".
- **Access to means and deterrents both asked,** since these are what the plan is built on.
- **The nosological point stated plainly,** that the recorded diagnosis is the underlying disorder.
- **Impulsive aggression named as the endophenotype,** which is the line that lifts the genetics section.

SEE ALSO Slot II - 98 prevention and post-attempt care Slot II - 100 biopsychosocial aspects **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

II - 97

Define aggression. How would you assess and manage an adult presenting with frequent aggression? [2+4+4]

2 + 4 + 4 | SUICIDE, SELF-HARM AND IMPULSE CONTROL

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Definition**	Aggression, and the impulsive against planned split
4 **Assessment**	Exclude organic, then pattern, then structured risk
3 **Acute management**	Environment, de-escalation, then medication
1 **Longer term**	Treat the syndrome, not the behaviour alone

- **Definition.** Behaviour intended to harm another who is motivated to avoid it. Two forms divide the management: impulsive aggression, with high arousal, a trigger and later remorse; and premeditated aggression, planned, goal directed and with low arousal.
- **Assessment.** Exclude delirium, intoxication and withdrawal, head injury, post-ictal states and dementia first. Then the psychiatric cause, an antecedent-behaviour-consequence analysis, a structured risk tool and collateral history.

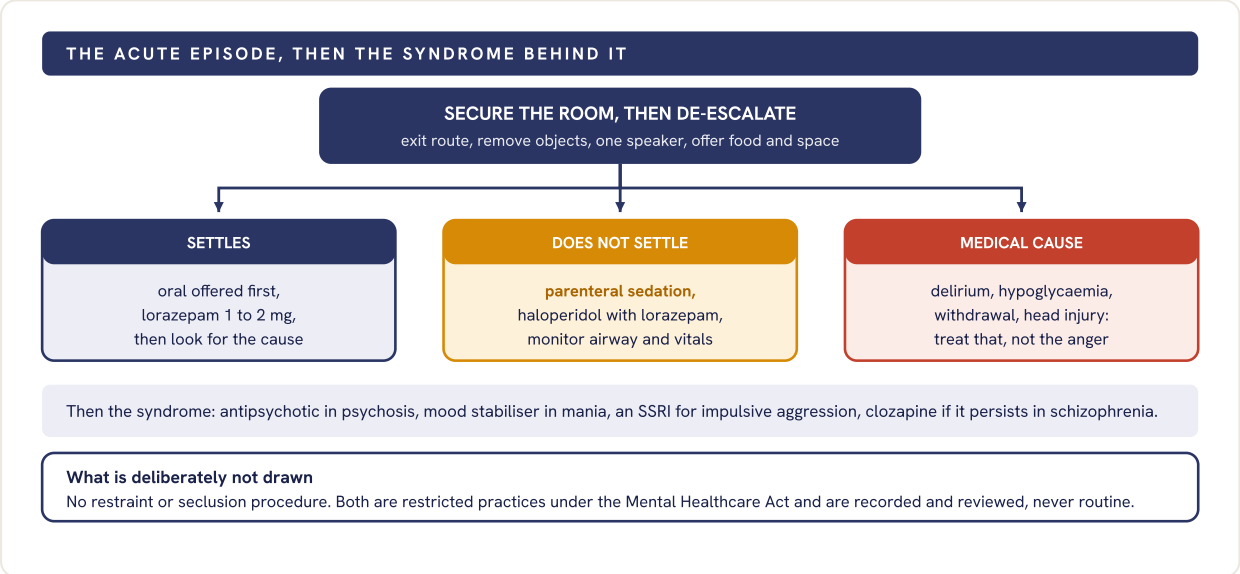

Fig 097.1 The room is made safe before anything is given, and the three lanes are separated by whether the episode settles and whether a medical cause is driving it.

PITFALL

Sedating before excluding a medical cause, which buries the delirium that produced the aggression. The second trap is running chronic aggression on as-required sedation.

WHAT EARNS THE MARKS

- **The impulsive against premeditated split,** stated in the definition because it decides the drug.
- **Organic causes excluded first,** written as a step rather than a caveat at the end.
- **De-escalation before medication,** with the environment described concretely.
- **A named risk tool and collateral history,** which separate assessment from impression.

SEE ALSO Slot II - 99 behavioural addiction **SPRINT** Day 18, Sexual, impulse-control disorders and suicide

II - 98

Describe the biological aspects of suicidal behaviour. Outline the prevention of suicide. Discuss the treatment approach following a self-harm attempt. [3+3+4]

3 + 3 + 4 | SUICIDE, SELF-HARM AND IMPULSE CONTROL

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Biology	Serotonin, stress axis, and what is inherited
3 Prevention	Three tiers, each with its own owner
4 After an attempt	Stabilise, assess, plan, follow up

- **Biology.** Reduced central serotonergic function, with lower cerebrospinal 5-hydroxyindoleacetic acid in violent attempts; altered prefrontal serotonin binding at post-mortem; stress-axis dysregulation; and heritability running through impulsive aggression rather than the act.

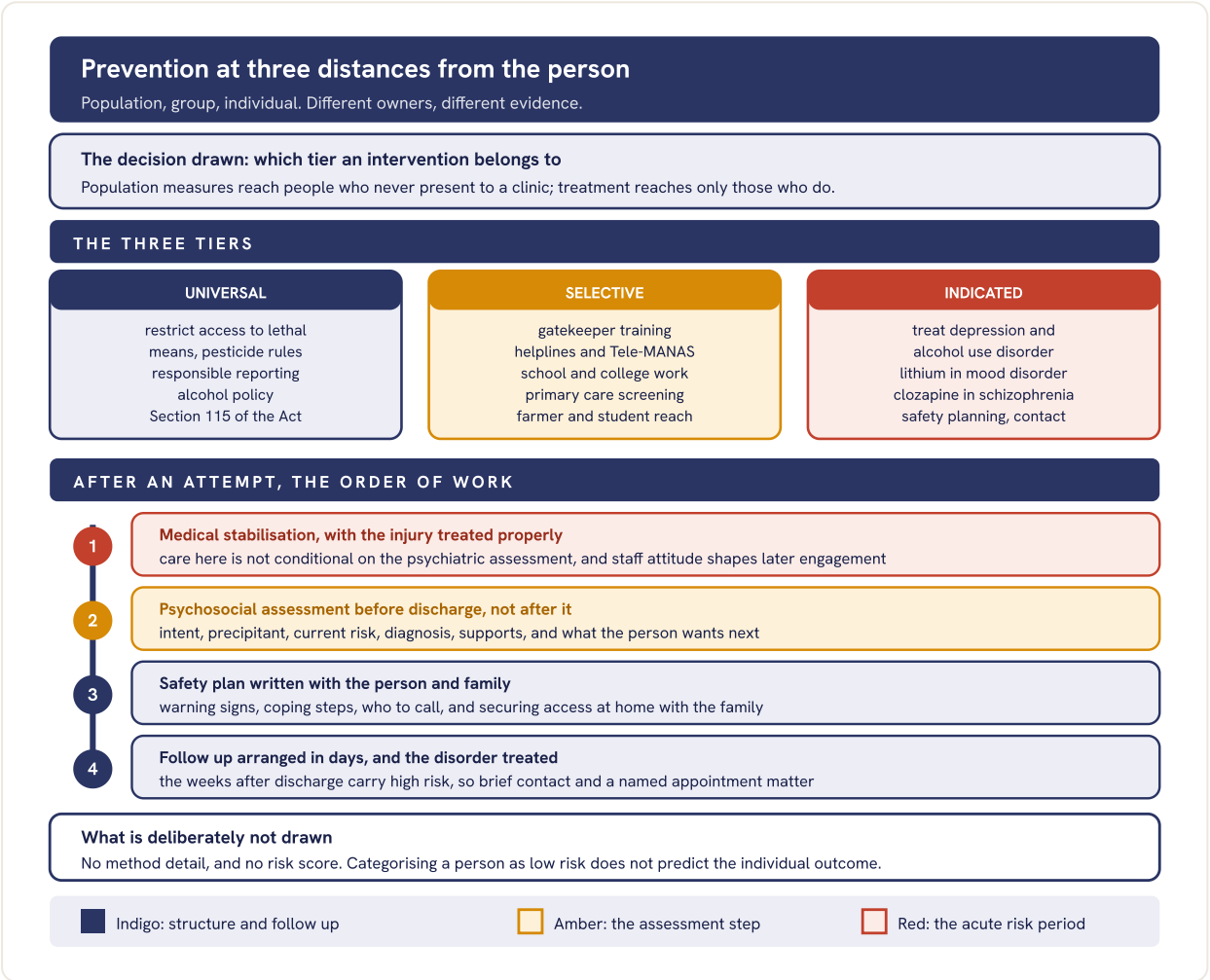

Fig 098.1 Prevention across three tiers, then the post-attempt sequence down a spine, with assessment before discharge, where it is most often lost.

WHAT EARNS THE MARKS

- **Serotonergic and stress-axis findings named,** not "biological factors".
- **Prevention split into three tiers,** with restriction of access at population level where its evidence sits.
- **Assessment placed before discharge,** the most examinable line here.
- **Follow-up named in days,** and the family involved in securing the home.

SEE ALSO Slot II - 96 suicidal enquiry Slot II - 100 the Indian strategy **SPRINT**

Day 18, Sexual, impulse-control disorders and suicide

II - 99

Discuss in detail the epidemiology, aetiology, clinical features and management of behavioural addiction. [10]

10 | ASKED AT 15 MARKS | SUICIDE, SELF-HARM AND IMPULSE CONTROL

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Where it sits	What the systems recognise, and what they still do not
2	Epidemiology	Who, when, and how soft the numbers are
3	Aetiology and features	Reward learning, design, and the clinical picture
3	Management	Screen, psychological work, then the drug question

CONDITION	STATUS IN THE SYSTEMS	REPORTED FREQUENCY	WHAT THE DIAGNOSIS RESTS ON
Gambling disorder	In ICD-11 and DSM-5-TR	Usually under one percent	**Impaired control, priority over life, harm continued, for a year**
Gaming disorder	ICD-11 only; research condition in DSM-5-TR	Estimates vary by instrument	**The same three features, applied to gaming**
Compulsive sexual behaviour	ICD-11: impulse control, not addiction	Not established	Deliberately not an addiction
Shopping, exercise, social media	In neither system	Not established	Described, not diagnosable

Amber marks the only two an examiner can hold you to. Naming what is not yet a diagnosis is itself a mark.

- **Aetiology and features.** Variable-ratio reinforcement, cue reactivity with blunted response to ordinary reward, weak prefrontal control, trait impulsivity, and product design built on near-misses. Clinically: preoccupation, escalation, chasing losses, concealment, irritability on stopping, falling attendance, debt, and comorbid depression, attention deficit and substance use.

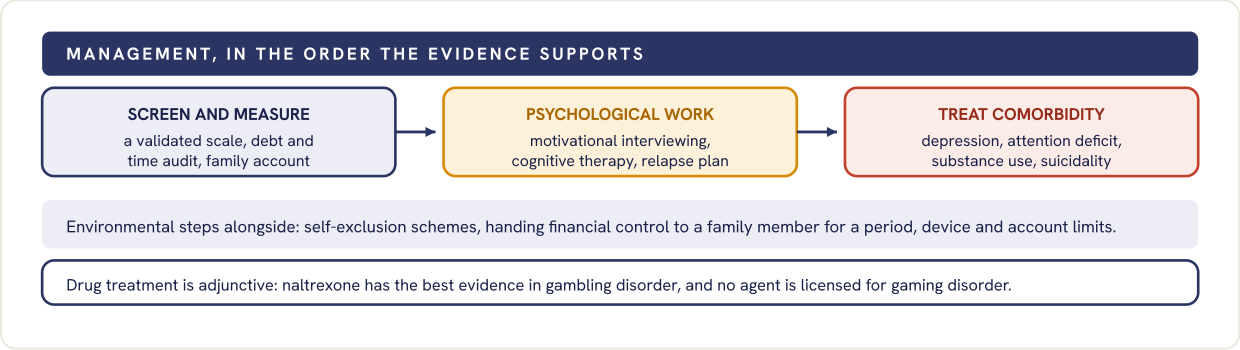

Fig 099.1 Screening, therapy and comorbidity run left to right, with environmental limits and the drug question carried as strips below.

WHAT EARNS THE MARKS

- **The three ICD-11 features named,** which is what makes a behaviour an addiction and not a habit.
- **Honesty about the prevalence figures,** which reads as command rather than evasion.
- **Psychological treatment placed first** and the drug named as adjunctive.
- **Environmental and financial measures,** which most answers omit.

SEE ALSO Slot II - 97 aggression SPRINT Day 18, Sexual, impulse-control disorders and suicide

II - 100

Discuss in detail the biopsychosocial aspects of suicide. Comment on the suicide prevention strategy adopted in India with reference to the National Suicide Prevention Policy. [10]

10 | ASKED AT 15 MARKS | SUICIDE, SELF-HARM AND IMPULSE CONTROL

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The frame	Why one axis alone explains none of it
4 The three domains	Biological, psychological, social, each with a named model
4 India	The statute, the strategy, and what is still missing

DOMAIN	WHAT IT CONTRIBUTES	THE MODEL TO NAME
Biological	Reduced serotonergic function, stress-axis dysregulation, heritable impulsive aggression	The stress-diathesis account
Psychological	Hopelessness, cognitive rigidity, entrapment with no perceived exit	Beck on hopelessness; the cry of pain
Psychiatric	Mood disorder, alcohol use, schizophrenia; most carry a disorder at the time	Psychological autopsy findings
Interpersonal	Thwarted belonging, perceived burdensomeness, acquired capability	The interpersonal theory
Social	Debt, unemployment, agrarian distress, examination pressure, alcohol, media portrayal	Durkheim's four types

Amber marks the two theories examiners expect by name. A model attached to each domain converts a list into an account.

- **The Indian picture.** National crime records show the recorded number rising over the past decade, with daily wage earners, homemakers and students among the largest recorded groups. Access to highly toxic agricultural chemicals is the most modifiable environmental factor.

STATUS: WHERE INDIAN POLICY SITS TODAY

Section 115 of the Mental Healthcare Act 2017 presumes severe stress in a person who attempts suicide, bars prosecution, and places a duty on the government to provide care. The National Suicide Prevention Strategy of 2022 targets a ten percent reduction in suicide mortality by 2030 through three time-bound goals: surveillance within three years, prevention services through district mental health services within five, and a mental well-being curriculum in educational institutions within eight. Tele-MANAS is the tele-counselling arm. Implementation is uneven and dedicated funding is not yet attached.

WHAT EARNS THE MARKS

- **A named model in each domain,** which separates an account from a list of risk factors.
- **Section 115 given by number and by effect,** including the duty to provide care and not only the removal of the offence.
- **The strategy's three time-bound goals,** stated as goals and not as achievements.
- **The closing line on uneven implementation,** which reads as command of the policy.

SEE ALSO Slot II - 96 suicidal enquiry Slot II - 98 prevention tiers **SPRINT**

Day 39, Community psychiatry and public health

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
II-S1	II-73 p 101	II-20 p 35	II-92 p 126	II-31 p 48	II-40 p 59	II-83 p 113	II-16 p 29	II-18 p 31	II-71 p 98	II-94 p 128
II-S2	II-28 p 44	II-38 p 57	II-47 p 68	II-12 p 25	II-56 p 79	II-15 p 28	II-26 p 41	II-68 p 94	II-97 p 132	II-85 p 115
II-S3	II-52 p 74	II-91 p 124	II-54 p 77	II-48 p 69	II-86 p 117	II-75 p 104	II-62 p 87	II-17 p 30	II-98 p 133	II-09 p 20
II-S4	II-01 p 11	II-03 p 14	II-96 p 131	II-11 p 24	II-33 p 50	II-14 p 27	II-34 p 51	II-08 p 19	II-72 p 99	II-90 p 122
II-S5	II-29 p 45	II-53 p 76	II-22 p 37	II-10 p 22	II-61 p 86	II-06 p 17	II-35 p 52	II-51 p 72	II-84 p 114	II-89 p 121
II-S6	II-02 p 12	II-30 p 47	II-39 p 58	II-74 p 103	II-67 p 93	II-05 p 16	II-42 p 61	II-93 p 127	II-78 p 107	II-100 p 136
II-S7	II-36 p 54	II-45 p 66	II-46 p 67	II-55 p 78	II-04 p 15	II-58 p 81	II-07 p 18	II-77 p 106	II-87 p 119	II-99 p 135
II-S8	II-44 p 64	II-21 p 36	II-80 p 110	II-66 p 92	II-13 p 26	II-41 p 60	II-43 p 62	II-27 p 42	II-70 p 97	II-65 p 90
II-S9	II-19 p 33	II-60 p 84	II-23 p 38	II-32 p 49	II-25 p 40	II-82 p 112	II-63 p 88	II-76 p 105	II-88 p 120	II-95 p 129
II-S10	II-37 p 56	II-59 p 83	II-24 p 39	II-81 p 111	II-49 p 70	II-57 p 80	II-50 p 71	II-69 p 96	II-64 p 89	II-79 p 108

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
II-S1	100	0.449	8	4	Other psychoses 2; Substance use: opioids 2; Personality disorders 1
II-S2	100	0.427	9	3	Substance use: opioids 2; Schizophrenia: aetiology 1; Trauma 1
II-S3	100	0.428	9	4	Mood disorders: pharmacotherapy 2; Other psychoses 1; Schizophrenia: treatment 1
II-S4	100	0.424	6	1	Eating 3; Substance use: opioids 2; Schizophrenia: aetiology 2
II-S5	100	0.423	9	2	Schizophrenia: aetiology 2; Mood disorders: pharmacotherapy 1; Anxiety 1
II-S6	100	0.415	7	2	Eating 2; Trauma 2; Personality disorders 2
II-S7	100	0.410	7	3	Schizophrenia: treatment 2; Mood disorders: pharmacotherapy 2; Eating 2
II-S8	100	0.409	7	3	Anxiety 2; Psychotherapies 2; Trauma 2
II-S9	100	0.398	7	3	Anxiety 3; Mood disorders: syndromes 2; Schizophrenia: aetiology 1
II-S10	100	0.401	8	3	Mood disorders: syndromes 2; Schizophrenia: treatment 2; Trauma 1

Mean score is the selection score of the ten questions in that set, on the same scale used across all four books. The spread between the strongest and the weakest set is small by construction.

Three hours, ten questions, one hundred marks. That is the shape of the real paper, so it is the shape of these. Eighteen minutes a question leaves you twenty minutes at the end, which is roughly what you will have and roughly what you will need.

Write the skeleton first, for all ten. Spend the first twenty minutes writing nothing but mark splits and section headings across all ten answers. The map on each page shows what that skeleton should look like, so you can mark your own against it before you have written a line of content. Most marks lost in this examination are lost to allocation, not to ignorance.

Then mark yourself against the anchors. Every map ends with three or four things an examiner ticks. Those are the marking scheme, as close as this book gets to one. Count how many you hit before you read the page, because reading it first will convince you that you knew it.

Sit them in order, or sit them backwards. Set one holds the strongest question in the book and set ten the weakest, but the decile deal keeps the mean scores close, so any set is a fair paper. In order simply means the first one you sit is the one most like the paper you will get.

What a set is not. It is not a mock paper from any board, it is not a leak, and it is not a prediction. It is ten questions a board set, arranged so that ten of them look like a paper. Sit five and you will have written a hundred answers in the shape the examination wants, which is the only preparation that has ever reliably worked.

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Every entry points at the map that answers it. A subject asked in several places carries up to three pages, and the one in bold is the map whose question is about it.

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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 psychotherapy, personality and trauma areas, and of the suicide and self-harm maps. 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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Eight boards were counted: DNB, KNRUHS, KUHS, MUHS, NTRUHS, RGUHS, TN Dr. MGR Medical University and WBUHS. Seventy-eight sittings of theory papers, weighted by how recent they are and how well the paper is evidenced.

From that count, fourteen areas were weighted and one hundred questions were drawn in proportion. Each is solved as a one-page answer map: mark split, skeleton, the content that earns the marks, and what the examiner ticks. Ten ready-made sets at the back turn the hundred into ten sittings.

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