weave

Centre for Integrative Psychiatry

WEAVE 100 / PAPER II

Psychoses, Mood and the Adult Disorders

One hundred ten-mark questions, solved as answer maps

MD Psychiatry theory. DNB revision fits too.



ILLUSTRATED REVIEW EDITION

weave.clinic

Dr. Wilfred Dsouza · Dr. Niharika Reddy

WEAVE

A NOTE ON WHO MADE THIS

Weave, Centre for Integrative Psychiatry

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

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

WEAVE 100 - PAPER 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.

WRITTEN BY

Dr. Wilfred Dsouza and Dr. Niharika Reddy

PUBLISHED BY

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EDITION

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

First edition (R1), August 29, 2026.
Illustrated edition, September 2026. Original answer maps with 134 selected plates. Page references updated for this edition.

SOURCES

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

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

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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% 40

03 **Anxiety, OCD and related disorders** II-19 to II-27 · 9 slots · 8.9% 60

04 **Schizophrenia: aetiology, phenomenology and course** II-28 to II-35 · 8 slots · 8.4% 86

05 **Trauma, dissociative and somatic symptom disorders** II-36 to II-43 · 8 slots · 8.4% 103

06 **Schizophrenia: treatment and antipsychotics** II-44 to II-51 · 8 slots · 8.3% 118

07 **Mood disorders: pharmacotherapy** II-52 to II-58 · 7 slots · 7.5% 134

08 **Mood disorders: syndromes and nosology** II-59 to II-65 · 7 slots · 7.5% 148

09 **Psychotherapies** II-66 to II-72 · 7 slots · 6.9% 162

10 **Personality disorders** II-73 to II-79 · 7 slots · 6.6% 202

11 **Clinical assessment and cross-cutting nosology** II-80 to II-85 · 6 slots · 5.5% 216

12 **Substance use: general concepts and alcohol** II-86 to II-90 · 5 slots · 4.5% 235

13 **Other psychoses and catatonia** II-91 to II-95 · 5 slots · 4.3% 254

14 **Suicide, self-harm and impulse control** II-96 to II-100 · 5 slots · 4.0% 262

BACK MATTER

Ten sets, ten sittings 271

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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

Anorexia nervosa

Restriction + low weight + weight / shape psychopathology

Concise criteria summary • supplied ICD-10, ICD-11 and DSM-5-TR (2022)

Criterion	ICD-10	ICD-11	DSM-5-TR
Weight	≥15% below expected weight OR **BMI ≤17.5.**	Significantly low weight; common threshold BMI <18.5 adults, BMI-for-age <5th percentile children/ adolescents. Rapid loss (e.g. >20% in 6 months) may substitute if other requirements met.	A. Restricted energy intake causes **significantly low weight in age, development and health context.**
Behaviour	Self-induced loss through avoidance of fattening foods; other weight-loss **behaviours may occur.**	Persistent restriction or other behaviour **establishing or maintaining abnormally low weight, typically with extreme fear of weight gain.**	**B. Intense fear of weight gain OR** persistent behaviour interfering with weight gain despite low weight.
Weight/shape psychology	Dread of fatness; self-imposed low weight threshold.	**Excessive weight/shape preoccupation:** low weight overvalued and central to self-evaluation OR body weight/shape **perceived as normal or excessive.**	C. Distorted weight/shape experience OR undue influence on self-evaluation **OR persistent failure to recognise** seriousness of low weight.
Endocrine/ **development**	Amenorrhoea in women; reduced sexual interest/potency in men. If prepubertal: development delayed or arrested.	Amenorrhoea not required.	**Amenorrhoea not required.**
Exclusions/ context	**BMI criterion used from age 16 in** supplied manual; assess expected growth in children.	Not better explained by another medical condition or food unavailability.	A, B and C all required. Low weight judged in developmental and physical-health context.

Sources: WHO ICD-10 CDDG pp138-139; WHO ICD-11 CDDR pp398-399 (PDF416-417); DSM-5-TR2022 p381 (PDF556). Paraphrased core criteria.

Anorexia nervosa - Diagnostic criteria. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-01.

Credits and references: retained within the illustration.

Bulimia nervosa

Loss-of-control binges + repeated compensation

Concise criteria summary • supplied ICD-10, ICD-11 and DSM-5-TR (2022)

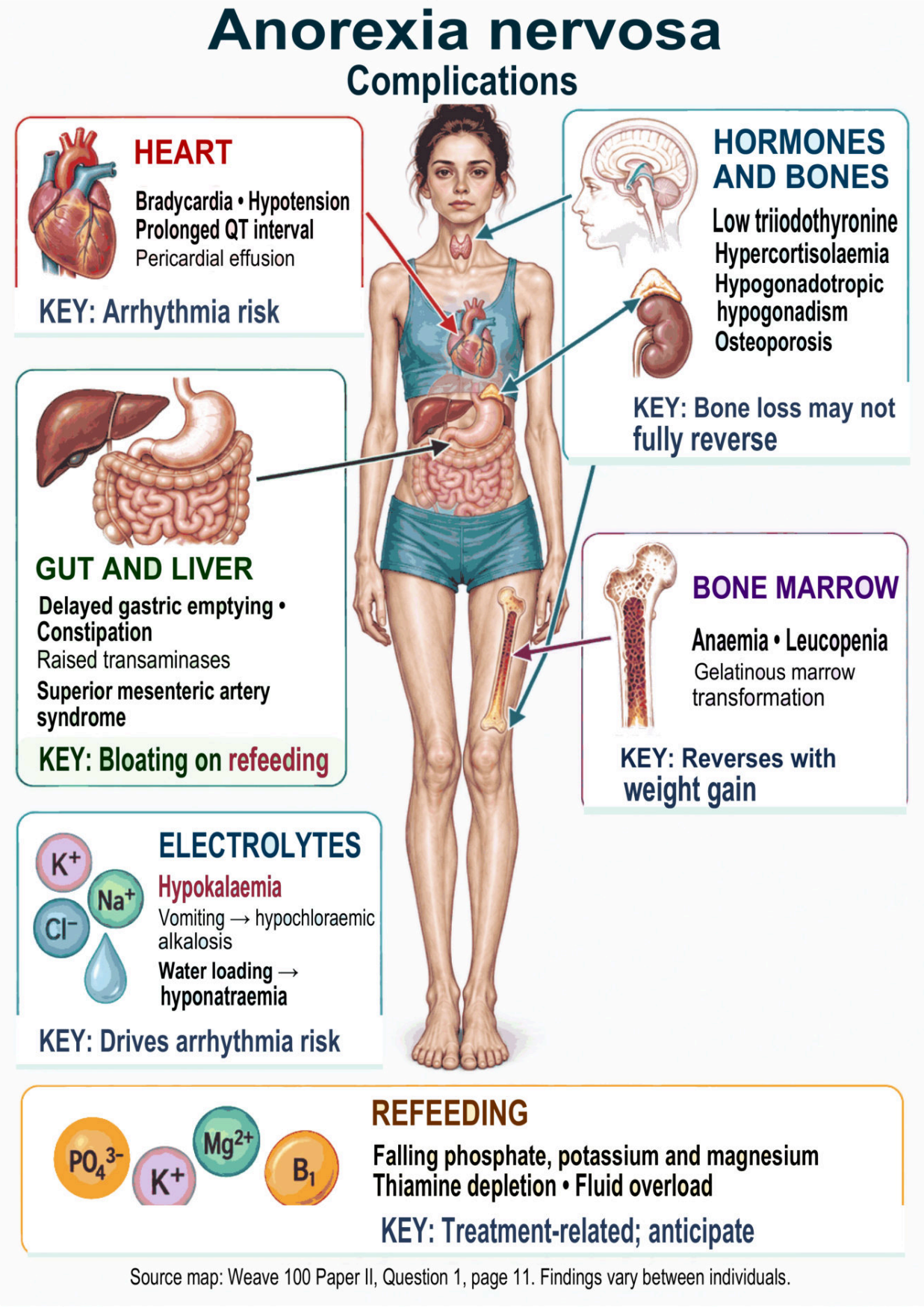
Clinical feature	ICD-10 (WHO clinical guidelines)	ICD-11	DSM-5-TR (APA, 2022)
Core eating features	**EATING PREOCCUPATION** Persistent preoccupation and craving; recurrent large amounts eaten in short periods.	**BINGES** Loss of control; eating notably more or differently than usual.	**A • BINGES** Objectively unusually large intake + loss of control in a discrete period.
Compensation	**COMPENSATION** Repeated attempts to counteract feared weight gain.	**COMPENSATION** Repeated inappropriate behaviour intended to prevent weight gain.	**B • COMPENSATION** Recurrent inappropriate weight-gain-preventing behaviour.
Weight / shape concerns	**DREAD OF FATNESS** A sharply defined low weight threshold.	**WEIGHT / SHAPE** Excessive preoccupation.	**D • SELF-WORTH** Unduly influenced by weight and shape.
Frequency / duration	**FREQUENCY** No numerical frequency or duration threshold in this supplied clinical-guidelines edition.	**FREQUENCY EXAMPLE** Binges and compensation each at least weekly for at least 1 month.	**C • FREQUENCY** Both at least once weekly for 3 months.
Distress / exclusion		**DISTRESS / EXCLUSION** Marked distress or impairment; does not meet anorexia requirements.	**E • EXCLUSION** Not exclusively during anorexia nervosa.
Key distinction	Compensation is broader than purging: it can also involve fasting or excessive exercise. Purging itself is not mandatory.		

ICD-10: PDF 140 • ICD-11: PDF 422 • DSM-5-TR: PDF 564 • Summary, not verbatim wording.

Bulimia nervosa - Diagnostic criteria. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-01.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 1.5



Anorexia nervosa - Complications. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-01.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 1.6

Bulimia nervosa

Signs and laboratory findings

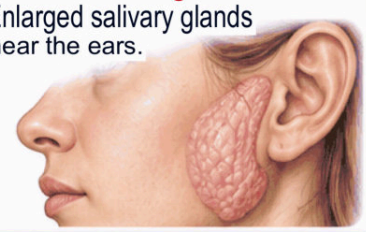
EYES

Subconjunctival haemorrhage
Small bleed beneath the clear covering of the eye.



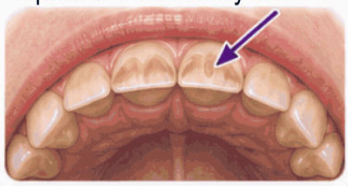
CHEEKS

Parotid swelling
Enlarged salivary glands near the ears.



TEETH

Dental erosion
Repeated stomach-acid exposure wears away enamel.



BLOOD TESTS

Hypokalaemia
- low potassium; arrhythmia risk.

Hypochloraemic alkalosis
- low chloride and raised bicarbonate, associated with vomiting.

Raised amylase
- may be salivary in origin.

KNUCKLES

Knuckle callus (Russell's sign)
Repeated contact with teeth during vomiting.



FLUID BALANCE

Rebound oedema
Fluid retention after stopping **laxatives; may appear as** ankle swelling.



Weight is often normal. These findings are clues, not required in every person.

Source findings: Weave 100 Paper II, Question 7, page 18. Mechanism labels: tutor explanations.

Bulimia nervosa - Signs and laboratory findings. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-01.

Credits and references: retained within the illustration.

Anorexia nervosa and bulimia nervosa - Management

Assess medical and psychiatric risk

Vitals, hydration, electrolytes, electrocardiogram when indicated, suicide risk → urgent care if unstable. [NICE; RCPsych MEED]

ANOREXIA NERVOSA

1 Restore nutrition and weight [NICE NG69; APA 2023]
Individualised plan; monitor growth, weight and physical recovery.

2 Prevent refeeding complications
Risk-based refeeding; thiamine; monitor phosphate, potassium, magnesium and fluid balance.

3 Adults: specialist therapy [NICE NG69; APA 2023]
Eating-disorder-focused cognitive behavioural therapy; Maudsley Anorexia Nervosa Treatment for Adults; specialist supportive clinical management.

Children and **adolescents: [NICE; APA]** Family-based therapy; involve carers.

4 Medication
No FDA-approved medicine for anorexia nervosa. Nutritional rehabilitation and psychotherapy remain central; olanzapine is an off-label specialist adjunct.

5 Follow-up
Monitor weight, nutrition, bone health and physical risk; relapse prevention.

BULIMIA NERVOSA

1 Correct purging-related medical risk [NICE; RCPsych MEED]
Check potassium, bicarbonate, hydration and electrocardiogram.

2 Adults: guided self-help [NICE NG69]
If unsuitable or ineffective after 4 weeks → individual eating-disorder-focused cognitive behavioural therapy: up to 20 sessions over 20 weeks.

3 Therapy targets
Regular eating → reduce dietary restraint, weight/shape overvaluation and binge triggers → relapse prevention.

Children and **adolescents: [NICE; APA]** Bulimia-focused family therapy.

4 Medication [APA 2023]
Fluoxetine 60mg/day WITH psychological treatment, initially or if minimal response by 6 weeks [APA 2023]. FDA-approved for adult bulimia nervosa.

5 Follow-up
Gradually stop laxative misuse; dental review; monitor binges, compensation, electrolytes and comorbidity.

⚠ Bupropion is contraindicated in current or previous anorexia nervosa or bulimia nervosa: seizure risk.

Nutrition and psychological care proceed together once medically appropriate.

Evidence and US approval - Anorexia nervosa

Olanzapine vs placebo: 16-week adult trial, n=152.
Body mass index gain 0.259 vs 0.095 kg/m²/month; standardised week-16 effect 0.63 (95% confidence interval 0.11-1.15).
No significant obsessionality benefit.
Attia et al., 2019.

Evidence and US approval - Bulimia nervosa

Fluoxetine 60 mg/day vs placebo, 8-16-week trials: additional median reduction of 1-2 binges/week and 2-4 vomiting episodes/week.
US prescribing information, section 14.3.
Cognitive behavioural therapy vs interpersonal therapy: recovery 29% vs 6% at end of treatment; absolute benefit 23 percentage points; calculated number needed to treat 5. Agras et al., 2000. Trial-specific, not a universal effect.

Guidelines are complementary; arrows show care priorities, not a mandatory rigid sequence.
National Institute for Health and Care Excellence (NICE) NG69 (2017, updated 2020). American Psychiatric Association (APA) - Eating Disorders Guideline (2023).
Royal College of Psychiatrists - Medical Emergencies in Eating Disorders (RCPsych MEED), CR233 (2022; updated 2025).

Anorexia nervosa and bulimia nervosa - Management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-01.

Credits and references: retained within the illustration.

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

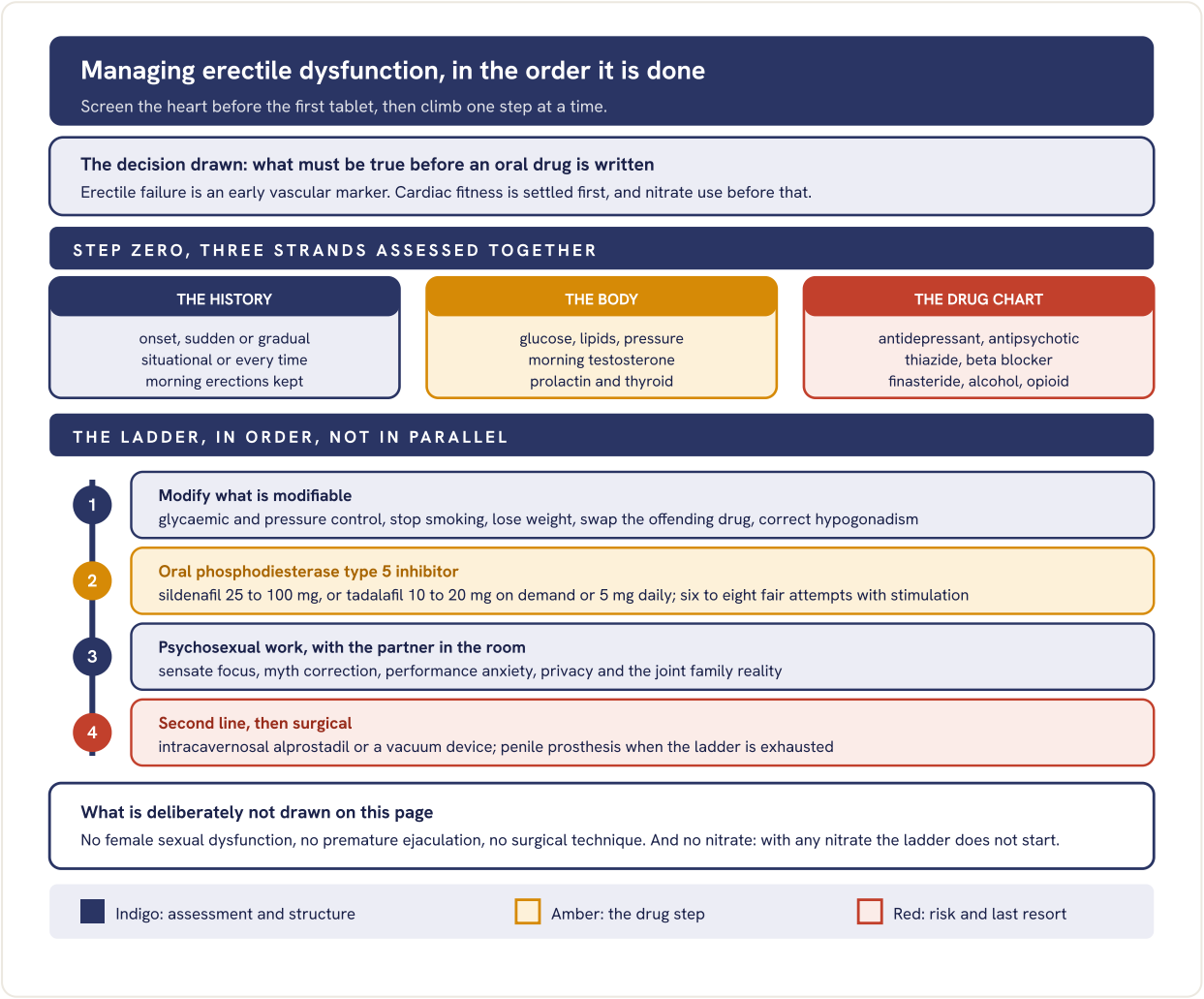


Fig 002.1 Three strands assessed together, then a four-rung ladder climbed in order, the nitrate question settled before rung two.

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

PITFALL

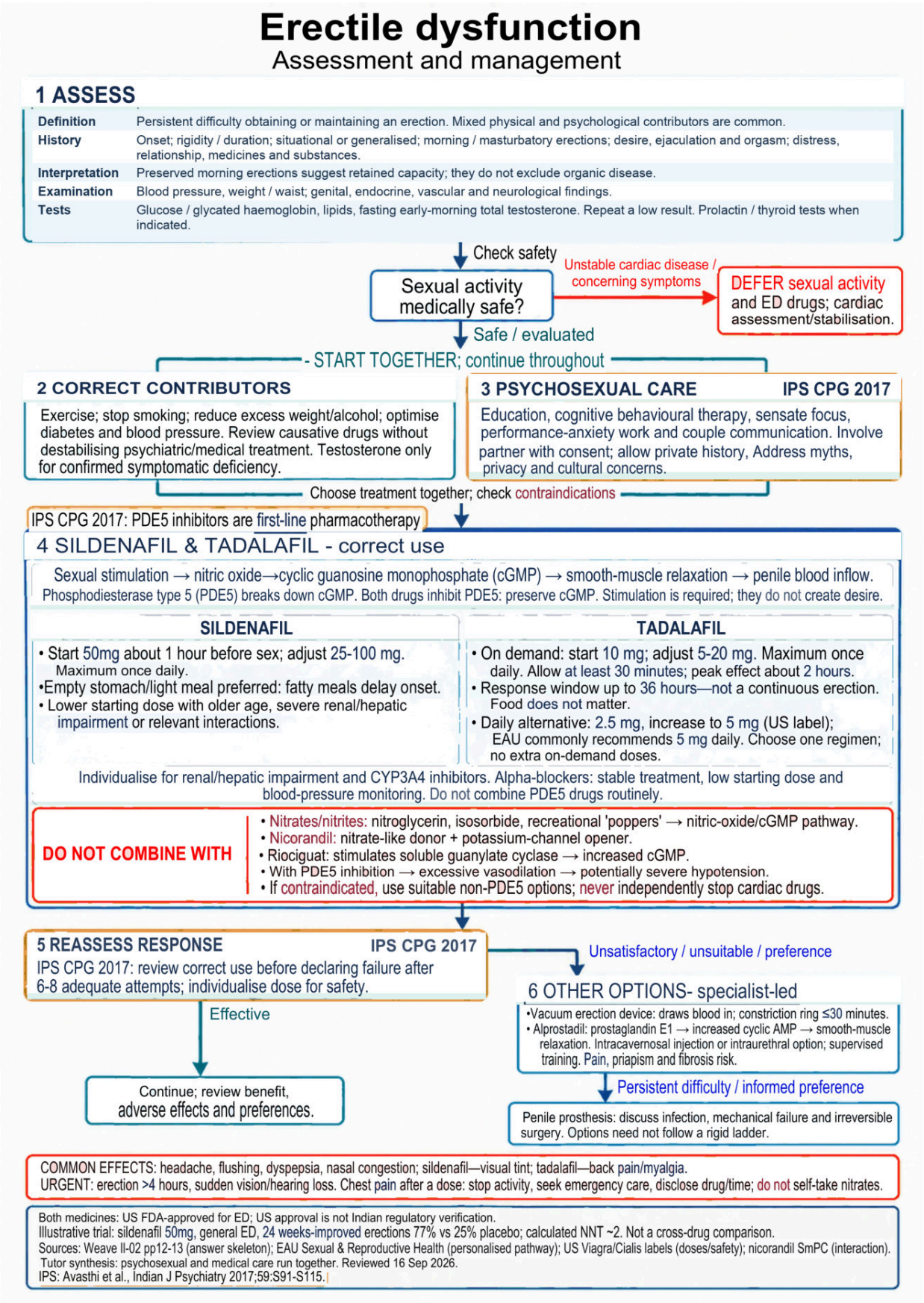
Calling the oral drug a failure too early. Most apparent non-response is an inadequate trial: no stimulation, a full stomach with sildenafil, or the lowest dose tried twice.

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

WEAVE 100 · PAPER II · 2.1



Erectile dysfunction - Assessment and management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-02.

Credits and references: retained within the illustration.

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.

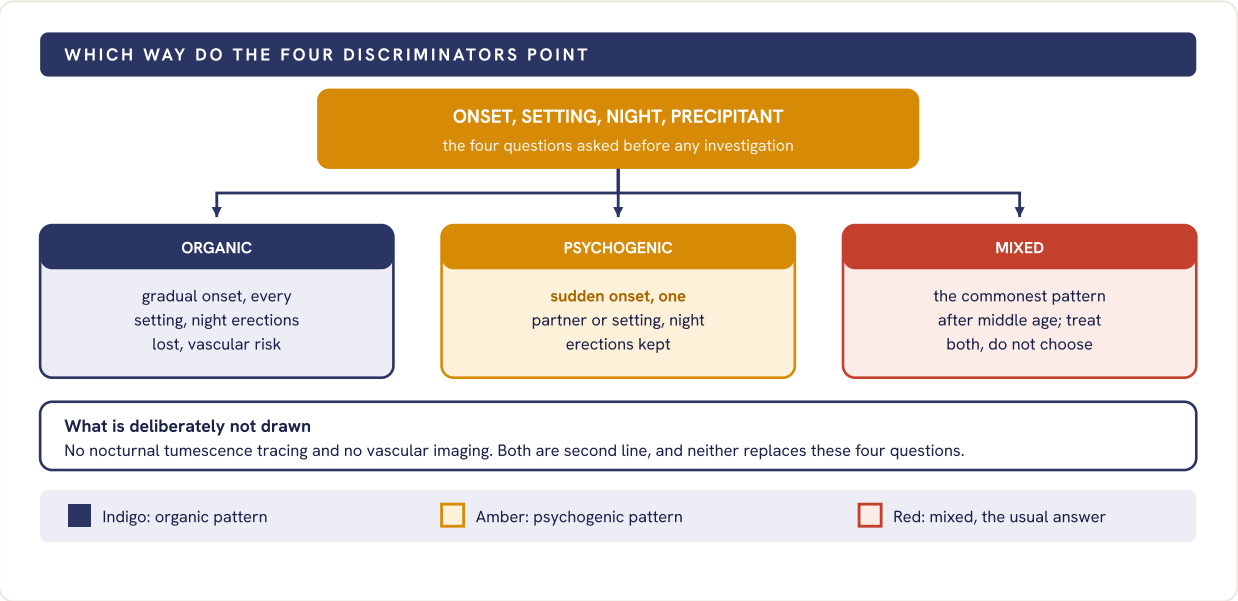


Fig 003.1 The psychogenic label rests on four bedside questions, and the honest third lane is mixed rather than either pure form.

- **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.

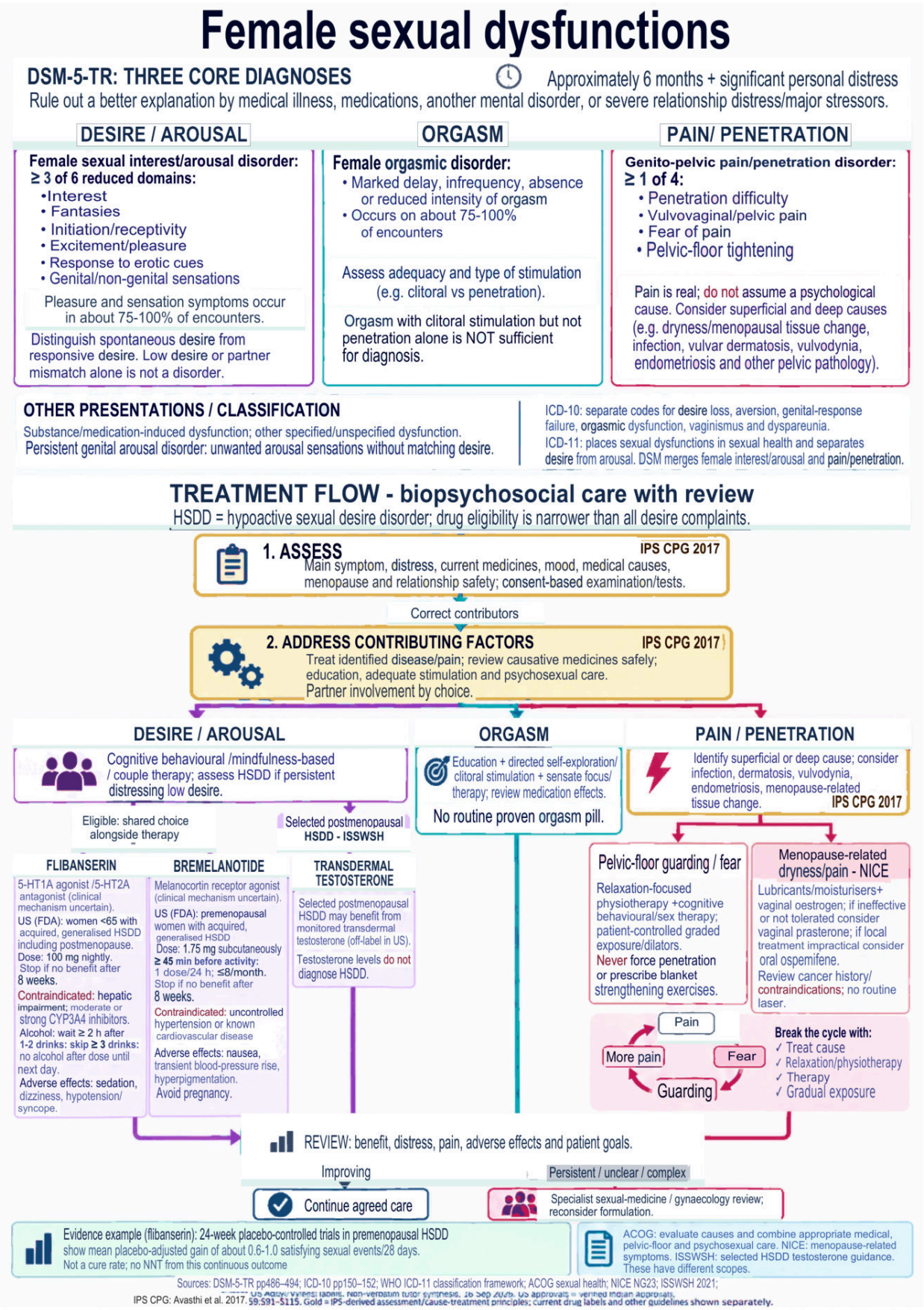
WHAT EARNS THE MARKS

- **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

WEAVE 100 · PAPER II · 3.1



Female sexual dysfunctions - Diagnosis and treatment. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-03.

Credits and references: retained within the illustration.

Male and female sexual dysfunctions

Clinical comparison • DSM-5-TR core features

DOMAIN	MALE	FEMALE
DESIRE	Hypoactive sexual desire disorder Persistently or recurrently reduced erotic thoughts/fantasies and desire, judged in age and context.	Female sexual interest/arousal disorder ≥3 of 6 reduced domains - interest, fantasies, initiation/ receptivity, pleasure, response to erotic cues, genital/non-genital sensations.
AROUSAL	Erectile disorder ≥1: marked difficulty obtaining an erection, maintaining it, OR markedly reduced rigidity; on ~75-100% of occasions.	Arousal is included in female sexual interest/arousal disorder Pleasure and sensation domains affected on ~75-100% of encounters. Lubrication alone is not the diagnosis.
ORGASM / EJACULATION	Delayed ejaculation Marked delay, infrequency or absence on ~75-100% of partnered occasions, without wishing delay. Orgasm and ejaculation are distinct.	Female orgasmic disorder Marked delay, infrequency or absence, or reduced orgasm intensity on ~75-100% of occasions; assess adequate stimulation.
EARLY EJACULATION	Premature (early) ejaculation Approximately ≤1 minute after vaginal penetration, before desired, on ~75-100% of occasions. Other activities: no established DSM time cutoff.	No corresponding named DSM-5-TR category.
PAIN / PENETRATION	Genital or ejaculatory pain Assess urological, dermatological and pelvic-floor causes; not a named male DSM pain category.	Genito-pelvic pain/penetration disorder ≥1 persistent or recurrent problem - penetration difficulty, pain, fear of pain, pelvic-floor tightening. Do not assume psychological causation.
DIAGNOSTIC GATE	For the primary DSM disorders above: approximately ≥6 months + clinically significant personal distress + exclusion of better explanations. Lifelong/acquired; generalized/situational. The 75-100% rule is diagnosis-specific, not universal. Substance/medication-induced dysfunction has its own criteria, not this six-month gate.	
ASSESS BOTH	Onset/context → medical and medication review → mood, anxiety, relationship and consent → focused examination / indicated tests. Mixed contributors are common.	
IPS CPG 2017	Comprehensive biopsychosocial assessment, sex education and psychosexual interventions; assess partner and relationship factors.	
NORMAL VARIATION	Asexuality, responsive desire, desire discrepancy or absent orgasm from penetration alone are not automatically disorders. Use current anatomy and individual clinical context, including gender-diverse people.	

Sources: APA DSM-5-TR (2022), Sexual Dysfunctions; IPS Avasthi et al. (2017). Core summary, not complete criteria.

Male and female sexual dysfunctions - Diagnostic comparison. Read across the labelled contrasts to distinguish the related concepts. Read with II-03.

Credits and references: retained within the illustration.

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.

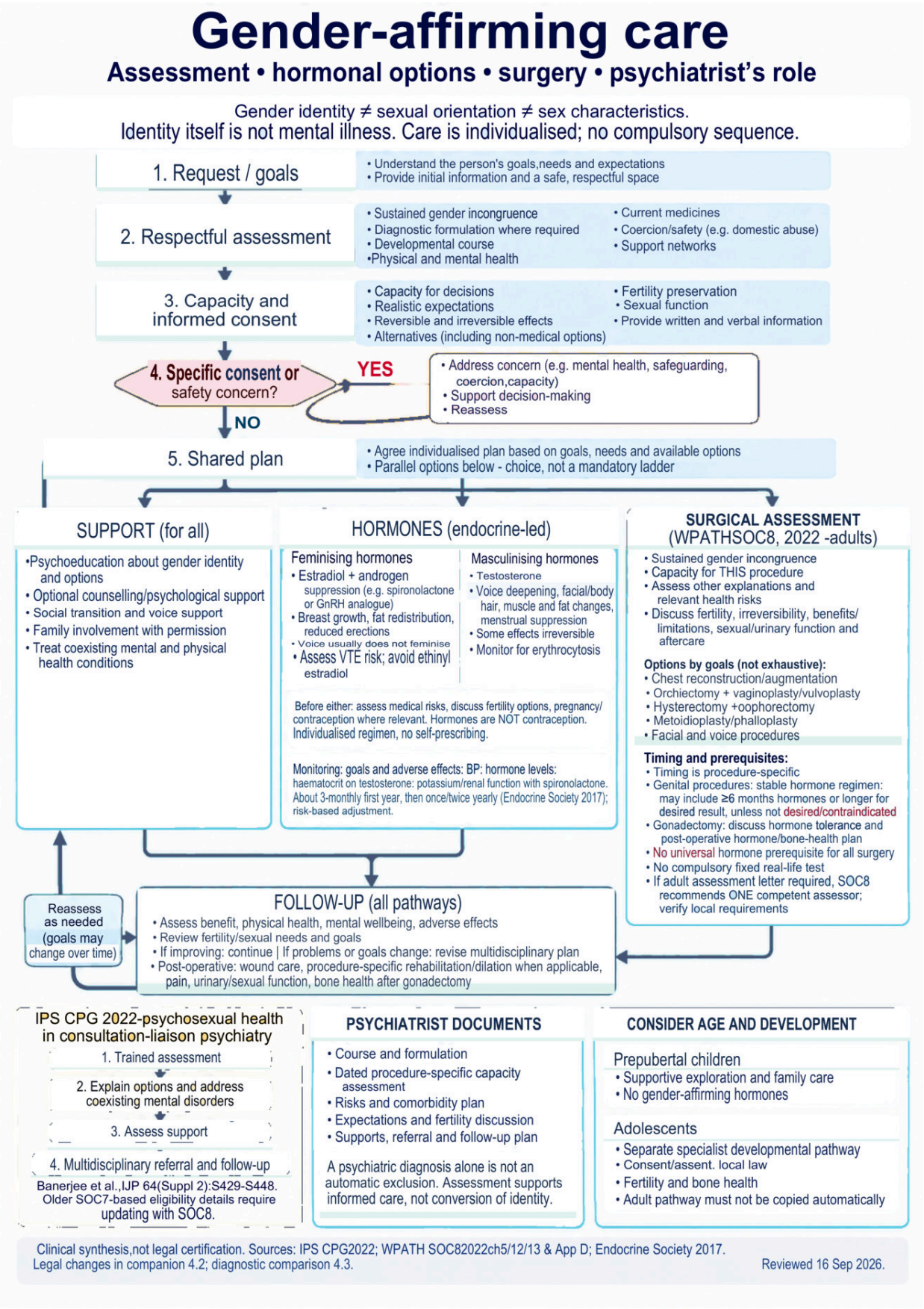
- **The Indian frame.** The 2014 Supreme Court judgment recognised the right to a self-identified gender, and the Transgender Persons Act of 2019 gives a certificate of identity through the District Magistrate. Surgery is not a precondition for that recognition.

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

WEAVE 100 · PAPER II · 4.1



Gender-affirming care - Assessment hormones and surgery. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-04.

Credits and references: retained within the illustration.

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.

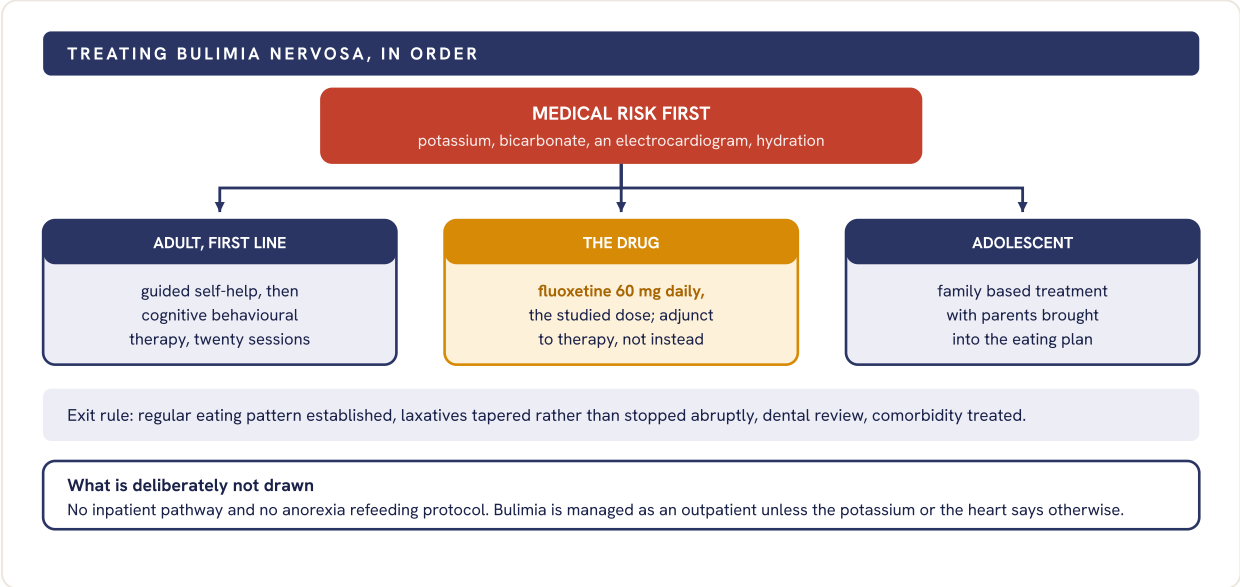


Fig 007.1 Medical risk settled first, then three treatment lanes chosen by age and by role, with the exit rule carried along the foot.

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.

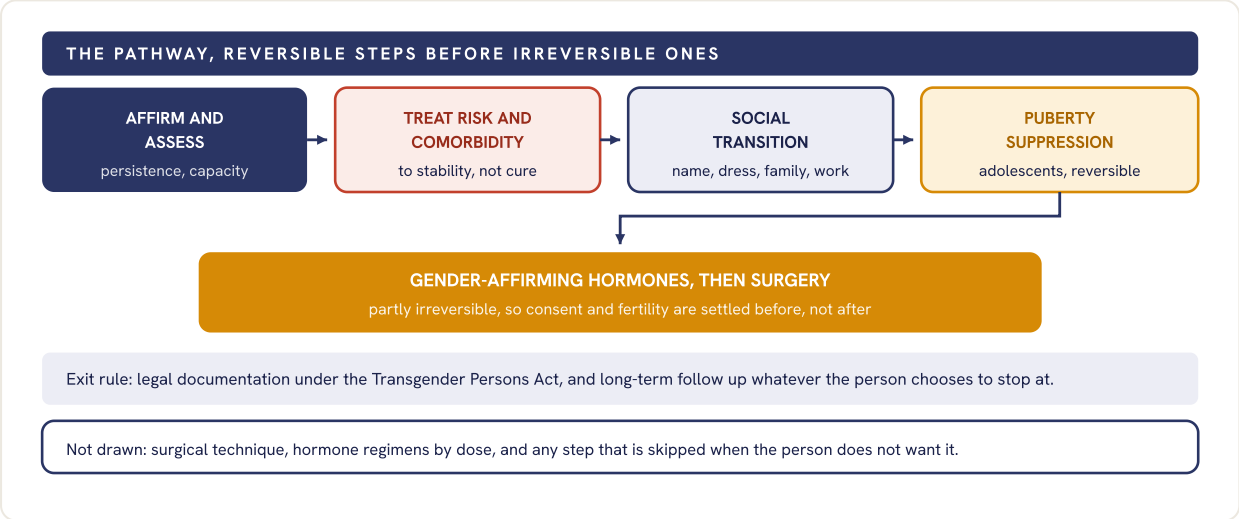


Fig 008.1 Reversible steps precede partly irreversible ones, and the person may stop at any point on the line without the pathway being incomplete.

- **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.

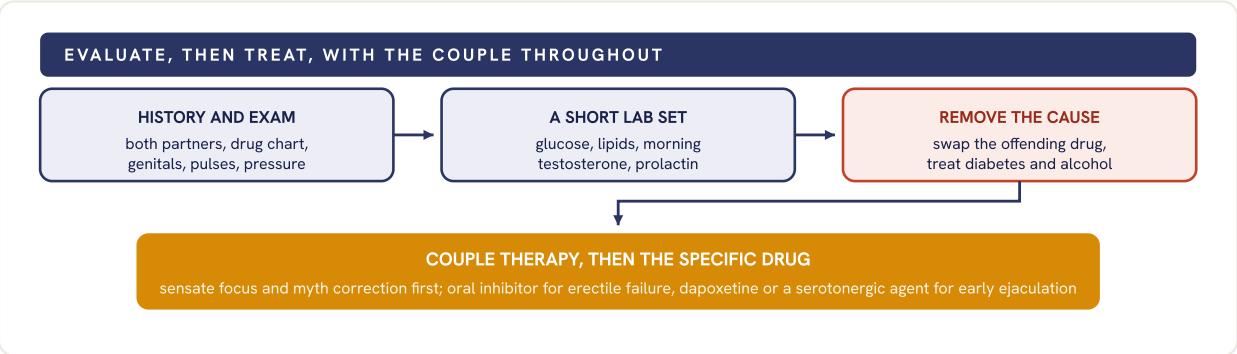


Fig 009.1 Evaluation runs left to right and converges on couple work, with the drug chosen by the phase named in the grid above.

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

WEAVE 100 · PAPER II · 1.10

Avoidant/restrictive food intake disorder

Diagnosis and clinical assessment

Three common drivers

Sensory sensitivity; little interest in food; fear of consequences such as choking or vomiting. These can overlap.

Sensory sensitivity

Texture (tastes, smells, colours, temperature, food appearance)

Little interest in food

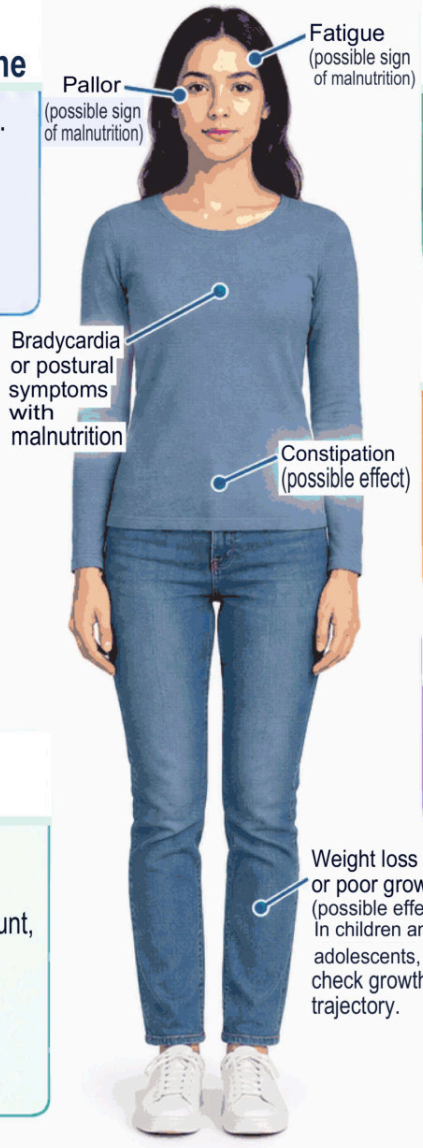
Low appetite or lack of interest in eating.

Fear of consequences

Fear of choking or vomiting.

DSM-5-TR: restriction or avoidance + at least one

- Weight loss or faltering growth.
- Nutritional deficiency.
- Dependence on supplements or tube feeding.
- Marked psychosocial interference.



Key boundary with anorexia nervosa

Restriction is not driven by disturbed body weight or shape experience.

Low weight is not required.

Exclude

- Food scarcity or cultural practice.
- Exclusively anorexia or bulimia.
- Another condition fully **explaining the problem.**
- **With a coexisting condition,** eating difficulty must exceed what is usual and need **separate attention.**

Possible physical effects - not universal

- Fatigue, pallor or weakness.
- **Weight loss or poor growth.**
- **Constipation.**
- Bradycardia or postural symptoms with malnutrition.

Medical priorities

- Check hydration and vital signs.
- Severe malnutrition can carry refeeding risk.

Assessment and laboratory tests

- **No diagnostic blood test.**
- Dietary history and growth trajectory guide tests: blood count, electrolytes and targeted iron, **vitamin B12, folate or vitamin D** testing.
- Normal results do not exclude the disorder.

More than picky eating

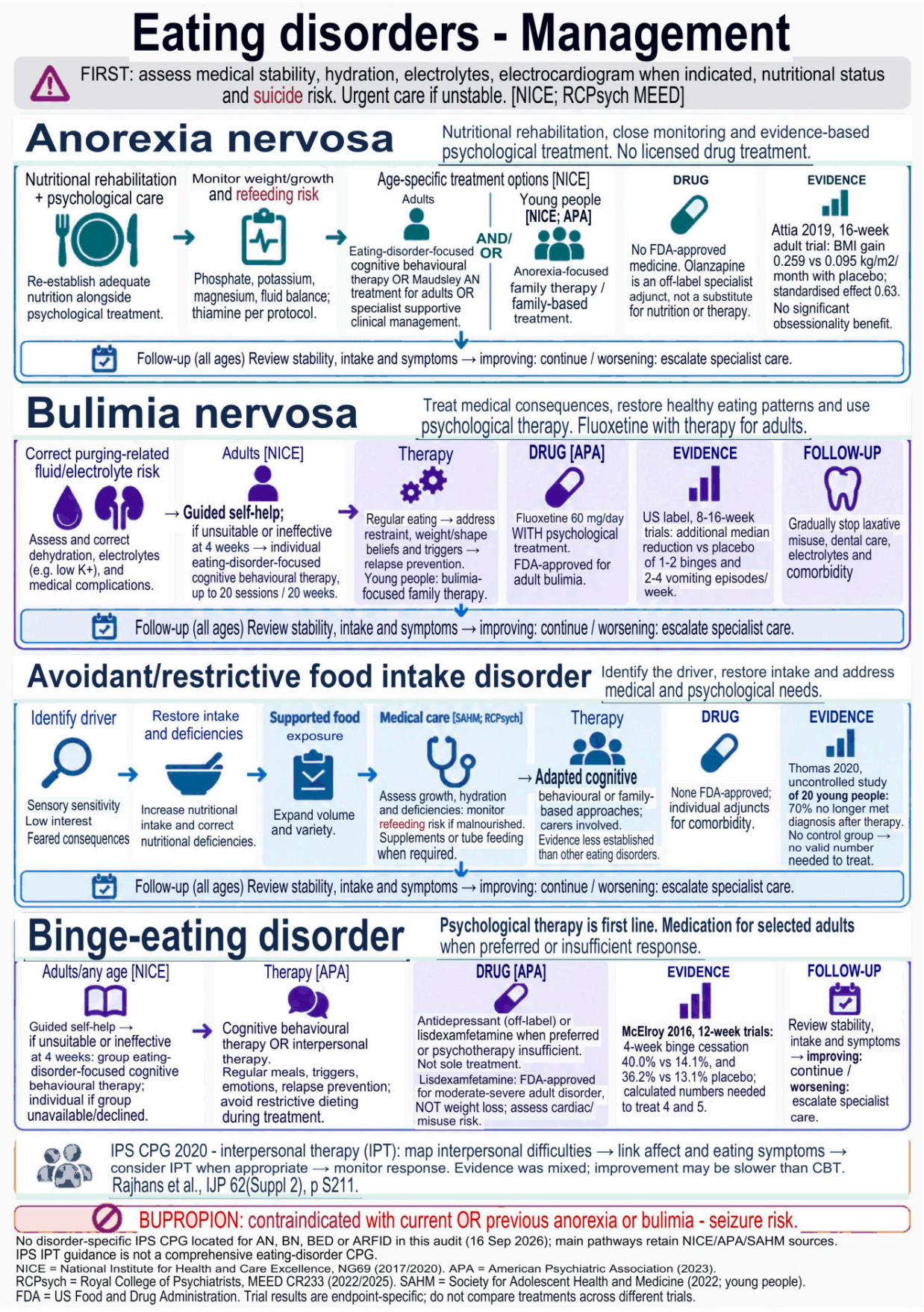
- Clinically significant nutritional or functional consequences.
- Can occur at any age.

Criteria summary: supplied DSM-5-TR, PDF 550. Clinical assessment: tutor synthesis.
Body illustration does not define the diagnosis.

Avoidant restrictive food intake disorder - Clinical summary. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Eating, sleep and sexual disorders.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 1.11



Eating disorders - Management essentials. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Eating, sleep and sexual disorders.

Credits and references: retained within the illustration.

Binge-eating disorder

Loss-of-control binges + distress, without repeated compensation

	ICD-10	ICD-11	DSM-5-TR
Binge definition	No separate criteria This supplied ICD-10 clinical-guidelines manual has no separate binge-eating disorder criteria set.	Binges Loss of control; eating notably more or differently than usual in a discrete period (e.g. 2 hours).	A • Binges Objectively unusually large intake+ loss of control in a discrete period (e.g. 2 hours).
Key features / associated features	No separate criteria Psychologically associated overeating and "other eating disorders" are not equivalent verbatim criteria.	Binge features Loss of control; eating notably more or differently than usual.	B • Three or more **Associated features (at least 3 of 5):** • Eating much faster than usual • Eating until uncomfortably full • Eating large amounts without physical hunger • Eating alone because of embarrassment • Disgust, depression or marked guilt afterward
Frequency	No standalone BED category in this WHO ICD-10 clinical-guidelines edition. Do not import ICD-10-CM criteria.	Frequent, recurrent binges; e.g. at least weekly over 3 months.	D. At least once weekly for 3 months.
No regular compensation		Binges are not regularly followed by inappropriate compensatory behaviour.	E • No compensation No recurrent inappropriate compensation; not exclusively during bulimia or anorexia.
Distress / impairment		Marked distress or significant functional impairment.	C • Distress Marked distress about binge eating
Exclusions		Exclude Better medical, mental-disorder, substance or medication explanations.	Not exclusively during bulimia or anorexia.
Edition notes		A shorter duration may suffice for multiple weekly binges with significant distress.	As above.
Key distinction	Rapid eating alone is not a binge. Loss of control matters. Obesity and shame are not individually required.		

Sources: ICD-10: PDF 138-142 • ICD-11: PDF 425-426 • DSM-5-TR: PDF 570 • Summary, not verbatim wording.

Binge-eating disorder - Diagnostic criteria. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Eating, sleep and sexual disorders.

Credits and references: retained within the illustration.

Eating disorders

ICD-10 • ICD-11 • DSM-5-TR | Diagnostic comparison

	Anorexia nervosa	Bulimia nervosa	Binge-eating disorder
ICD-10	**WEIGHT** ≥15% below expected OR body mass index ≤17.5. **MIND / BEHAVIOUR** Self-induced loss + dread of fatness. **ENDOCRINE** Amenorrhoea / reduced male sexual function; puberty delay if prepubertal.	**EATING** Preoccupation + recurrent large, brief overeating episodes. **COMPENSATION** Attempts to prevent weight gain. **MIND** Dread of fatness. No numerical frequency specified in this edition.	**NO SEPARATE SET** No standalone binge-eating disorder criteria in this supplied ICD-10 manual. **EDITION** Do not replace it with ICD-10-CM criteria.
ICD-11	**WEIGHT** Common adult body mass index <18.5; developmental context matters. **BEHAVIOUR / MIND** Maintains low weight; weight / shape preoccupation. **EXCEPTION** Rapid loss may substitute; exclude medical cause / food unavailability.	**BINGES + COMPENSATION** Example: each ≥weekly for ≥1 month. **MIND / IMPACT** Weight / shape preoccupation + distress or impairment. **EXCLUDE** Anorexia nervosa requirements not met.	**BINGES** Loss of control; example ≥weekly for 3 months. **NO COMPENSATION** No regular compensatory behaviour. **IMPACT** Distress or impairment; exclude other explanations.
DSM-5-TR	**A** Restriction → significantly low weight. **B** Fear of gain OR behaviour preventing gain. **C** Disturbed body experience / weight-based self-worth / unrecognised seriousness.	**A + B** Objective binges with loss of control + recurrent compensation. **C** Both ≥weekly for 3 months. **D+E** Weight-based self-worth; not exclusively during anorexia.	**A + B** Objective binges + loss of control; ≥3 associated features.* **C + D** Marked distress; ≥weekly for 3 months. **E** No recurrent compensation; not exclusively during bulimia / anorexia.

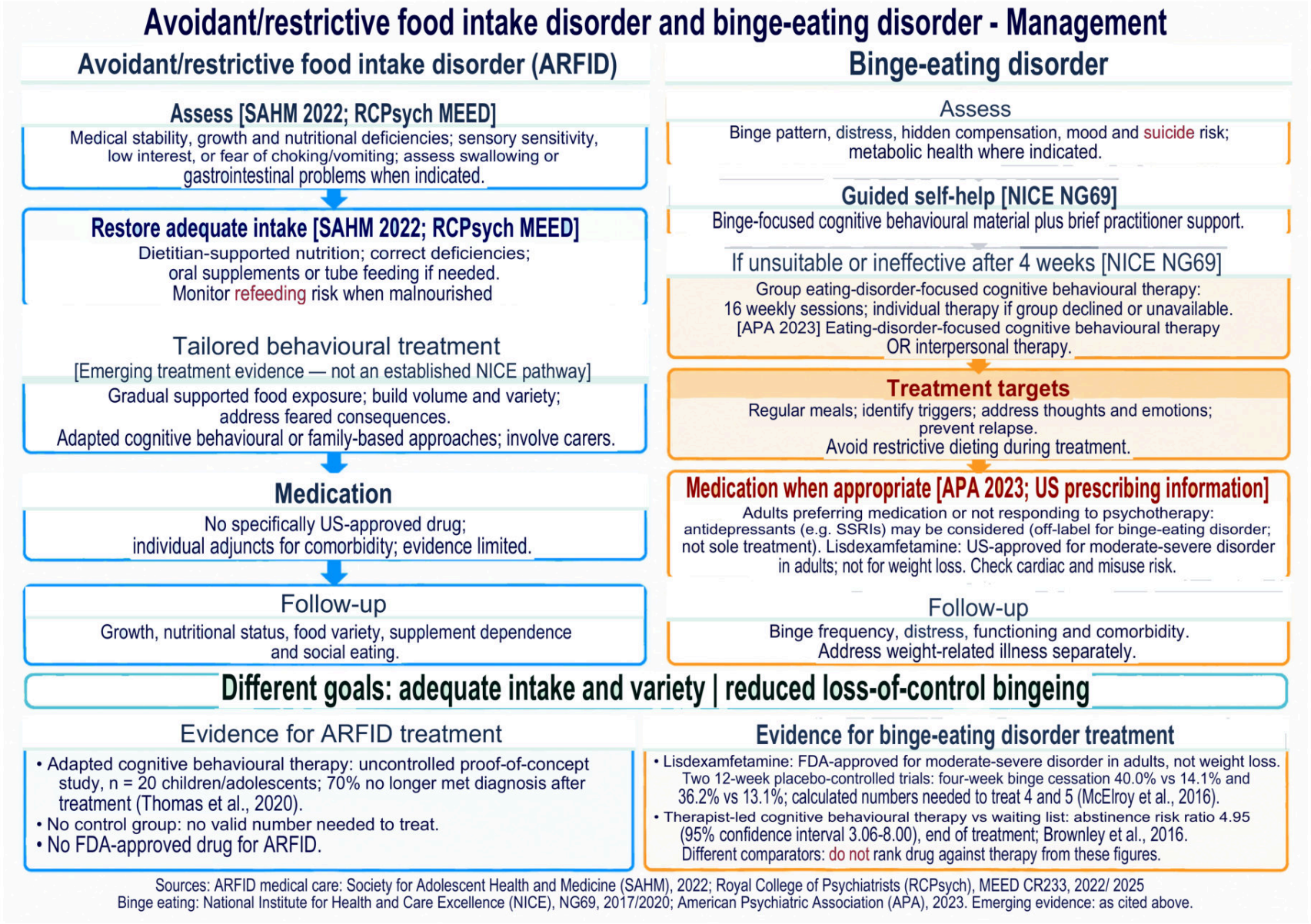
* Associated features: rapid eating; uncomfortable fullness; eating without hunger; eating alone from embarrassment; disgust, depression or guilt afterward

ICD-11: objective or subjective binges. DSM-5-TR: objectively large binges. Loss of control matters in both.

Summary, not full criteria. Sources: supplied manuals; exact page references appear on each disorder sheet.

Eating disorders - Diagnostic comparison. Read across the labelled contrasts to distinguish the related concepts.
Section companion: Eating, sleep and sexual disorders.

Credits and references: retained within the illustration.



Avoidant restrictive food intake disorder and binge-eating disorder - Management. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Eating, sleep and sexual disorders.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 1.9

Binge-eating disorder

Diagnosis, clinical assessment and the bulimia boundary

DSM-5-TR diagnostic core

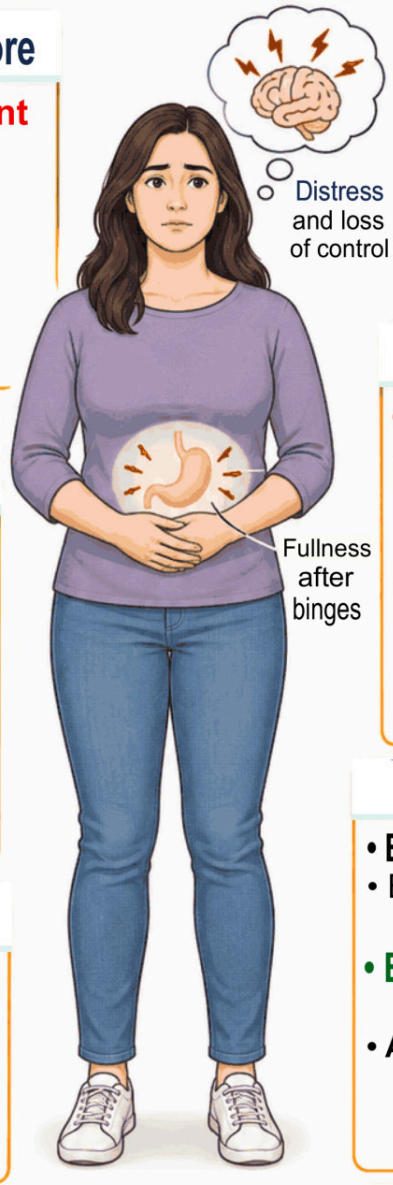
- Unusually large amount in a discrete period + loss of control.
- Marked distress.
- At least once weekly for 3 months.

Three or more associated features

- Rapid eating.
- Uncomfortably full.
- Eating when not hungry.
- Eating alone from embarrassment.
- Disgust, depression or guilt afterward.

Exclusion

- No recurrent inappropriate compensation.
- Not exclusively during anorexia nervosa or bulimia nervosa.



Clinical clues

- Secretive episodes, distress, uncomfortable fullness.
- Weight can vary; obesity is not required.

Assessment and tests

- No diagnostic blood test.
- Assess weight trajectory, blood pressure, mood and eating pattern.
- Glucose or glycated haemoglobin, lipids and liver tests when metabolic risk warrants.

Boundary with bulimia

- Both: loss-of-control binges.
- Bulimia: recurrent compensation.
- Binge-eating disorder: no regular compensation.
- Ask about fasting and driven exercise, not only vomiting.

ICD-11 allows objectively or subjectively large binges; DSM-5-TR requires objectively large binges. This supplied ICD-10 manual has no separate criteria set.

Classification

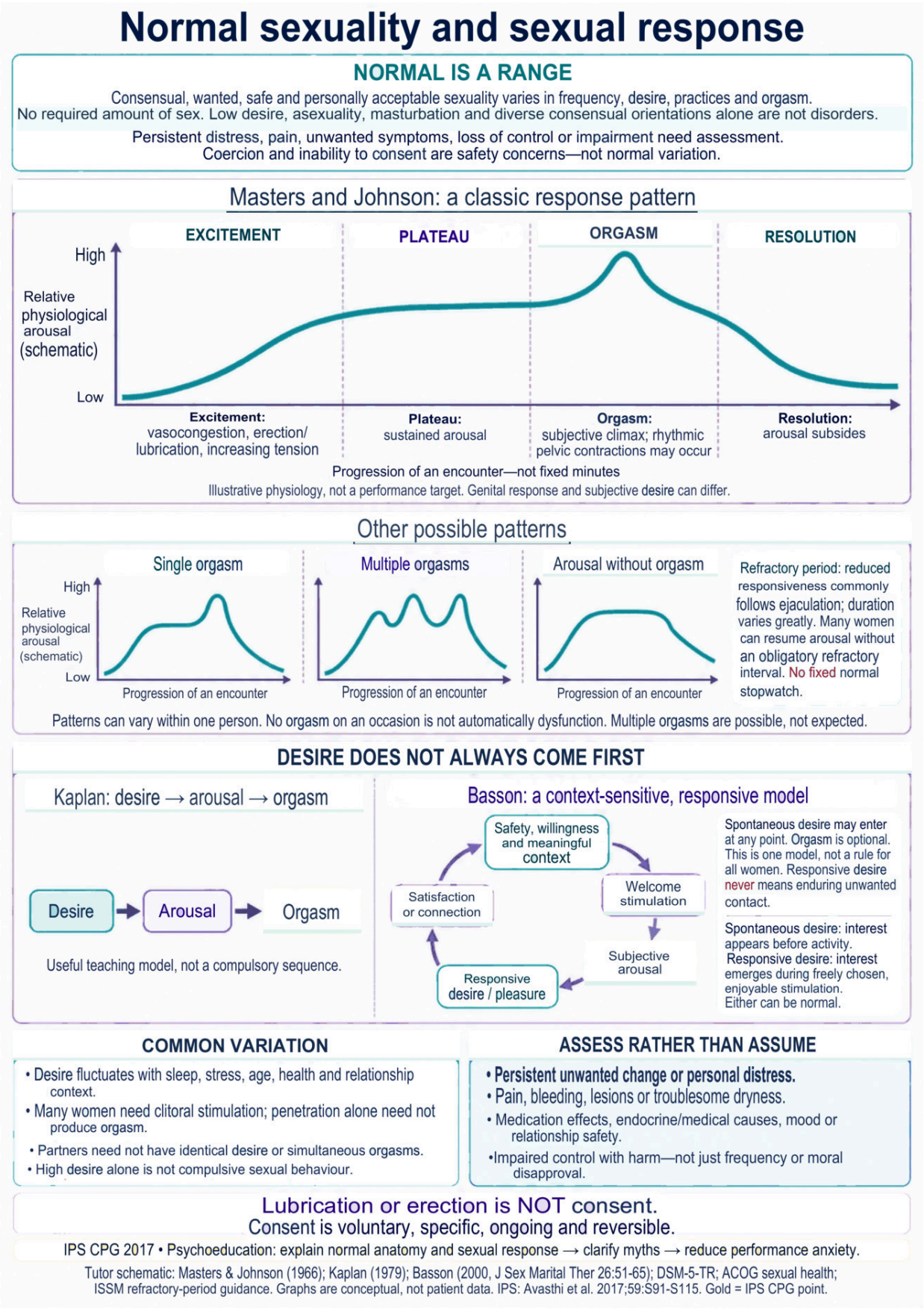
- Feeding and eating disorder, not classified as a behavioural addiction.

Diagnostic summary: supplied DSM-5-TR PDF 570 and ICD-11 PDF 425. Assessment labels are tutor additions. No physical sign is required.

Binge-eating disorder - Clinical summary. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Eating, sleep and sexual disorders.

Credits and references: retained within the illustration.

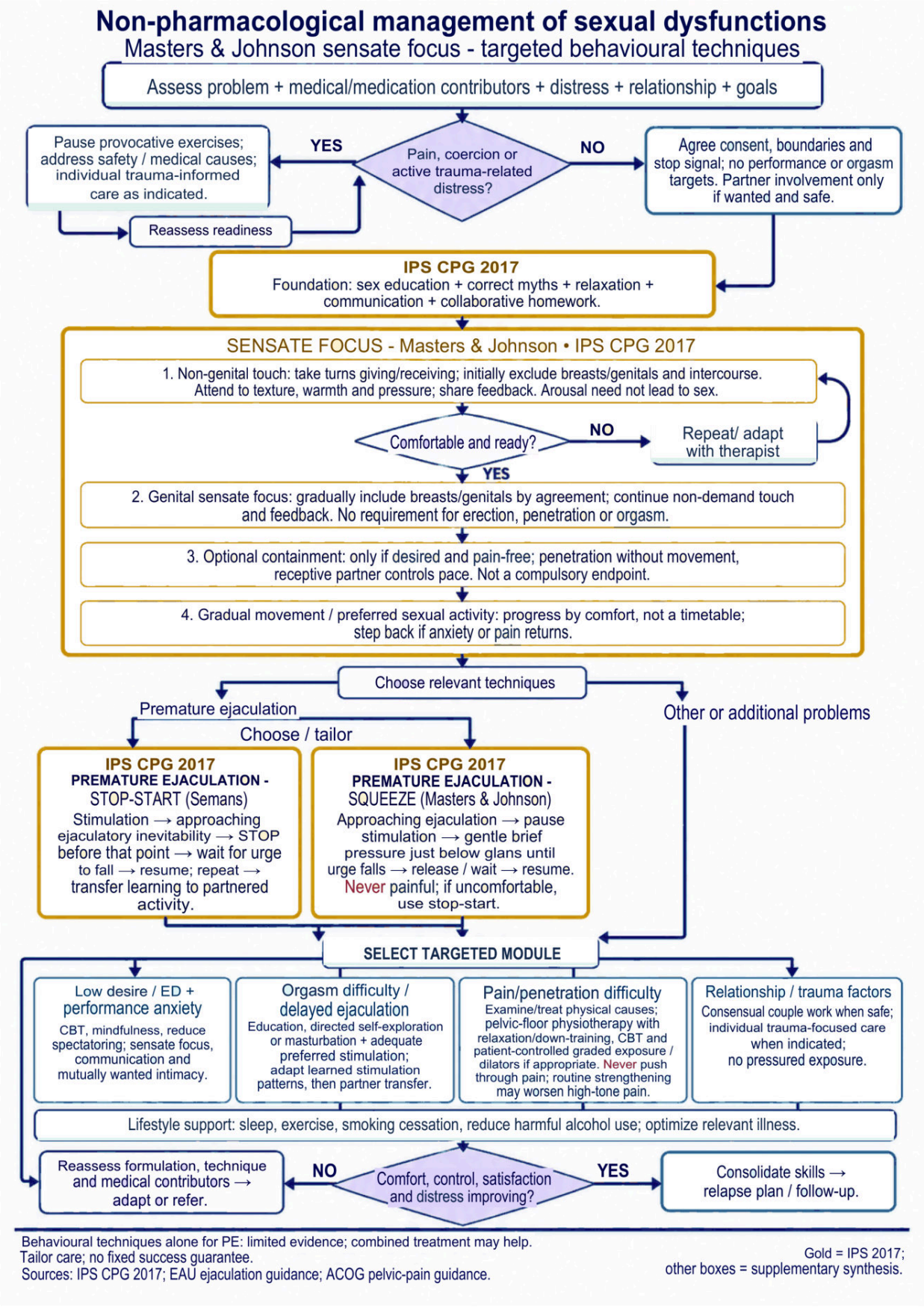
WEAVE 100 · PAPER II · 3.2



Normal sexuality - Response and variation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Eating, sleep and sexual disorders.

Credits and references: retained within the illustration.

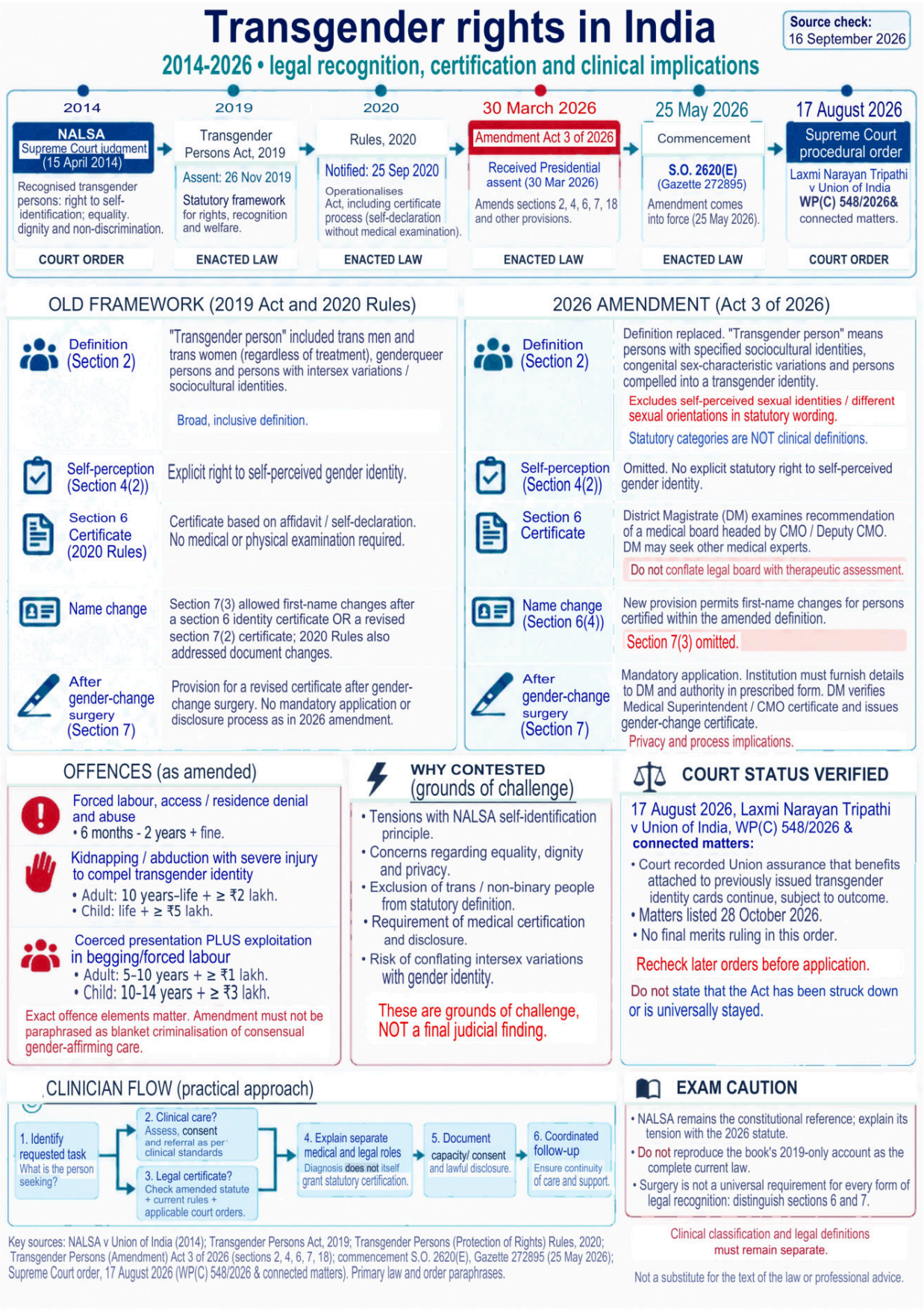
WEAVE 100 · PAPER II · 3.4



Sexual dysfunctions - Sensate focus and non-pharmacological management. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Eating, sleep and sexual disorders.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 4.2



Transgender rights in India - Legal changes 2026. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Eating, sleep and sexual disorders.

Credits and references: retained within the illustration.

Gender incongruence and gender dysphoria

ICD-10 • ICD-11 • DSM-5-TR — diagnostic comparison

SAME PEOPLE
DIFFERENT SYSTEMS
CLINICAL PRECISION
BETTER CARE

	ICD-10 (WHO, 1992) F64.0 Transsexualism	ICD-11 (WHO, ICD-11) HA60 Gender incongruence of adolescence and adulthood	DSM-5-TR (APA, 2022) Gender dysphoria
Status / classification	Mental and behavioural disorders Gender identity disorders (F64)	Conditions related to sexual health NOT a mental disorder	Psychiatric diagnostic category Gender dysphoria
Core description	A desire to live and be accepted as the other sex, usually accompanied by discomfort with one's anatomical sex and a wish for physical transition.	A marked and persistent incongruence between an individual's experienced gender and the assigned sex.	A marked incongruence between one's experienced gender and assigned sex, associated with clinically significant distress or impairment.
Key requirements	Clear desire to live and be accepted as the other sex. Usually discomfort with anatomical sex and wish for physical transition.	• Marked, persistent gender incongruence. • Puberty must have begun. • Desire for transition is common, but hormones or surgery are not mandatory. • Gender-variant behaviour alone is insufficient.	In adolescents and adults: at least 2 of 6 specified features, for at least 6 months, PLUS clinically significant distress OR impairment in social, occupational or other important areas of functioning.
Duration	At least 2 years.	No fixed duration for adults in the WHO category description.	At least 6 months.
Distress / impairment	No separate mandatory distress or impairment criterion (not required in the diagnostic description).	Distress is not required for the diagnosis.	Clinically significant distress OR impairment is required.
Transition	Wish for physical transition usually present.	Desire for transition common, but not mandatory. Hormones or surgery not required for diagnosis.	Desire for medical transition may be present, but is not required for diagnosis.
Exclusions / clarifications	Should not be a symptom of another mental disorder. Should not be associated with intersex conditions, genetic disorders or sex-chromosome abnormalities.	Gender-variant behaviour alone is insufficient to make the diagnosis.	Can include nonbinary gender experiences. Specifiers: with disorder/difference of sex development; posttransition.
Childhood criteria (prepubertal)	**ICD-10 - F64.2**	**ICD-11 - HA61**	DSM-5-TR - Children
Core description	Persistent and intense distress about assigned sex and a desire or insistence to be the other sex.	Marked gender incongruence in prepubertal children, for about 2 years. Includes a desire for a different gender, a mismatch between experienced gender and anatomical or anticipated sex characteristics, and corresponding play and peer patterns.	At least 6 of 8 specified features,for at least 6 months, MUST include a desire or insistence of being another gender, PLUS clinically significant distress or impairment.
Key features	Preferences alone are insufficient to make the diagnosis.	Preferences alone are insufficient to make the diagnosis.	Eight domains: (1) identity; (2) clothing; (3) fantasy roles; (4) toys/activities; (5) playmates; (6) rejection of assigned-gender activities; (7) dislike of anatomy; (8) desired sex characteristics.
Duration	No specific duration stated; do not apply the 2-year adult rule.	For about 2 years.	At least 6 months.

DSM-5-TR: six adolescent/adult features (paraphrased); any 2 for ≥6 months + distress/impairment

1. Mismatch with current/anticipated sex characteristics
2. Desire to remove/prevent these characteristics
3. Desire for another gender's sex characteristics
4. Desire to be another/alternative gender
5. Desire to be treated as that gender
6. Conviction of corresponding feelings/reactions

Key points Identity ≠ diagnosis ≠ eligibility for a particular intervention. Gender expression ≠ sexual orientation. ICD-11 does not require distress; DSM does.

Sources: WHO. ICD-10. Classifications and Diagnostic Guidelines (CDDG). F64, pp 168-169. WHO. ICD-11. HA60, HA61. APA. DSM-5-TR. 2022. APA criteria summary.

Paraphrased criteria, reviewed 16 Sep 2026

Gender incongruence - ICD-10 ICD-11 DSM-5-TR comparison. Read across the labelled contrasts to distinguish the related concepts. Section companion: Eating, sleep and sexual disorders.

Credits and references: retained within the illustration.

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

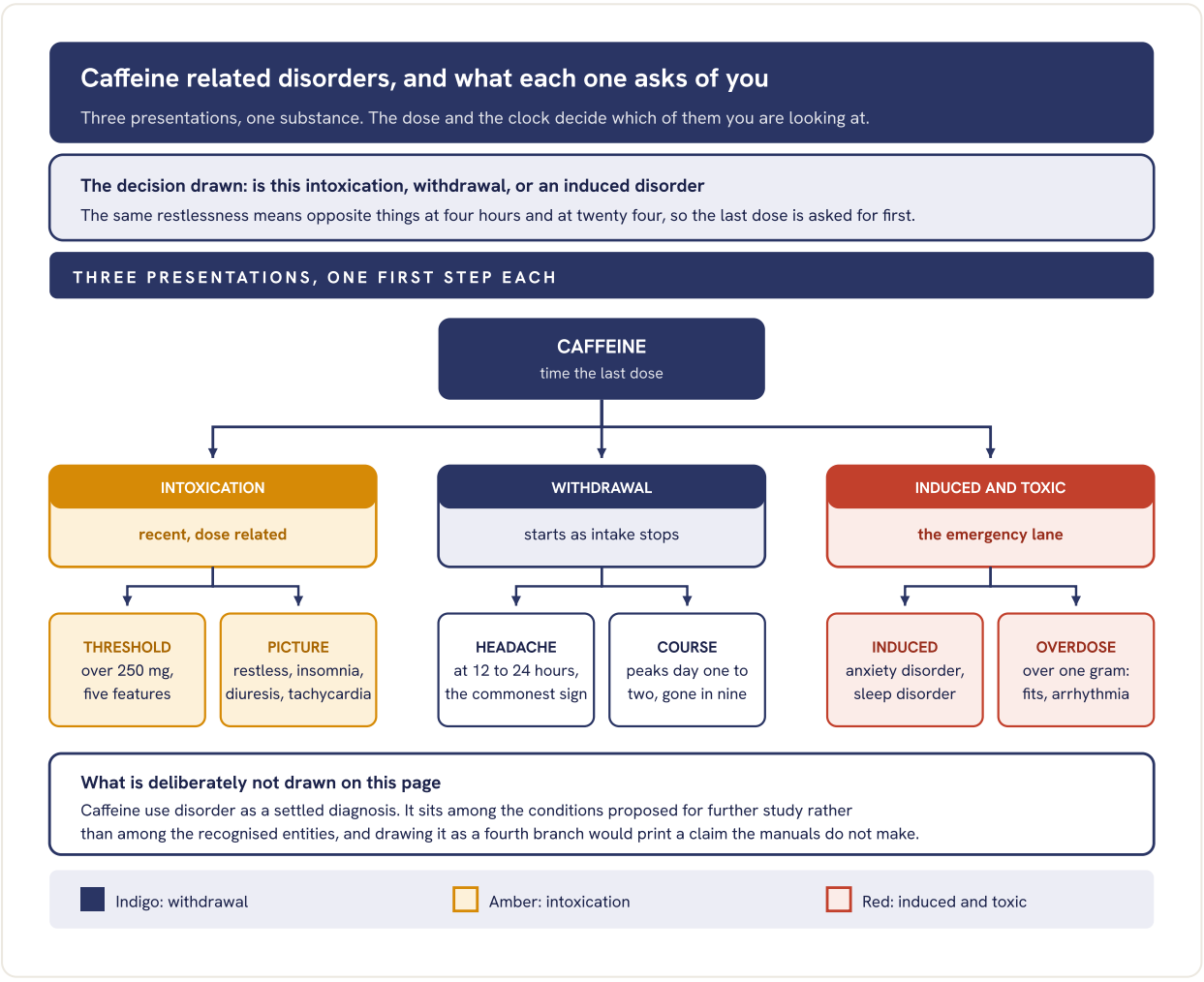


Fig 010.1 The three presentations set against the clock since the last dose, with the toxic lane kept separate because it is the only one that is managed in a resuscitation bay.

- **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

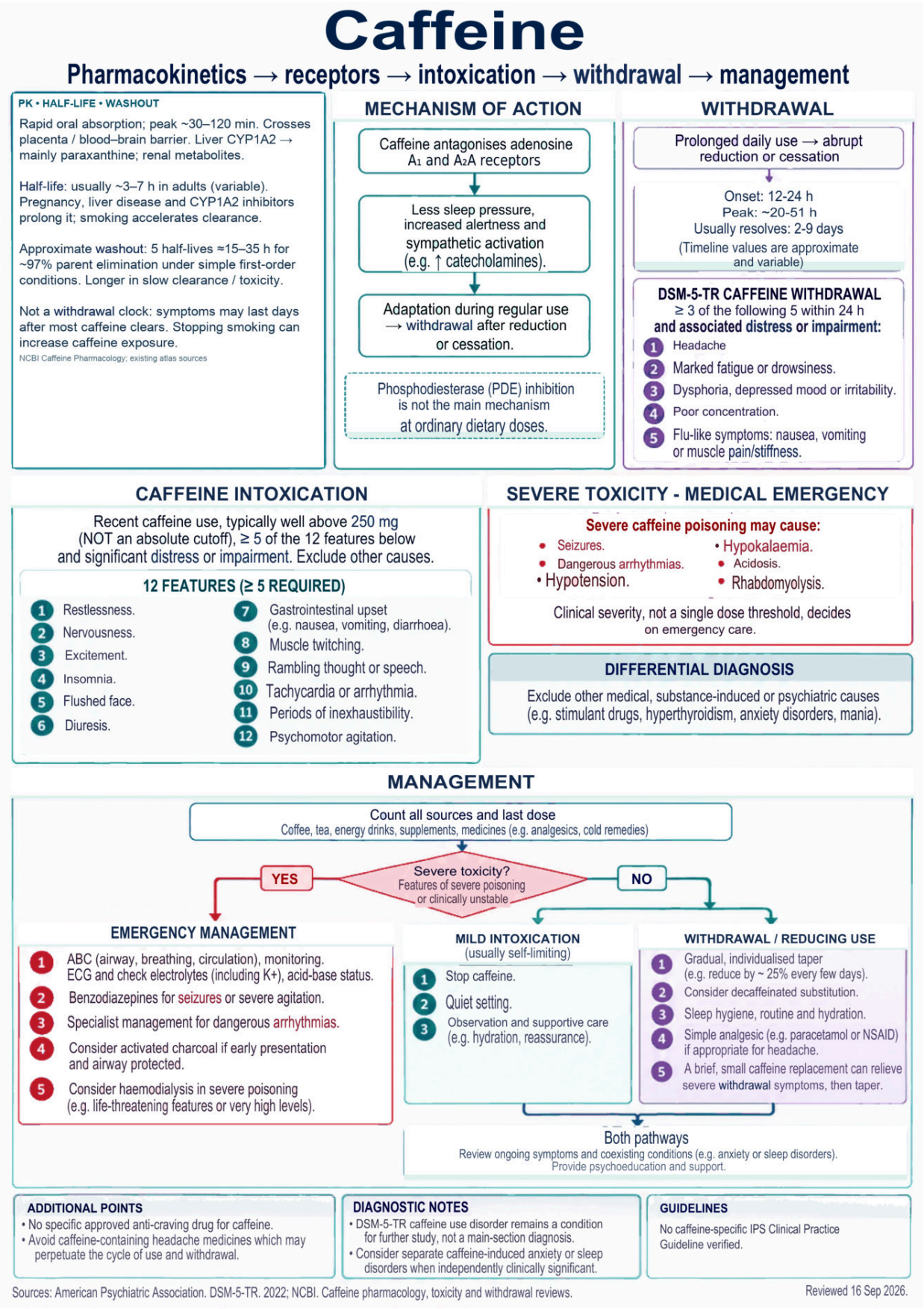
Do not diagnose an anxiety disorder in a heavy user without first timing the caffeine. Panic attacks are readily provoked at high intake, and the treatment is a taper, not an antidepressant.

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

WEAVE 100 · PAPER II · 10.1



Caffeine - Pharmacology intoxication withdrawal and management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-10.

Credits and references: retained within the illustration.

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

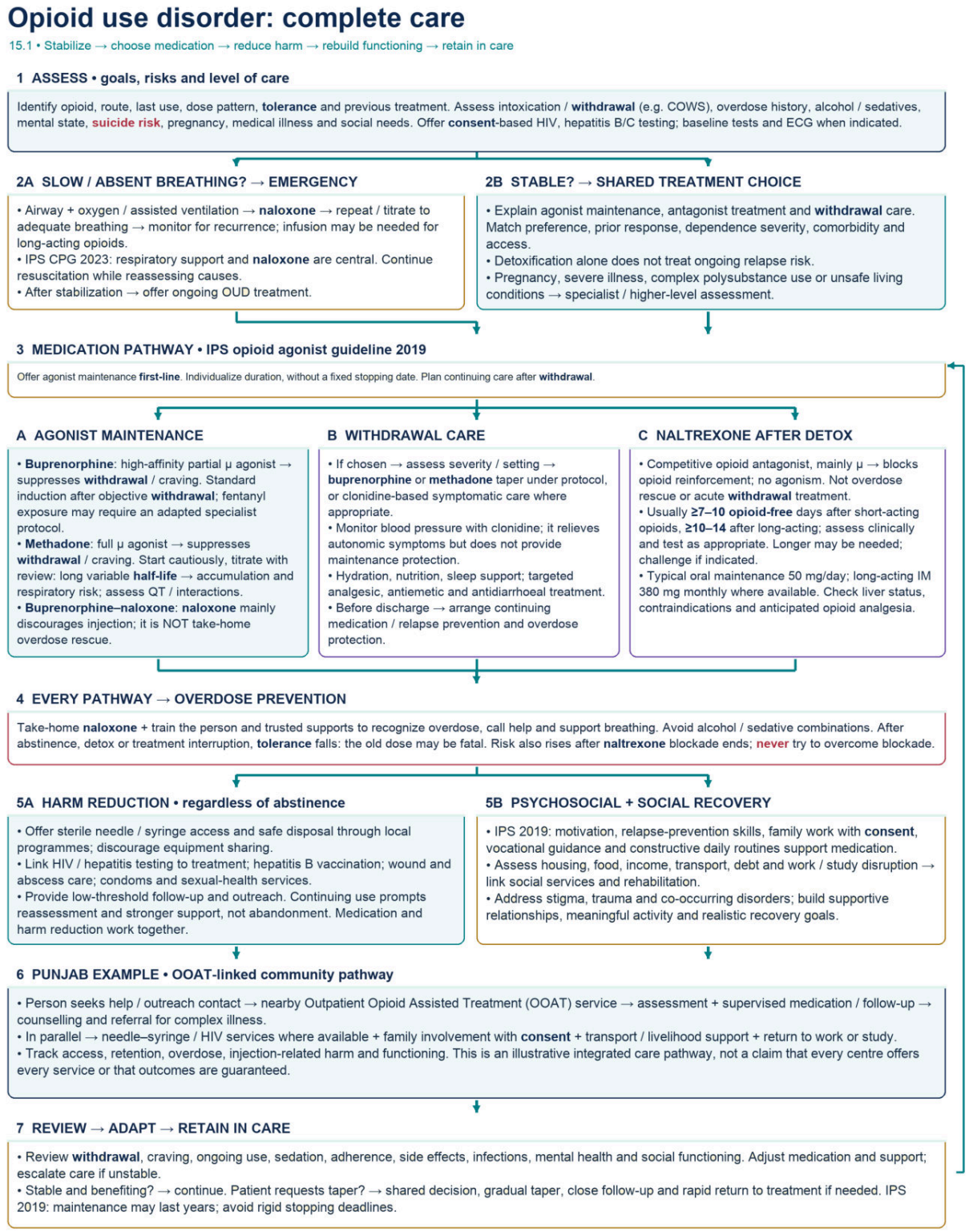
The Act licenses and supplies; it does not fund a service, train a team or create demand reduction on its own. It does not touch alcohol or tobacco. Consumption of a narcotic drug remains an offence, and the immunity for an addict who volunteers for treatment is conditional, not a general decriminalisation.

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

WEAVE 100 · PAPER II · 15.1



Opioid addiction - Complete management and Punjab community care. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-15.

Credits and references: retained within the illustration.

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.

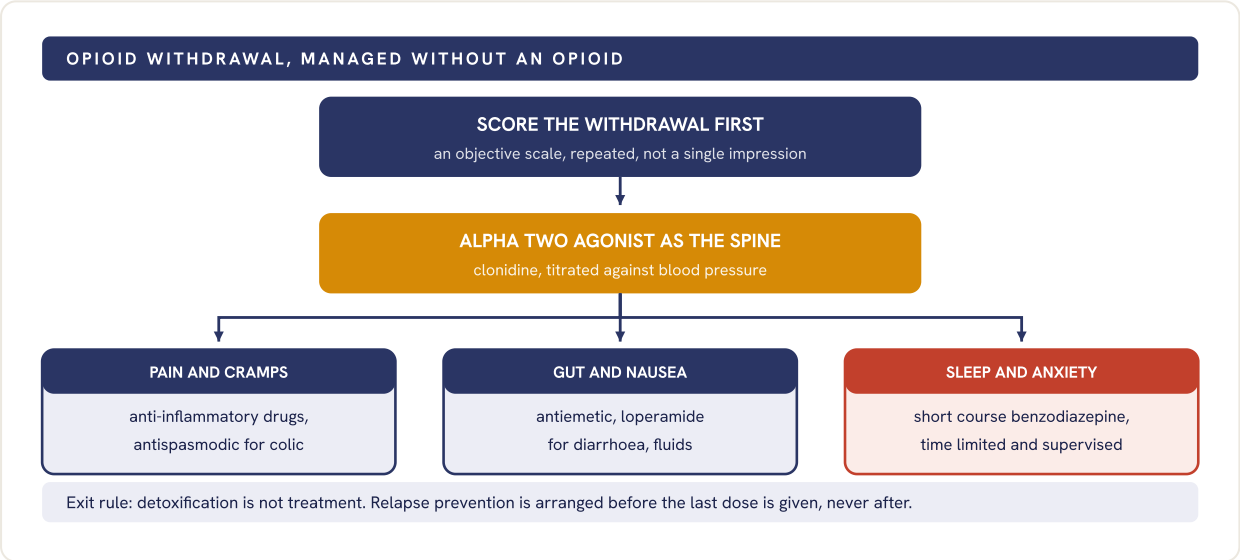


Fig 016.1 The alpha two agonist carries the syndrome and the three lanes mop up what it leaves, with the red lane marked because that is where iatrogenic dependence begins.

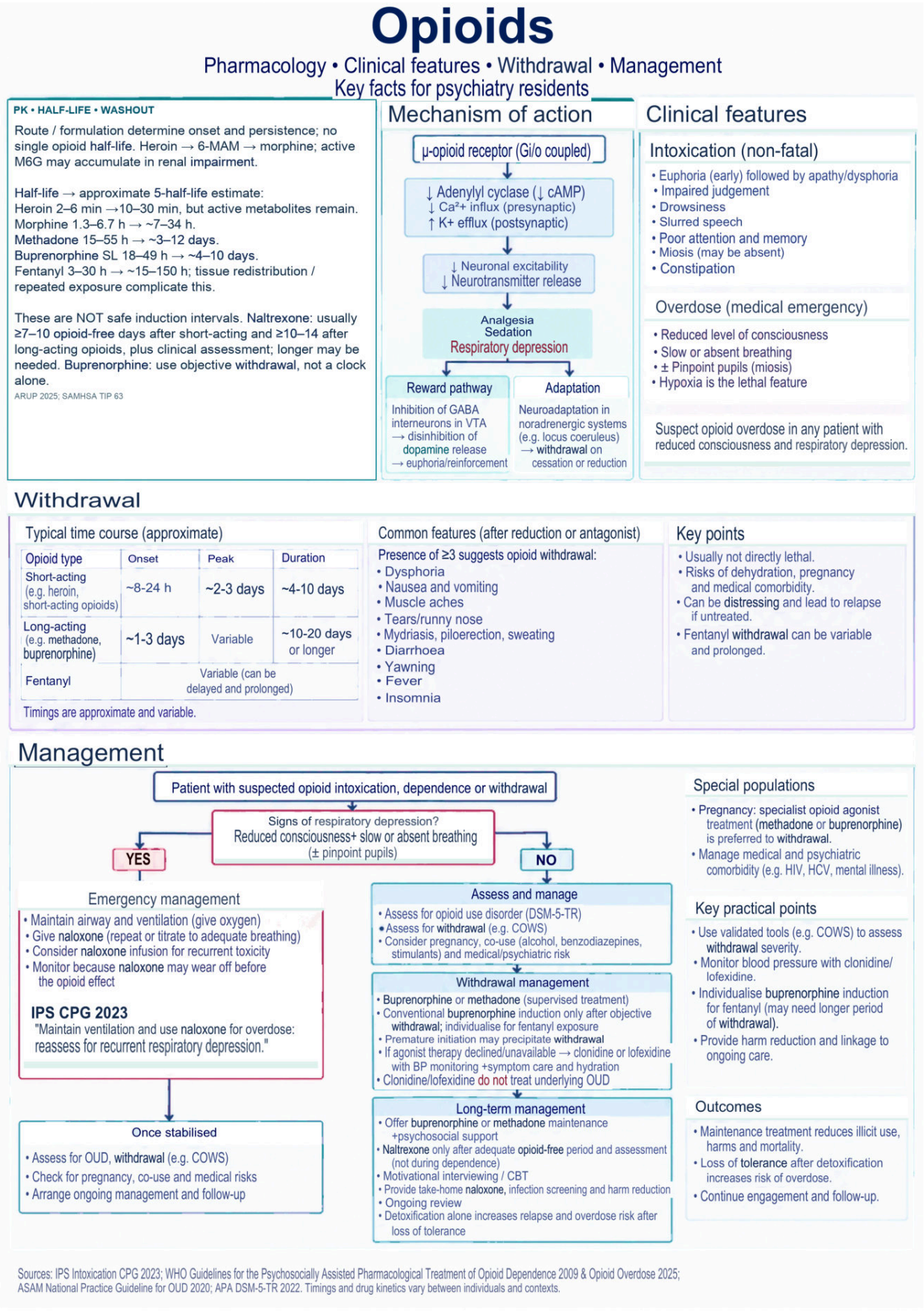
- **Why clonidine works.** Chronic opioid use disinhibits the locus coeruleus, and withdrawal releases a noradrenergic storm. An alpha two agonist suppresses it directly, which is why it treats sweating, cramps and restlessness but does very little for craving.

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

WEAVE 100 · PAPER II · 16.1



Opioids - Pharmacology intoxication withdrawal and management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-16.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 16.2

Opioids, naloxone & naltrexone

Receptor mechanisms • pharmacokinetics • overdose rescue • sustained blockade

PHARMACOKINETICS • not one class half-life

• Route and formulation determine onset. IV is rapid; oral absorption and first-pass metabolism vary. Lipid solubility affects brain entry.
• Heroin → 6-MAM → morphine. Morphine is glucuronidated; active M6G can accumulate in renal **impairment**.
• Fentanyl: lipophilic; repeat exposure may prolong effects. **Methadone**: long, variable **half-life** and accumulation. **Buprenorphine**: high affinity and slow receptor dissociation.

RECEPTORS • Gi/o-coupled

• μ (mu): analgesia, reward, **respiratory depression**, miosis and reduced gut motility.
• κ (kappa): analgesia and dysphoria.
• δ (delta): modulation of analgesia and affect.
• **Methadone** / morphine: full μ agonists.
• **Buprenorphine**: partial μ agonist, high affinity.
• **Naloxone** / **naltrexone**: antagonists; no agonist activity. Effects depend on drug, dose and context.

AT THE RECEPTOR • schematic, not a molecular structure



Receptor activation → Gi/o
↓ adenylyl cyclase / cAMP
↓ presynaptic Ca2+ entry; ↑ K+ conductance
↓ transmitter release / hyperpolarization

Both: competitive blockade; no receptor activation
Naloxone → rapid opioid overdose reversal
Naltrexone → sustained blockade / relapse prevention
Naloxone does NOT reverse alcohol or benzodiazepines

Reward circuit: μ activation on VTA GABA neurons → less GABA inhibition → **dopamine** neurons disinhibited → increased nucleus accumbens signalling.
Respiratory depression reflects opioid effects on brainstem breathing networks.

NALOXONE • rapid rescue, short action

• IV onset usually within 2 minutes; effect often shorter than the opioid. Adult plasma **half-life** roughly 30–80 minutes. Hepatic glucuronidation; urinary metabolites.
• IV / IM / intranasal routes; oral systemic availability is poor. Formulations and doses are route-specific.
• Can precipitate acute **withdrawal**: agitation, pain, vomiting and autonomic symptoms. Titrate to adequate ventilation, not necessarily full wakefulness.

TOLERANCE LOST → OVERDOSE RISK

• Repeated exposure → adaptations and **tolerance**.
• Abstinence / detoxification / interrupted treatment → **tolerance** falls.
• Return to a previously tolerated dose → greater opioid effect → respiratory depression / fatal overdose.
• Alcohol or benzodiazepines increase risk. **Tolerance** differs by effect and is **never** reliable protection. Risk also rises after **naltrexone** blockade ends.

SUSPECTED OVERDOSE • ADULT EMERGENCY PATHWAY

1 Recognize and support breathing

Reduced consciousness + slow / absent breathing ± pinpoint pupils → call emergency help; airway, oxygen and assisted ventilation. CPR when indicated. Check glucose and co-ingestants; **do not** delay for toxicology results.

2 Naloxone + ongoing respiratory support • IPS CPG 2023

IPS: 0.2–0.4 mg IV initially; 1–2 mg for apnoea / cardiorespiratory arrest. Repeat every 2–3 minutes and titrate to ventilation. IPS describes escalation to 10 mg; absent response requires diagnostic reassessment and specialist escalation, not abandonment of resuscitation.

3A Response sustained?

• YES → continue monitoring consciousness, breathing and oxygenation; assess opioid duration and other causes.
• Nasal rescue products: follow device label; e.g. 4 mg single spray, repeat with a new device every 2–3 min if needed while help arrives.

3B Recurrent depression / long-acting opioid?

• YES → repeat **naloxone**; monitored infusion may be required. IPS: starting hourly infusion ≈ two-thirds of the effective reversal bolus, then titrate clinically.
• Continue ventilation; high-affinity opioids may need prolonged specialist management.

4 Observe → reassess → connect to treatment • IPS CPG 2023

IPS recommends observation for 4–6 hours after stopping **naloxone** infusion; longer if clinically indicated. **No universal** discharge clock for all overdoses. After stabilization: overdose education + take-home **naloxone** + offer ongoing opioid-use-disorder treatment.

NALOXONE CHALLENGE ≠ overdose rescue

• Before **naltrexone**, a clinician may use a monitored challenge to assess residual opioid dependence when appropriate. It is not a test for opioid use disorder.
• **Do not** challenge during active **withdrawal**. A negative test does not guarantee safe **naltrexone** induction; **opioid-free** interval, clinical assessment and appropriate urine testing remain essential.

NALTREXONE • mechanism and purpose

• Competitive opioid blockade, mainly μ; no agonism.
• Opioids: blocks reinforcing effects after safe induction.
• Alcohol: reduces endogenous-opioid-mediated reward; not an alcohol–disulfiram reaction.
• Oral / long-acting IM; active metabolite 6β-naltrexol.
• NOT overdose rescue. Starting before opioids clear can precipitate **withdrawal**; assess **opioid-free** interval.

Sources: IPS intoxication CPG (Sarkar et al., IJP 2023), pp204–205; DailyMed **naloxone** injection / NARCAN labels; SAMHSA TIP 63 (2021). Gold = verified IPS recommendations; other pharmacology = supplementary explanation.

16.2 • A4 / 300 DPI • Educational clinical reference • Doses require route- and patient-specific judgment

Opioids and naloxone - Receptors pharmacokinetics and overdose reversal. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-16.

Credits and references: retained within the illustration.

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

WEAVE 100 · PAPER II · 17.1

New psychoactive substances

17.1 • Mechanisms, chemical names, clinical features and treatment

DEFINITION • "new" means newly encountered, not newly invented

Substances of abuse outside the 1961 / 1971 international drug conventions that threaten public health. Historical examples may since have become controlled. Plant origin is not a mechanism or assurance of safety. The book's five rows are expanded below; plant-based substances and sedatives are separate.

1 SYNTHETIC CANNABINOIDS

- Laboratory-made chemicals: many strongly and fully activate **cannabinoid** type one receptors. Natural delta-nine-tetrahydrocannabinol is a partial agonist.
- Effects: anxiety, agitation, paranoia, psychosis; **seizures**, vomiting, rapid heartbeat. Severe cases: coma, kidney injury or cardiac complications.
- Routine urine cannabis tests often miss these agents. Dependence and **withdrawal** can occur.

2 SYNTHETIC CATHINONES

- Mephedrone is a laboratory-made relative of cathinone from khat. Transporter effects increase release and/or reduce reuptake of **dopamine**, **noradrenaline** and **serotonin**; compounds differ.
- Effects: energy / euphoria → insomnia, agitation, paranoia. Severe: overheating, **seizures**, muscle breakdown.
- After use: fatigue, low mood, sleep changes and craving; assess **suicide risk**.

3 HALLUCINOGENIC PHENETHYLAMINES

- Mescaline is the natural cactus relative, not the newer synthetic drug. These examples mainly stimulate **serotonin** type two A receptors.
- "Two C" names a family, not one substance. "Two C B" is 4-bromo-2,5-dimethoxyphenethylamine; the letter distinguishes a chemical member.
- "N-methoxybenzyl" relatives, written NBOMe in the book, have an additional chemical group. "Twenty-five I" is related to the iodine-containing two C compound. Many are highly potent.
- Effects: altered perception, hallucinations, panic. Severe toxicity: vasoconstriction, overheating, **seizures**.

4 SYNTHETIC OPIOIDS

- Fentanyl is an established controlled medicine; newer fentanyl-related compounds are not one uniform drug. Nitazenes are a chemically distinct opioid family.
- Mu opioid receptor activation → analgesia / reward, but also respiratory depression. Potency and duration vary substantially.
- Poisoning: hard to wake + slow / absent breathing ± pinpoint pupils. Unexpected exposure and lost **tolerance** increase overdose risk.
- Breathing support → **naloxone** → reassess / repeat; infusion and prolonged monitoring may be needed. **Naloxone** can reverse these opioids.



KHAT • cathinone

Memory cue • schematic



CACTUS • mescaline

Memory cue • schematic



IBOGA • root bark

Memory cue • schematic



AYAHUASCA • vine + leaves

Memory cue • schematic

5 PLANT-BASED • three different pharmacological stories

KRATOM • Mitragyna speciosa

- Leaves contain **mitragynine** and 7-hydroxy**mitragynine**. Opioid-receptor activity plus other actions → stimulation or analgesia / sedation, depending on exposure and product.
- Nausea, constipation, dependence; **withdrawal** can cause aches, sweating, irritability, insomnia and gastrointestinal upset. Serious toxicity is possible.
- Not an established replacement for **buprenorphine** or **methadone**.
- LEAF → opioid-like effects → dependence.

IBOGAINE • Tabernanthe iboga

- Root-bark alkaloid; active metabolite noribogaine. Acts on several neurotransmitter systems → prolonged waking dream-like experiences.
- Investigated for addiction, but not established routine treatment.
- Key danger: delayed cardiac electrical recovery → potentially fatal ventricular **arrhythmias**. Vomiting / electrolyte loss can worsen risk. Ataxia may occur.
- ROOT → waking dream → cardiac toxicity.

AYAHUASCA • a preparation

- Psychotria viridis leaves commonly supply N,N-**dimethyltryptamine**: hallucinogenic **serotonin** type two A receptor stimulation.
- Banisteriopsis caapi vine supplies **harmine**, **harmaline** and **tetrahydroharmine**.
- **Harmine** / **harmaline** reversibly inhibit monoamine oxidase type A in gut / liver → less first-pass breakdown → oral **dimethyltryptamine** reaches brain. **Tetrahydroharmine** also inhibits **serotonin** reuptake.
- VINE + LEAVES → reduced breakdown + hallucinations.

6 SEDATIVES • etizolam

- A thienodiazepine with benzodiazepine-like effects. Enhances **gamma-aminobutyric** acid at its type A receptor's benzodiazepine site → increased inhibitory signalling.
- Use: sedation, slurred speech, poor coordination, amnesia. Opioids / alcohol increase breathing risk.
- Abrupt cessation after dependence: anxiety, insomnia, tremor, **seizures** or delirium.
- Poisoning → support breathing / monitor. Dependence → supervised gradual taper. Flumazenil is not routine in unknown or mixed overdose.

AYAHUASCA RISKS + ADDITIONAL FAMILY

- Ayahuasca: vivid imagery, emotional intensity, vomiting; panic, agitation, mania or psychosis may occur. **Serotonin**-enhancing co-exposures can cause dangerous toxicity: fever, sweating, agitation, exaggerated reflexes and muscle contractions.
- Book addition: ketamine-type dissociatives, e.g. methoxetamine, block N-methyl-D-aspartate receptors → detachment, altered perception, nystagmus and poor coordination.
- Plant-based and sedative are NOT one mechanism.

TREAT THE CLINICAL SYNDROME • do not wait for a positive drug screen

ASSESS → STABILIZE • Indian Psychiatric Society guidance, 2023

History of product, timing, route and co-exposures → airway, breathing, circulation, glucose, temperature and mental state. Routine tests may miss newer substances. Unknown potency / interactions matter; involve critical care and psychiatric services when indicated.

AGITATION / SEIZURES / FEVER

Benzodiazepines with monitoring → active cooling and guided fluids as needed → assess muscle breakdown, kidney injury and **serotonin** toxicity. Severe illness → emergency care.

SLOW BREATHING / SEDATION

Airway / ventilation → **naloxone** if opioid toxicity suspected → repeat assessment and monitored escalation. **Naloxone** does not reverse pure **cannabinoid** or sedative effects.

IBOGAINE / CARDIAC SIGNS

Electrocardiogram + continuous cardiac monitoring + electrolytes → correct abnormalities and treat **arrhythmias** in specialist care. Fainting or palpitations need urgent assessment.

RECOVERY → REASSESS → CONTINUING CARE

Observe to clinical stability → review mental state, **withdrawal** and **suicide risk** → offer harm reduction and addiction follow-up. No reliable class-wide **half-life** or washout: identify the compound; detection, clearance and recovery differ.

Sources: Weave 100, Paper II, Question 17; United Nations Office on Drugs and Crime; European Union Drugs Agency **cannabinoid** and phenethylamine reports; World Health Organization etizolam review; United States Food and Drug Administration kratom information; Indian Psychiatric Society emergency intoxication guideline (2023); published ibogaine and ayahuasca evidence. Gold = verified Indian Psychiatric Society points.

WEAVE 100+ • PAPER II • QUESTION 17 • A4 PORTRAIT • 300 DPI

New psychoactive substances - Classification clinical features and management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-17.

Credits and references: retained within the illustration.

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.

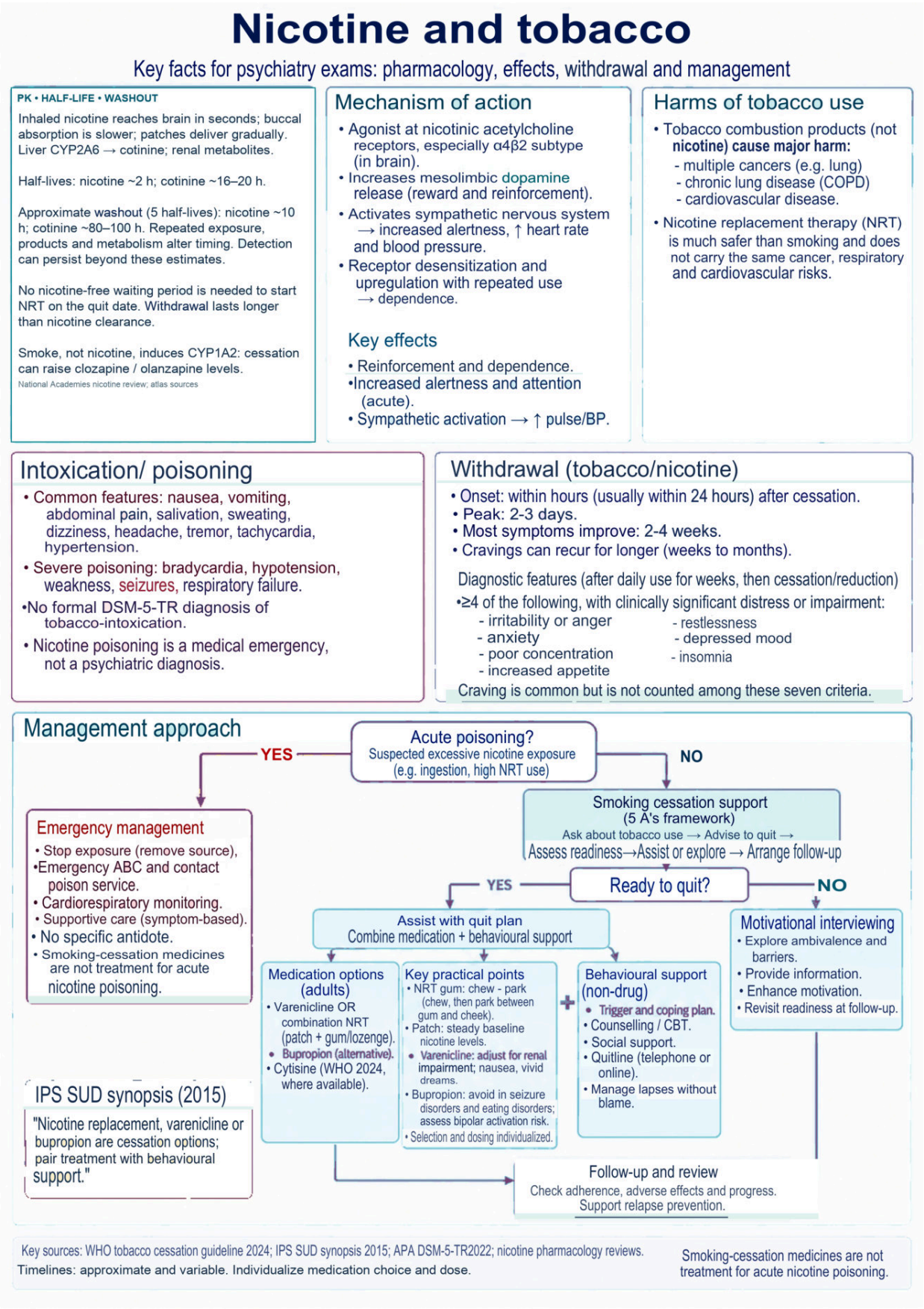
WHAT EARNS THE MARKS

- **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

WEAVE 100 · PAPER II · 18.1



Nicotine and tobacco - Pharmacology intoxication withdrawal and management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-18.

Credits and references: retained within the illustration.

Substance use: ICD-10 vs ICD-11 vs DSM-5-TR

Dependence and use-disorder criteria • substance-specific exceptions • paraphrased exam reference

ICD-10 • Dependence syndrome

GATE: at least 3 of 6 features together at some time during the previous year.

1 Strong desire or compulsion.
2 Difficulty controlling onset, stopping or amount.
3 **Withdrawal**, or use to prevent / relieve it.
4 **Tolerance**: more needed or reduced effect.
5 Neglect of other interests; increased time obtaining, using or recovering.
6 Continued use despite clear harm, with awareness of that harm.

Withdrawal and **tolerance** are NOT individually compulsory.

Harmful use is a separate category: actual physical or mental health damage. Social disapproval alone is insufficient.

Timing here follows WHO clinical guidelines (CDDG); **do not** mix this wording with research criteria.

ICD-11 • Substance dependence

GATE: at least 2 of 3 central domains.

1 IMPAIRED CONTROL
Over onset, frequency, intensity, duration, stopping or context.

2 INCREASING PRIORITY
Use takes precedence over health, activities and responsibilities; persists despite harm or negative consequences.

3 PHYSIOLOGICAL ADAPTATION
Tolerance OR **withdrawal** OR repeated use to prevent / relieve **withdrawal**. Count this as ONE domain.

Usually evident for at least 12 months; diagnosis may be made with continuous daily / almost-daily use for at least 3 months.

Harmful episode and harmful pattern are separate diagnoses. Physiological features apply only where relevant.

DSM-5-TR • Substance use disorder

GATE: problematic use causing significant **impairment** / **distress**, with **≥2** applicable criteria within 12 months.

IMPAIRED CONTROL
1 More / longer than intended.
2 Failed efforts or persistent wish to cut down.
3 Much time obtaining, using or recovering.
4 Craving.
SOCIAL **IMPAIRMENT**
5 Failure in major roles.
6 Continued use despite interpersonal problems.
7 Important activities reduced / abandoned.
RISKY USE
8 Repeated use in hazardous situations.
9 Continued use despite physical / mental harm.
PHARMACOLOGICAL
10 **Tolerance**. 11 **Withdrawal** / relief use.

Severity: mild 2–3 • moderate 4–5 • severe **≥6**.

WHAT CHANGES BY SUBSTANCE?

Teal = standard framework • Red = criterion omitted • Purple = classification exception • Blue = medical-use caveat

Alcohol • cannabis • opioids • sedatives • cocaine / stimulants • tobacco	DSM: usual 11-criterion framework. Tobacco **withdrawal** counts. Intoxication / **withdrawal** syndromes have separate substance-specific criteria.
PCP • other hallucinogens • inhalants	DSM: omit **withdrawal** (criterion 11); use the remaining 10. **Tolerance** remains. Diagnosis still **≥2**; severity thresholds do not change. This is a diagnostic rule, not proof that every cessation symptom is impossible.
Caffeine	DSM: caffeine use disorder is a condition for further study, not the routine **≥2**-of-11 diagnosis. Intoxication and **withdrawal** are recognized. ICD-11 has caffeine-related categories but no caffeine-dependence category; ICD-10 places caffeine under F15.
MDMA • ketamine / PCP • synthetic cannabinoids	DSM places MDMA with other hallucinogens and ketamine with phencyclidine-like substances. ICD-11 separates MDMA, dissociatives and synthetic **cannabinoids**. **Do not** transplant a DSM exception into ICD-11.
Appropriate prescribed opioid • sedative • stimulant treatment	DSM: **do not** count **tolerance** / **withdrawal** arising solely during appropriate medical treatment. Other criteria may still establish a disorder. ICD-11: physiology alone is insufficient; impaired control or increasing priority is also required.

Across systems: diagnose the pattern, not a positive drug screen or daily use alone. Class-specific symptoms do not automatically add new dependence criteria. Gold is reserved for verified IPS treatment guidance; these diagnostic criteria come from WHO / APA.

Sources: WHO ICD-10 CDDG, F1x.2 (1992), p70; WHO ICD-11 CDDR (2024), pp463–467; APA DSM-5-TR (2022), pp545–548 and class-specific chapters. • 11.1 • A4 / 300 DPI

Substance use disorders - ICD-10 ICD-11 DSM-5-TR comparison. Read across the labelled contrasts to distinguish the related concepts. Section companion: Substance use: opioids, other drugs and treatment.

Credits and references: retained within the illustration.

Substances × diagnostic categories

ICD-11 applicability matrix • ICD-10 code key • withdrawal and mood-disorder distinctions

Tick = category listed in WHO ICD-11 CDDR (2024). Cross = no category listed in these tables; NOT proof that a symptom can never occur. This is a classification map, not a frequency or causal-risk table.

SUBSTANCE / ICD-11 FAMILY		Depend-ence	Intoxi-cation	With-drawal	Delirium	Psychotic disorder	Mood*	Anxiety	OCD / related	Impulse control	Amnestic	Dementia
Alcohol	6C40	✓	✓	✓	✓	✓	✓	✓	✗	✗	✓	✓
Cannabis	6C41	✓	✓	✓	✓	✓	✓	✓	✗	✗	✗	✗
Synthetic cannabinoids	6C42	✓	✓	✓	✓	✓	✓	✓	✗	✗	✗	✗
Opioids	6C43	✓	✓	✓	✓	✓	✓	✓	✗	✗	✗	✗
Sedatives / hypnotics / anxiolytics	6C44	✓	✓	✓	✓	✓	✓	✓	✗	✗	✓	✓
Cocaine	6C45	✓	✓	✓	✓	✓	✓	✓	✓	✓	✗	✗
Amphetamine-type stimulants	6C46	✓	✓	✓	✓	✓	✓	✓	✓	✓	✗	✗
Synthetic cathinones	6C47	✓	✓	✓	✓	✓	✓	✓	✓	✓	✗	✗
Caffeine	6C48	✗	✓	✓	✗	✗	✗	✓	✗	✗	✗	✗
Hallucinogens	6C49	✓	✓	✗	✓	✓	✓	✓	✗	✗	✗	✗
Nicotine	6C4A	✓	✓	✓	✗	✗	✗	✗	✗	✗	✗	✗
Volatile inhalants	6C4B	✓	✓	✓	✓	✓	✓	✓	✗	✗	✓	✓
MDMA / related drugs	6C4C	✓	✓	✓	✓	✓	✓	✓	✗	✗	✗	✗
Dissociatives: ketamine / PCP	6C4D	✓	✓	✗	✓	✓	✓	✓	✗	✗	✗	✗
Other specified psychoactive	6C4E	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Multiple specified / unknown	6C4F/G	✓	✓	✓	✓	✓	✓	✓	✓	✓	✗	✗
Non-psychoactive substances	6C4H	✗	✗	✗	✗	✗	✗	✗	✗	✗	✗	✗

HOW TO READ "MOOD*"

ICD-11 specifies depressive, manic, or mixed depressive/manic symptoms within substance-induced mood disorder. A tick marks availability of that parent category; it does not mean each presentation is common or automatically substance-caused. Timing, severity and alternative diagnoses must fit.

WITHDRAWAL • important exam distinctions

Alcohol / sedatives: uncomplicated, perceptual disturbances, **seizures**, or both; delirium is coded separately. ICD-11 recognizes inhalant and MDMA **withdrawal**. Hallucinogen and dissociative **withdrawal**: not listed. DSM **withdrawal**-criterion exceptions differ—see sheet 11.1.

ICD-10 CODE KEY • F1x is the substance family, the decimal is the syndrome

Families: F10 alcohol | F11 opioids | F12 **cannabinoid**s | F13 sedatives | F14 cocaine | F15 other stimulants, including caffeine | F16 hallucinogens | F17 tobacco | F18 volatile solvents | F19 multiple / other psychoactive drugs.
Suffixes: .0 acute intoxication | .1 harmful use | .2 dependence | .3 **withdrawal** | .4 **withdrawal** with delirium | .5 psychotic disorder | .6 amnesic syndrome | .7 residual / late-onset psychotic disorder | .8 other | .9 unspecified.
Example: F10.2 = alcohol dependence. Not every suffix applies to every substance. These suffixes are NOT ICD-11 suffixes.

CAFFEINE / NICOTINE • avoid absolute claims

Caffeine: anxiety category is listed; psychotic and mood categories are not. Nicotine: none of the eight induced mental-disorder columns above is listed. This does not deny **withdrawal** symptoms, toxicity or coincident illness.

INDUCED DISORDER ≠ expected intoxication symptoms

Symptoms arise during / soon after use or **withdrawal**, exceed the usual syndrome, and are not better explained by a primary disorder. Harmful episode / harmful pattern categories exist for every row; sleep and sexual disorders are outside this selected matrix.

Source: WHO ICD-11 CDDR (2024), Tables 6.13–6.14, pp450–454; mood requirements pp491–493. ICD-10 WHO CDDG (1992), F10–F19. Classification-specific; not a DSM matrix. • 11.2 • A4 / 300 DPI

Substance-induced disorders - ICD category applicability matrix. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: opioids, other drugs and treatment.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A25

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

AMBIVALENCE / EVOKING CHANGE

Motivational Interviewing / MET

Help people voice their own reasons for change while respecting autonomy and avoiding the urge to argue them into it.



William R. Miller and Stephen Rollnick
Co-developers of motivational interviewing

THE CLINICAL IDEA

The clinician guides the conversation; the person owns the reasons and the decision.

AI-ILLUSTRATED PORTRAIT

A25

William Miller and Stephen Rollnick • MET: structured motivational enhancement

SECTION 01 THE MODEL

1. Engage
2. Focus
3. Evoke
4. Plan

Ambivalence is expected. Change talk expresses movement toward change; sustain talk supports the status quo. Discord concerns the relationship and calls for an adjustment in approach.

SECTION 02 THE TECHNIQUES

- **Hold the spirit.** Work in partnership with acceptance, compassion and empowerment; draw out the person's own knowledge and reasons.
- **Use OARS.** Open questions, affirmations, reflective listening and summaries; use reflections to understand, not covertly pressure.
- **Evoke and strengthen.** Explore importance and confidence, reflect change talk, ask permission to share information, and use elicit-provide-elicit.
- **Know MET.** Combine assessment feedback with an MI-consistent style and a negotiated change plan; the classic Project MATCH MET protocol used four sessions.

SECTION 03 IN THE ROOM

Fictional example: A person values family connection but worries that change will remove their way to unwind. The clinician reflects both sides and explores a first feasible step.

SECTION 04 WHAT THE EVIDENCE SAYS

A 2023 Cochrane review found modest, comparator-dependent substance-use benefits, with little or no clear advantage over regular care or another active intervention. MI is not a substitute for comprehensive addiction treatment. [1,2]

CLINICAL CAUTION

Do not label disagreement as resistance or use MI as manipulation. Assess withdrawal and other risks separately; autonomy does not remove clinical responsibility.

SELECTED REFERENCES
1. Miller WR, Rollnick S. Motivational Interviewing: Helping People Change and Grow. 4th ed. Guilford; 2023.
2. Motivational interviewing for substance use reduction. Cochrane; 2023. doi:10.1002/14651858.CD008063.pub3

Motivational interviewing MET. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: opioids, other drugs and treatment.

Credits and references: retained within the illustration.

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

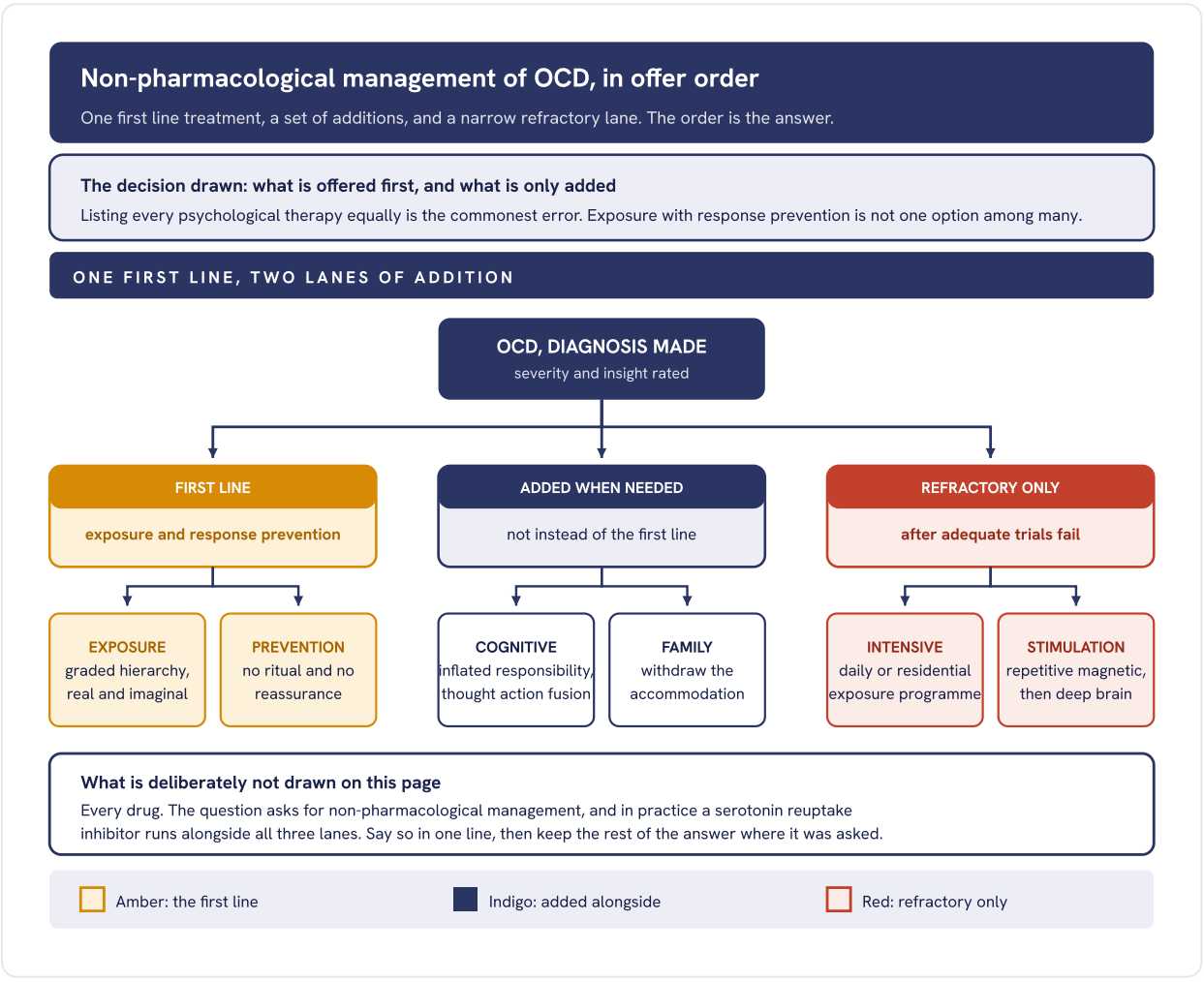


Fig 019.1 Exposure with response prevention drawn as the trunk rather than as one option among six, with the refractory lane kept visibly separate.

- **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

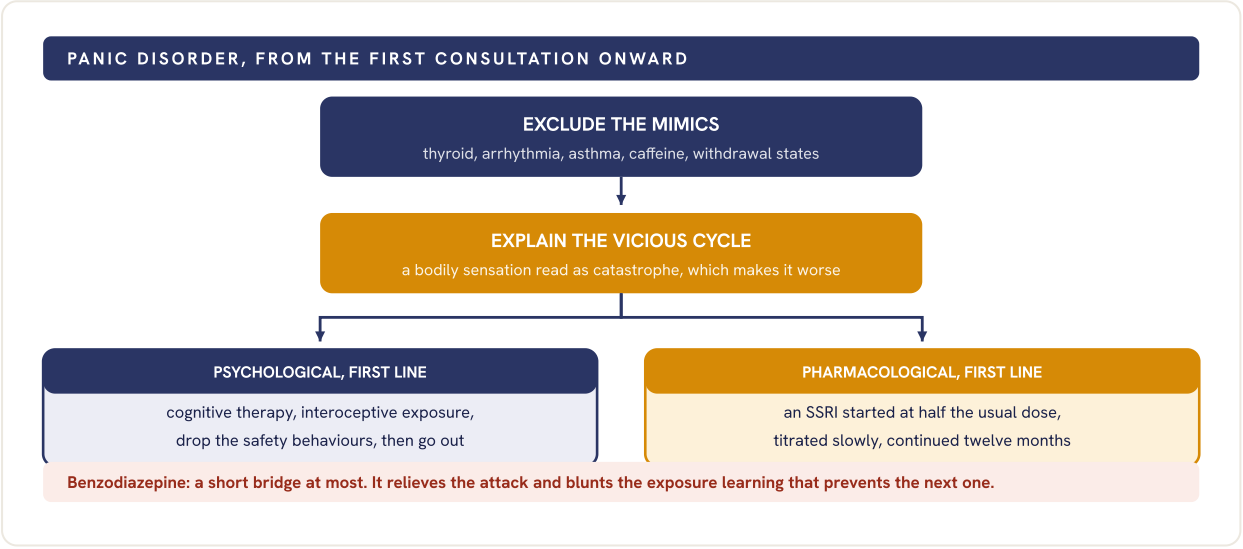


Fig 020.1 The two first line treatments drawn side by side rather than in sequence, with the benzodiazepine caution carried as a red strip along the foot.

- **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

Panic disorder: diagnosis and mechanisms

International Classification of Diseases (ICD) • Diagnostic and Statistical Manual (DSM) • distilled comparison

FEATURE	ICD-10 • clinical guidelines	ICD-11 • CDDR 2024	DSM-5-TR • 2022
Attack / recurrence	F41.0: recurrent severe panic, not confined to a particular situation; unpredictable. Sudden autonomic anxiety; attacks usually last minutes.	6B01: recurrent attacks; **at least some unexpected** . Rapid concurrent onset of **several** characteristic symptoms. No fixed four-symptom threshold.	Recurrent **unexpected** attacks. Abrupt fear/discomfort, **peak within minutes**, **≥4 of 13 symptoms**. Expected attacks may also occur.
Time / aftermath	Several severe attacks within **about 1 month**, without objective danger or predictable situations; comparative freedom between attacks. Anticipatory anxiety can occur.	Persistent concern (e.g. **several weeks**) about recurrence/significance **OR** avoidance. Significant impairment, or function maintained only with extra effort. No rigid DSM one-month gate.	After ≥1 attack: **≥1 month** persistent concern about recurrence/consequences **OR** significant maladaptive behaviour change.
Exclusions / agoraphobia	Medical/substance differential; older hierarchy gives established phobias precedence. Agoraphobia with panic: F40.0 with appropriate subdivision; depression may take precedence.	Exclude medical/substance/medication effects and better explanations. **Agoraphobia is separate**; diagnose both if independently met.	Exclude medical/substance causes and better psychiatric explanations. Only obsession-, trauma-, phobia- or separation-triggered attacks do not establish panic disorder. **Agoraphobia is separate.**

DSM PANIC ATTACK: FOUR OF THIRTEEN

Palpitations • sweating • trembling • breathlessness/smothering • choking • chest discomfort • nausea/abdominal distress • dizziness/unsteadiness/faintness • chills/heat • numbness/tingling • **derealisation OR depersonalisation (one item)** • fear of losing control • fear of dying. Culture-specific additional symptoms do not count toward four.

EPIDEMIOLOGY / COURSE

Annual **2–3%** in US/several European adult/adolescent populations; World Mental Health surveys **1.7% global lifetime**. Women ≈2:1. US median onset 20–24 years. Do not relabel these as Indian estimates. Avoidance and anticipatory fear often persist between attacks; assess depression/substance use.

VULNERABILITY / NEUROBIOLOGY

Familial/polygenic vulnerability + stress + **anxiety sensitivity** (fear that anxiety sensations signify harm). Insula: internal body signals; amygdala/hippocampus: threat/context; prefrontal/anterior cingulate: regulation; brainstem/locus coeruleus: autonomic/noradrenergic arousal. No single “panic centre” or diagnostic scan.

CLARK’S LOOP + RESPIRATORY MODEL

Bodily sensation → catastrophic appraisal → fear/arousal → stronger sensation → apparent confirmation. Escape/checking gives relief but blocks corrective learning. **Klein’s false-suffocation alarm** is a hypothesis for a subgroup: carbon-dioxide sensitivity is not a routine diagnostic test. Overbreathing may cause tingling/dizziness; it is **not required** in every attack.

Sources: WHO ICD-10 CDDG F41.0 (not separate research criteria); WHO ICD-11 CDDR (2024), pp271–274; APA DSM-5-TR (2022), pp235–240; IPS CBT (2020). Peak within minutes ≠ end within 10 minutes.

WEAVE 100 · PAPER II · 20.8

Panic disorder: treatment and follow-up

IPS 2017 pharmacological framework + IPS 2020 cognitive behavioural therapy (CBT) • adult dosing

TRIAGE → CONFIRM / FORMULATE

• New chest pain, syncope, hypoxia or concerning examination → **urgent medical assessment**, even with known panic. Exclude arrhythmia, cardiopulmonary/endocrine illness, hypoglycaemia, stimulants and withdrawal.
• No acute medical concern → calm setting, stay/explain, gentle slower breathing if overbreathing; **no paper-bag rebreathing**. Assess depression, suicidality, bipolarity and agoraphobia; track panic severity AND avoidance/function.

FIRST LINE: CBT ± SSRI / SNRI • IPS 2017 / 2020

• CBT: **interoceptive exposure** to medically suitable benign sensations + cognitive testing of catastrophe predictions + graded real-life exposure; reduce checking, escape and companion dependence. Usually **10–15 weekly sessions**, individualised.
• Selective serotonin reuptake inhibitor (SSRI) / serotonin–noradrenaline reuptake inhibitor (SNRI): **start low, titrate slowly**; explain early activation and delayed benefit. Combine by severity, preference and response.

ADULT ORAL DOSE • label context	START → RANGE / DAY
Sertraline	25 mg for 1 week → 50 mg; **50–200 mg**
Fluoxetine	10 mg for 1 week → 20 mg; **20–60 mg** studied
Venlafaxine extended release	37.5 mg for 7 days → 75 mg; **75–225 mg**

MONITOR / CONTRAINDICATIONS

Review activation, suicidality, sexual/gastrointestinal effects, adherence and interactions; **blood pressure with venlafaxine**, sodium if at risk. Avoid monoamine oxidase inhibitor combinations / unsafe serotonergic overlap; bipolarity → stabilise mood. Titrate within drug/patient limits. Tricyclics later: cardiac, anticholinergic and overdose risks.

Adequate trial: improving?

YES

YES → CONTINUE

Maintain gains and exposure practice. IPS 2017: **≥6–8 months**, longer if needed; stopping section requires ≥6 months stable/asymptomatic. Then gradual taper + relapse plan.

NO

NO → REASSESS / CHANGE

Check diagnosis, dose, adherence, substances, comorbidity and exposure quality. Partial benefit → optimise/add CBT; no benefit/intolerance → switch SSRI/SNRI. **NICE:** after no improvement at 12 weeks, another class or therapy.

BENZODIAZEPINES: SELECTIVE SHORT TERM, WITH AN EXIT

• **IPS 2017 permits short-term acute use; NICE advises against them for panic disorder.** Rapid gamma-aminobutyric acid type A enhancement does not replace exposure learning. Clonazepam label: **0.25 mg twice daily → usual 1 mg/day**; 4 mg/day is a ceiling, not a target.
• Sedation, amnesia, falls, driving impairment and dependence; alcohol/opioids increase respiratory/overdose risk. Clonazepam contraindications: significant liver disease, acute narrow-angle glaucoma, benzodiazepine hypersensitivity. Caution with sleep apnoea, older age, pregnancy and substance-use history.
• Regular dependent use → **never stop abruptly** (seizures/delirium). ASAM 2025: often **5–10% reductions every 2–4 weeks**, generally not >25%/2 weeks; slow/pause for withdrawal. Severe withdrawal/instability → higher-level care.

VR / AR: TOOLS, NOT REPLACEMENTS

• **Virtual reality:** simulated buses/crowds can assist situational exposure, especially agoraphobia; retain interoceptive work and real-world transfer. **Augmented reality:** overlays the real setting; evidence mainly specific phobias, insufficient panic-specific support. A small 2025 blended VR pilot does not prove equivalence to full CBT.
• Check cybersickness, dissociation, fall risk and privacy; severe symptoms → pause/assess. No transfer to real life → revise exposure plan. **NICE:** medication reviews at 2, 4, 6, 12 weeks; maintain ≥6 months after optimal dose. IPS CBT progress review every 1–2 weeks.

Sources: IPS panic (2017) / CBT (2020); NICE CG113; DailyMed labels; ASAM-led taper guideline (2025); VR pilot PMID40875824 / AR review 2025. Supplementary guidance is labelled.

20.8 • WEAVE 100+ / PAPER II • DISTILLED • A4 / 300 DPI • Paraphrased educational reference

Panic distilled - Management doses benzodiazepines and VR AR. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-20.

Credits and references: retained within the illustration.

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.

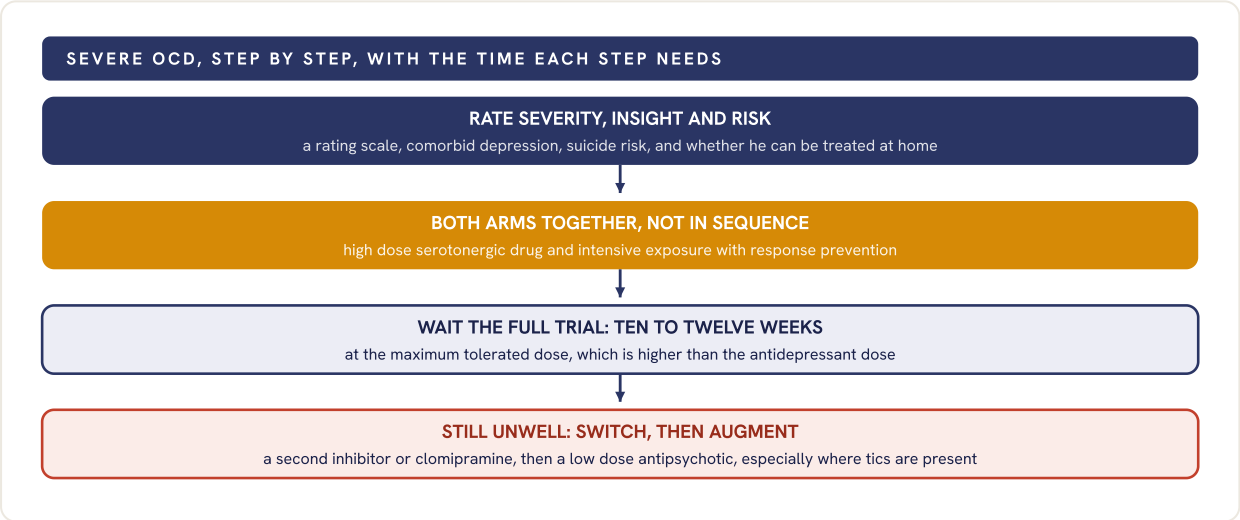


Fig 022.1 The ladder with the waiting time written into each step.

- **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

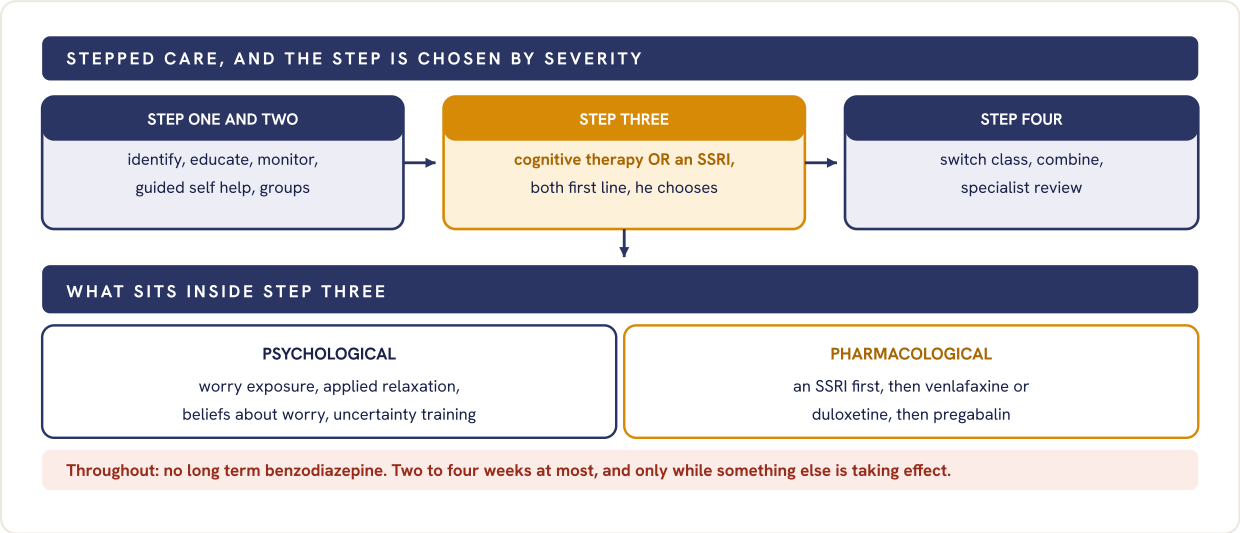


Fig 023.1 Four steps across the top, then the contents of the step that matters opened out below it.

- **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

WEAVE 100 · PAPER II · 23.1

Generalised anxiety disorder

Diffuse apprehension / worry · diagnostic systems · cognitive behavioural therapy and stepped care

Diagnosis · ICD-11 6B00 versus DSM-5-TR 2022

ICD-11: general apprehension or excessive worry across life domains, with additional tension, autonomic, concentration, irritability or sleep symptoms; more days than not for at least several months.
DSM: excessive, difficult-to-control worry for ≥6 months; ≥3 of 6 symptoms (restlessness, fatigue, poor concentration, irritability, muscle tension, disturbed sleep); only 1 symptom needed in children. Both require distress / impairment and appropriate exclusions.

Assess before treating · IPS GAD CPG 2017

Map worry, avoidance, reassurance, sleep and function. Assess depression, bipolarity, self-harm, substances and comorbid anxiety. Consider thyroid / cardiac illness, medication effects, caffeine and alcohol / benzodiazepine withdrawal; tests follow clinical findings.

Cognitive behavioural therapy (CBT) · IPS anxiety CBT CPG 2020

Worry versus problem	Separate hypothetical worry from a practical, solvable problem; use problem-solving for the latter.
Uncertainty / beliefs	Test overestimated danger, underestimated coping, the need for certainty and beliefs that worry is protective or uncontrollable.
Behavioural work	Graded exposure to avoided tasks / worry, reduce reassurance and overpreparation; practise between sessions.
Regulation / follow-up	Optional relaxation / mindfulness, regular routines and relapse planning. Initial 12–15 sessions can be spaced further as progress develops.

Stepped-care supplement · NICE CG113 (2011; amendments 2020–24)

1 Identify, educate, monitor → low-intensity CBT self-help / guided self-help or psychoeducational groups if symptoms persist.
2 Marked impairment or inadequate response → high-intensity CBT / applied relaxation OR medication, chosen collaboratively.
3 Inadequate response → review diagnosis, adherence, dose, duration and therapy quality; switch modality or combine after partial response. Severe refractory illness / major risk → specialist care.

Medication · IPS 2017 first; NICE sequence labelled separately

IPS: SSRIs, SNRIs and pregabalin are first-line options for GAD; choice considers comorbidity, preference and adverse effects. SSRI = selective serotonin reuptake inhibitor; SNRI adds noradrenaline reuptake inhibition.
NICE: choose an SSRI (consider sertraline); if ineffective, another SSRI or SNRI; if not tolerated, consider pregabalin. Discuss activation, withdrawal, sexual effects, interactions and pregabalin misuse / dependence; review pregnancy and renal considerations.

Continuation and safety · NICE CG113 supplement

Review early response and adverse effects, including suicidality in younger adults; monitor function as well as symptom score. If effective, continue medication for at least a year and taper gradually. Benzodiazepines only briefly during crises; no routine antipsychotic treatment in primary care.

Distinguish the feared outcome

GAD: many everyday possibilities. Panic disorder: recurrent unexpected attacks and their consequences. Social anxiety: negative evaluation. Agoraphobia: escape / help unavailable. Obsessive–compulsive disorder: obsessions and rituals. A GAD-7 score supports screening / follow-up, not diagnosis alone.

Sources: Weave Q23 p38 read / inspected. WHO ICD-11 CDDR (2024), pp266–270; DSM-5-TR (2022), GAD. IPS GAD / panic CPG (2017), PMC5310105; IPS anxiety CBT CPG (2020), S233–236. NICE CG113 (2011; current amended recommendations consulted Sep2026). Gold = IPS-only guidance; purple = named supplement / comparison.

Q23 · anxiety set 1 of 3 · 17 Sep 2026 A4 · 300 dpi

Generalised anxiety diagnosis and management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-23.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 23.2

Social anxiety disorder

Fear of negative evaluation · maintenance cycle · cognitive behavioural treatment

Diagnosis · ICD-11 6B04 and DSM-5-TR 2022

Marked fear of social interaction, observation or performance because of possible humiliation, rejection or negative evaluation. Situations are avoided or endured with intense anxiety; fear is disproportionate to actual threat / culture and causes distress or impairment.
ICD-11: at least several months. DSM: typically ≥6 months; "performance only" if restricted to speaking / performing in public. Shyness alone is insufficient.

Assessment · IPS anxiety CBT CPG 2020

Identify the feared prediction, anticipatory anxiety, avoidance, safety behaviours and functional cost. Assess depression, alcohol use and other anxiety. Liebowitz Social Anxiety Scale (LSAS) and Social Phobia Inventory (SPIN) can track severity; clinical assessment establishes the diagnosis.

What maintains it · IPS CBT formulation 2020

Social situation → prediction of rejection → self-focused attention / negative self-image → anxiety and safety behaviours → less opportunity to test the prediction → post-event rumination.
Examples: excessive rehearsal, avoiding eye contact, hiding trembling. The behaviour may reduce discomfort now while maintaining fear over time.

CBT components · IPS 2020; individual formulation

Attention / imagery	Shift attention externally; use feedback and behavioural experiments to test the imagined view of the self.
Prediction testing	Agree a concrete feared outcome, practise a social task, drop selected safety behaviours and compare prediction with what happened.
Repeated exposure	Graded in-session and real-world social practice, role-play and homework; skills training when an actual deficit is present.
Cognitive / relapse work	Review unhelpful beliefs, anticipatory and post-event processing; generalise gains. Typically 14–16 sessions across 3–4 months.

Adult treatment pathway · NICE CG159 (2013) supplement

Offer individual CBT (Clark–Wells / Heimberg). If medication is preferred after CBT is declined: a selective serotonin reuptake inhibitor (SSRI), escitalopram or sertraline. Partial response at 10–12 weeks → add CBT; no response / intolerance → another SSRI or venlafaxine.
After good response in the first 3 months, continue for at least 6 further months; taper gradually. Do not routinely use benzodiazepines or antipsychotics. Review activation, interactions, withdrawal and safety.

Clinical distinctions

Agoraphobia centres on escape / help; specific phobia on a stimulus; body dysmorphic disorder on appearance defects; avoidant personality disorder involves a pervasive pattern. Autism-related social differences may coexist. Assess the actual feared outcome rather than deciding by avoidance alone.

Other anxiety diagnoses · complete the spectrum map

Separation anxiety: excessive fear of separation from attachment figures, beyond developmental norms, with impairment; adults can be affected. ICD-11: several months. DSM: ≥4 weeks in youth, typically ≥6 months in adults.
Selective mutism: speaks in some settings but consistently not in others; ≥1 month, beyond the first school month, with impairment. Exclude language unfamiliarity and other explanations. These need age-appropriate specialist assessment; they are not simply "stubbornness" or adult social anxiety.

Sources: Q23/Q26 anxiety-area supplement; no separate Weave social-anxiety question identified. WHO ICD-11 CDDR (2024), pp284–296; DSM-5-TR (2022), anxiety disorders. IPS anxiety CBT CPG (2020), S237–240. NICE CG159 (2013), §§1.3 and 1.6; adult medication pathway. Anxiety map includes separation anxiety / selective mutism; existing panic and OCD sheets retained.

Anxiety set 2 of 3 · SAD here means social anxiety disorder · 17 Sep 2026 A4 · 300 dpi

Social anxiety and related anxiety diagnoses. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-23.

Credits and references: retained within the illustration.

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

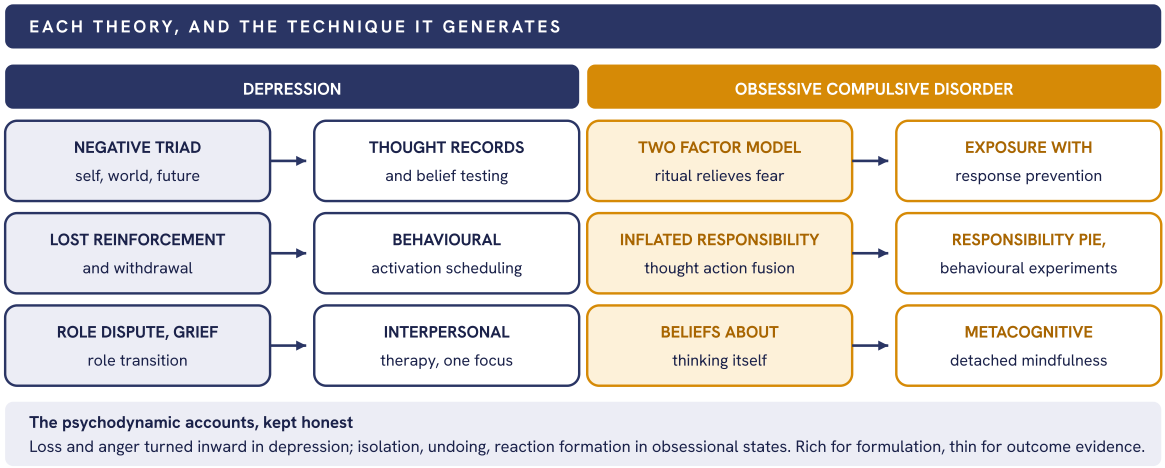


Fig 024.1 Each row reads left to right as one claim and the technique it licenses, which is exactly what the second half of the question asks for.

- **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

Depression: theory into treatment

Theory → maintaining process → intervention • Models can coexist; they are not separate diagnoses

FORMULATE + ASSESS

- Levels: beliefs/attention, lost reward, interpersonal stress and unconscious conflict.
- Assess severity, suicide risk and bipolarity before selecting psychological treatment.

Model	Maintaining process	Technique / limits
Beck • cognitive	Latent negative schemas + stress → biased interpretation / automatic thoughts. Negative triad: self ("worthless"), world ("unsupported"), future ("no improvement"). Errors: overgeneralisation; selective attention to failure.	Situation → thought → emotion/behaviour → evidence / alternative → behavioural experiment. "Nobody will respond" → send one planned message → review response. Aim: balanced evidence, not compulsory positive thinking.
Lewinsohn • behavioural	Lost response-contingent positive reinforcement → inactivity/withdrawal → fewer rewards → low mood. Avoidance relieves immediately but reduces mastery, social contact and reward.	Behavioural activation: monitor activity/mood → small scheduled pleasure/mastery/value-based tasks → graded action before motivation. Example: brief walk / manageable work task; review avoidance/reward. Avoid overwhelming timetables.
Helplessness / hopelessness	Uncontrollability → "actions make no difference". Attributional reformulation: internal + stable + global explanations of negative events → hopelessness.	Identify controllable steps; test specific alternatives; problem-solve; build mastery. Vulnerability/maintenance models—not inevitable depression after adversity.
Interpersonal	Grief, role disputes, role transitions and interpersonal difficulties may trigger/maintain episodes.	Interpersonal psychotherapy: focal problem → roles/emotions → communication analysis, role-play, support → review mood/relationships.
Psychodynamic	Loss, ambivalence, self-criticism, inward anger and recurrent relational patterns can shape distress.	Explore affect, defences and relationships. Modern psychodynamic therapies have evidence; this does not validate all classical causal claims or a universal cause.

REVIEW → CONTINUE / COMBINE / REFORMULATE • IPS 2026

- Evidence-based options: CBT, behavioural activation and interpersonal approaches. Add medication by severity, preference and response.
- Track symptoms/function: improvement → consolidate; poor progress → reassess barriers/diagnosis.
- Severe suicidal or psychotic depression → appropriate acute care.

Sources: Weave 100 II Q24; IPS depression CPG update (2026), pp80–82; NIMH depression / psychotherapies.

CBT = cognitive behavioural therapy • Models/examples are explanatory synthesis • Gold = attributed IPS guidance

Depression theories - Cognitive behavioural interpersonal and psychodynamic. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-24.

Credits and references: retained within the illustration.

OCD: psychological theories into practice

Pair the maintaining process with its technique and evidence limits

INTRUSION ≠ DISORDER

- Unwanted intrusions are common. Meaning + neutralising + avoidance + functional impact make persistence clinically important.
- Fear is not universal: disgust and incompleteness also matter.

Model	Process / concepts	Intervention / qualification
Mowrer • two-factor	Classical: cue → threat/distress. Operant: ritual/avoidance removes distress → negative reinforcement. Maintenance model; no recalled conditioning event required.	Exposure and response prevention (ERP): agreed hierarchy → cue → block overt/covert rituals → observe → repeat across contexts. New learning competes with fear; collaborative exposure respects real safety.
Salkovskis / Rachman • appraisal	Intrusion interpreted as danger, moral significance or responsibility. Inflated responsibility, threat overestimation, perfectionism and uncertainty intolerance → distress / neutralising.	Responsibility pie: distribute causation realistically. Thought–action fusion: thinking ≠ doing / changing probability. Experiments test beliefs; avoid repeated reassurance / perfect-certainty seeking.
Wells • metacognition	"Thought is dangerous / must be controlled" → threat monitoring, rumination, neutralising. Target relationship to thoughts, not endless content debate.	Detached mindfulness: notice a mental event without elaboration, suppression or ritual. Metacognitive techniques do not share an interchangeable evidence base with established CBT/ERP.
Psychodynamic • historical / formulation	Isolation of affect: idea detached from emotion. Undoing: symbolic cancellation of unacceptable thought/act. Reaction formation: exaggerated opposite attitude against an unacceptable impulse.	Intrusive thoughts do not prove hidden intentions. IPS 2026: dynamic psychotherapy is not primary OCD treatment. Do not replace effective CBT/ERP with interpretation alone.

SELECT → MEASURE → ADAPT • IPS 2026

- First-line psychological options: CBT including ERP, ERP alone, or cognitive therapy; address family accommodation/beliefs.
- Track ritual time, avoidance and function.
- Stalled → check covert rituals, formulation, engagement and comorbidity. Improving → generalise learning + boosters.

Sources: Weave 100 II Q24; IPS OCD update (2026), Tables 13–15; IPS CBT CPG (2020).

CBT = cognitive behavioural therapy • Theory-to-technique examples are tutor synthesis • Q24

OCD theories - Learning appraisal metacognition and defences. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-24.

Credits and references: retained within the illustration.

Depression, OCD and anxiety: overlap

Assess each full syndrome • Similar thoughts can serve different functions

SHARED VULNERABILITY ≠ ONE DISORDER

• Negative affectivity, stress, repetitive negative thinking, attentional bias, avoidance and uncertainty intolerance overlap.
• Genetic/neural overlap is partial; no single shared transmitter defect explains comorbidity. Similar symptoms can inflate apparent overlap.

OCD → DEPRESSIVE BURDEN

• Ritual time, shame, impaired work/relationships and less reward → low mood/hopelessness.
• One pathway; depression is not always secondary to OCD.

DEPRESSION / ANXIETY → OCD BURDEN

• Low energy/hopelessness reduce ERP participation; rumination can join mental checking. Anxiety increases threat attention/avoidance.
• Bidirectional effects; independent episodes also occur.

Pattern	Focus	Function / clue
Depressive rumination	Loss / failure / guilt; past or present	Dwelling, not effective problem-solving; often mood-congruent, not always ego-syntonic.
Generalised worry	Future problems across daily domains	Verbal “what if” chains to anticipate/control uncertainty; not automatically obsession.
Obsession + neutralising	Intrusive doubt / taboo / contamination / harm / incompleteness	Ritual/mental act for neutralisation or certainty. Content alone does not diagnose OCD.
Panic appraisal	Immediate bodily danger	Sudden fear + checking, escape, anticipatory avoidance; distinguish continuous worry.

DIAGNOSTIC BOUNDARIES

• Anxiety dimension ≠ separate anxiety disorder. OCD is outside the anxiety-disorder group in ICD-11 / DSM-5-TR.
• Diagnose comorbidity only when each syndrome is met; not every obsessional fear is generalised anxiety disorder.

Severe depression / acute risk blocks therapy?

YES → STABILISE FIRST • IPS OCD 2026

• Assess suicidality, psychosis, nutrition/function; treat severe depression promptly.
• May defer ERP until severe depression improves.
• ECT may treat indicated depression, not OCD itself.

NO → INTEGRATE TARGETS

• Rituals: ERP/cognitive work. Withdrawal: behavioural activation. Role stress: interpersonal work. Panic/worry: targeted CBT.
• SSRI may help both; retain OCD-specific trial adequacy.

FOLLOW-UP

• Track each disorder’s symptoms/function/safety separately; persistent symptoms → reformulate, not just total-score review.
• Screen bipolar disorder before antidepressant escalation. Intrusive harm thoughts ≠ intent; still assess suicide risk directly.

Sources: Weave 100 II Q24; DSM-5-TR; ICD-11 CDDR; IPS OCD / depression updates (2026), OCD p57.

ERP = exposure and response prevention; CBT = cognitive behavioural therapy; ECT = electroconvulsive therapy; SSRI = selective serotonin reuptake inhibitor

Depression OCD and anxiety - Comorbidity differences and integrated care. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-24.

Credits and references: retained within the illustration.

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.

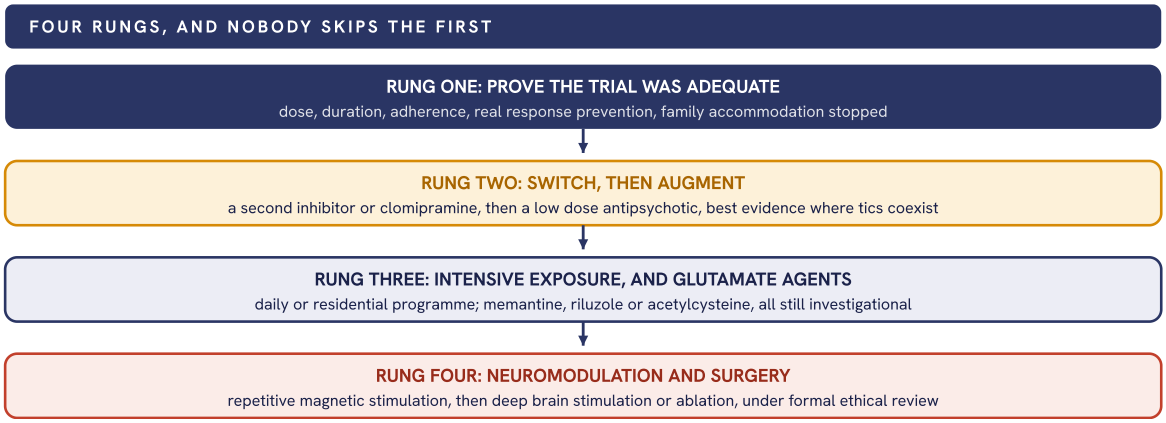


Fig 025.1 The ladder for resistant disease, with the audit of the first trial drawn as a rung rather than assumed, because most resistance is found there.

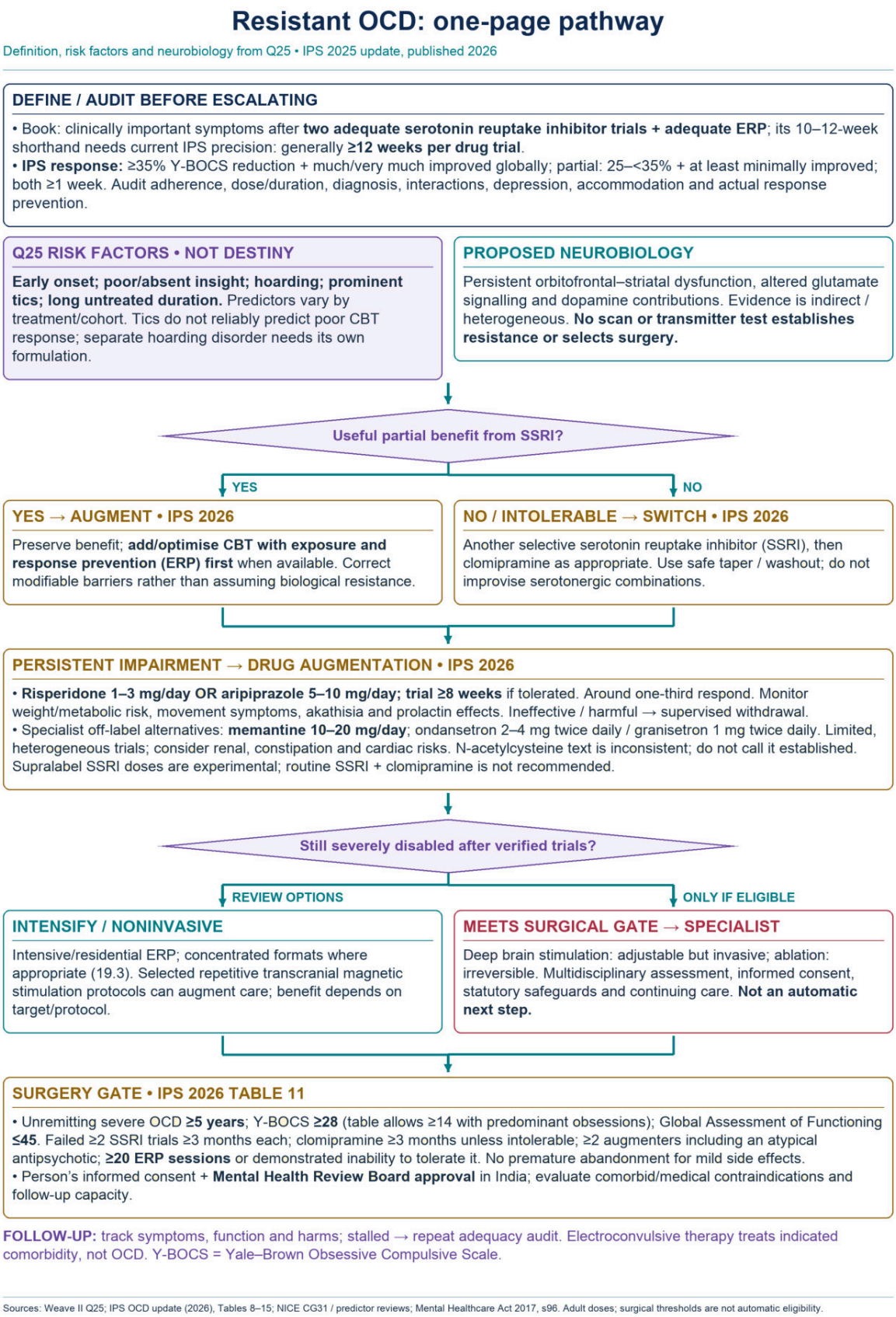
- **The proposed neurobiology.** Persistent overactivity in the orbitofrontal to caudate loop that does not normalise with treatment; raised glutamate in the caudate and cerebrospinal fluid with reduced inhibitory tone; and dopaminergic involvement, which is why antipsychotic augmentation helps where 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

WEAVE 100 · PAPER II · 25.3



Resistant OCD distilled - Definition risk factors and treatment. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-25.

Credits and references: retained within the illustration.

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.

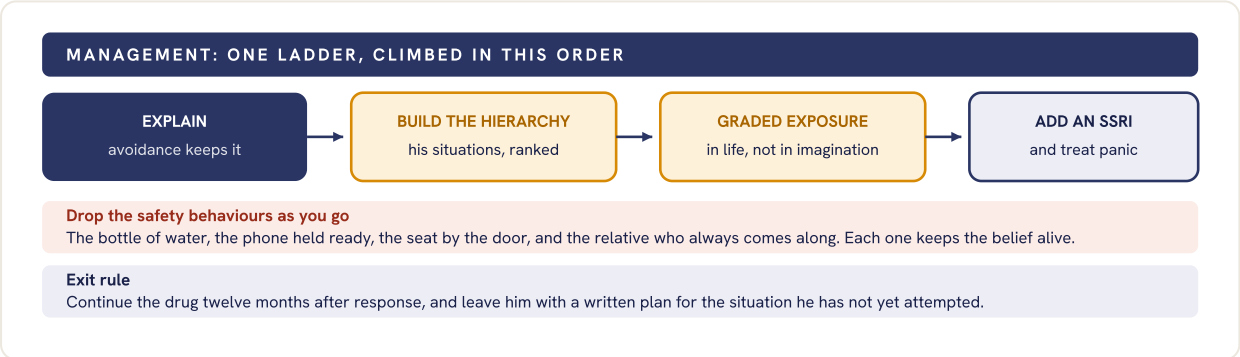


Fig 026.1 Exposure drawn as the spine of treatment with the drug alongside it, and the safety behaviours carried as a red strip because they are what quietly defeats the ladder.

- **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.

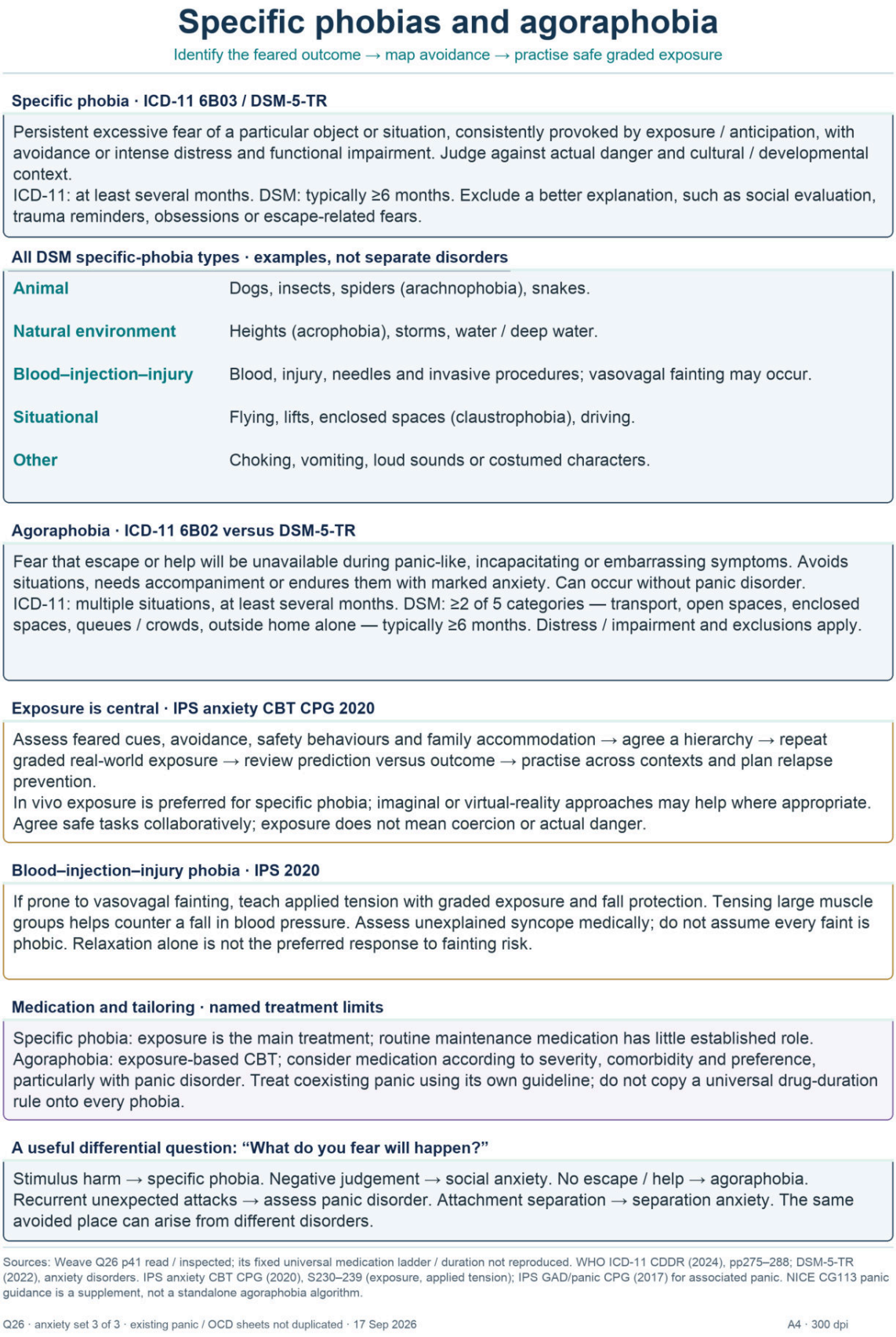
WHAT EARNS THE MARKS

- **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

WEAVE 100 · PAPER II · 26.1



Specific phobias and agoraphobia. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-26.

Credits and references: retained within the illustration.

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.

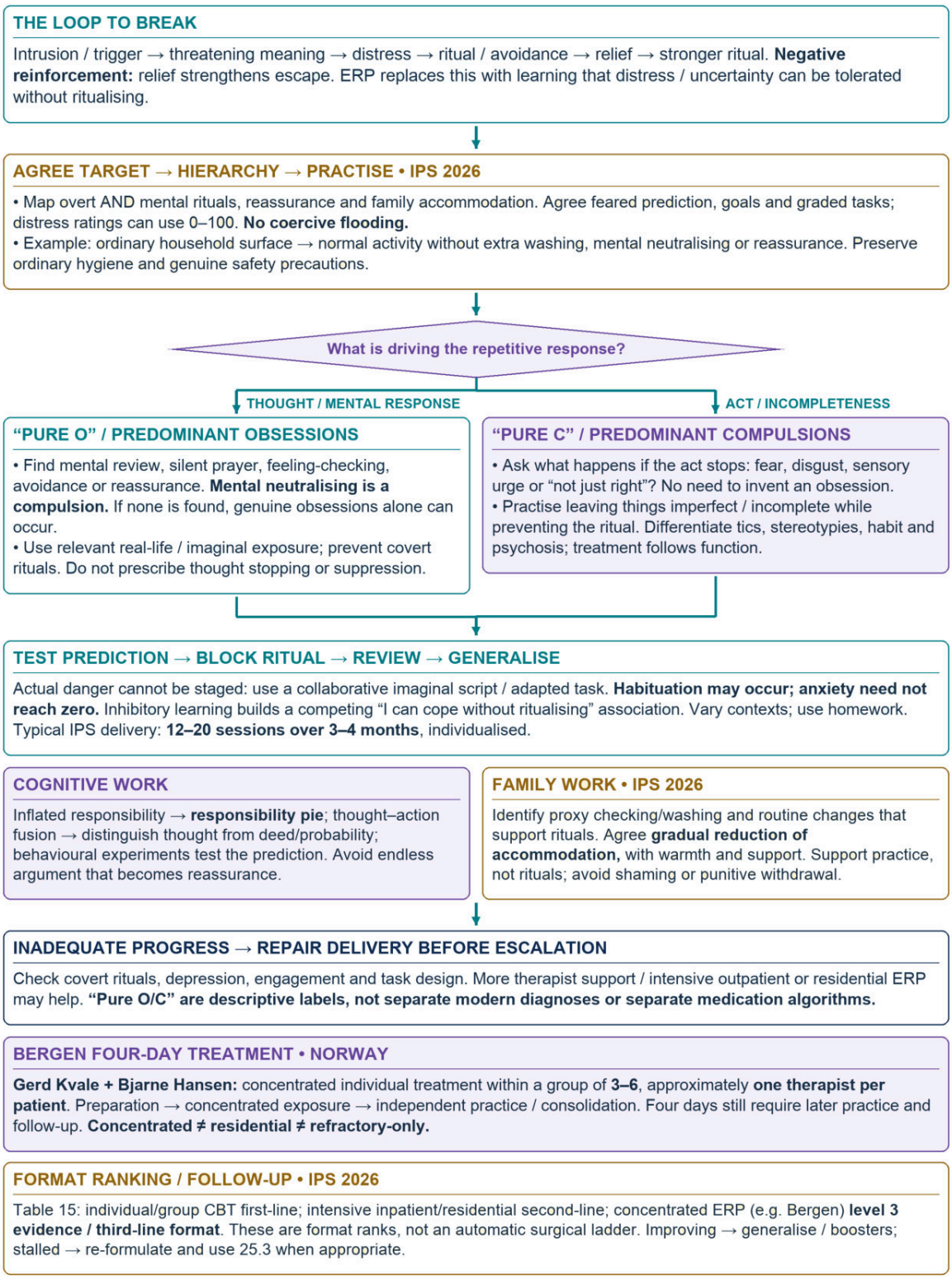
SEE ALSO Slot II - 21 etiopathology of OCD Slot II - 22 severe OCD SPRINT

Day 14, Anxiety, OCD and trauma-related disorders

WEAVE 100 · PAPER II · 19.3

ERP, hidden rituals and Bergen

Exposure and response prevention (ERP) within cognitive behavioural therapy (CBT) • adapted targets / delivery



OCD distilled - ERP pure O pure C and Bergen. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Anxiety, OCD and related disorders.

Credits and references: retained within the illustration.

OCD: vulnerability, circuits and course

Obsessive–compulsive disorder • Epidemiology → mechanisms → clinical implications

PREVALENCE • source populations

- Lifetime: 1–3% (IPS overview).
- 12-month: US adults 1.2%; international ≈1.1–1.8% (DSM-5-TR).
- These are not Indian survey estimates.

ONSET / PHENOTYPE

- Childhood / early adulthood; US mean 19.5 years; 25% by age 14.
- Earlier in males; slightly more females among adults.
- Themes overlap and change.

MULTIFACTORIAL VULNERABILITY

- Polygenic + developmental/environmental influences → altered networks and learning.
- Family/twin evidence supports heritability: a population statistic, not destiny. No single diagnostic gene or scan.



Domain	Mechanism / finding	Qualification
Circuit / imaging	Facilitation–suppression imbalance → repetition / difficult disengagement. Parallel loops support valuation, monitoring, action and habits.	Wider frontoparietal/frontolimbic networks implicated. Group associations, not one lesion; arrows are connections, not uniformly excitatory.
Neurochemistry	Serotonergic response: therapeutic target. Glutamate abnormalities: region-dependent. Dopamine: partly supported by augmentation evidence.	Response does not prove serotonin deficiency. Direct/indirect pathway balance is a model, not a diagnostic test.
Psychological maintenance	Intrusion → catastrophic meaning / inflated responsibility → distress → ritual/avoidance → relief → stronger ritual.	Negative reinforcement = distress removal strengthens behaviour. Thought–action fusion: thought equated with action or greater likelihood.
Immune / developmental	Abrupt paediatric onset → consider acute-onset neuropsychiatric or streptococcal-associated presentations.	Debated subgroup; not all OCD. Does not justify routine immune treatment.

COURSE + COMORBIDITY • IPS update published 2026

- Often chronic/fluctuating, not uniformly poor. Long-term prospective evidence: nearly half remit; definitions/follow-up differ.
- Depression, anxiety and tics common; assess suicide risk. Response ≠ remission ≠ recovery.
- Severity, persistent family accommodation and untreated comorbidity may worsen outcome; predictors are not deterministic.

Sources: DSM-5-TR (2022), pp267–270; IPS OCD update (2026), pp45–47; NIMH OCD overview.

Circuit and maintenance arrows are explanatory models • No individual diagnostic gene or scan • Q21

OCD - Etiology prevalence neurobiology and course. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Anxiety, OCD and related disorders.

Credits and references: retained within the illustration.

OCD: diagnostic distinctions

Obsessive-compulsive disorder • three systems • retain the OR gates and system-specific exceptions

FEATURE	ICD-10 • WHO clinical guidelines	ICD-11 • WHO CDDR 2024	DSM-5-TR • APA 2022
Symptom gate	Obsessions, compulsions **or both**; F42. Repetitive thoughts / images / impulses recognised as one's own. At least one thought or act is unsuccessfully resisted.	Persistent obsessions **and/or** compulsions; 6B20. Intrusions are unwanted; repetitive acts / mental rituals may follow obsessions, rigid rules or incompleteness.	Obsessions, compulsions **or both**. Recurrent/persistent, intrusive, unwanted thoughts / urges / images; attempts to suppress, ignore or neutralise. Compulsions include mental acts.
Nature of ritual	Stereotyped acts are **not inherently pleasurable**; relief of tension is not pleasure.	Acts / mental rituals are excessive or not realistically connected with the feared outcome; a need for completeness can drive them.	Driven by obsessions or rigid rules; intended to reduce distress / prevent harm, but **excessive or unrealistically connected**.
Duration / impact	Most days for **≥2 successive weeks**, causing distress or interference. **No one-hour threshold.**	Time-consuming (**e.g. >1 hour/day**) **OR** significant distress **OR** impairment. **No fixed minimum illness duration.** Maintained function may require extra effort.	Time-consuming (**e.g. >1 hour/day**) **OR** clinically significant distress **OR** impairment. **No fixed minimum duration**; one hour is not mandatory if impact is sufficient.
Insight / types	Predominant obsessions F42.0; predominant compulsions F42.1; mixed F42.2. Own-thought recognition does not imply judging the belief reasonable.	Fair-to-good insight (6B20.0) or poor-to-absent insight (6B20.1). No obsession/compulsion predominance subtypes.	Good/fair, poor, or absent insight/delusional beliefs. **Tic-related:** current/past tic disorder. No formal "pure O" subtype.
Exclusions / hierarchy	Older hierarchy attributes obsessional symptoms within schizophrenia, Tourette syndrome or organic disorder to those conditions. Depression precedence depends on course.	Exclude medical / substance / medication effects and better explanations. Comorbid syndromes can coexist when independently met.	Exclude substance / medical causes and a better psychiatric explanation: generalised worry, depressive rumination, psychosis, body-image preoccupation or stereotypies.

VIVA TRAPS

Poor insight ≠ automatic psychosis. Obsessions alone are possible; hidden mental rituals are common. Distress may be fear, disgust or incompleteness. "Ego-dystonic" = unwanted / inconsistent with wishes. Diagnose from the function of a thought or act, not its content alone.

Sources: WHO ICD-10 Clinical Descriptions and Diagnostic Guidelines, F42; WHO ICD-11 CDDR (2024), pp300–304; APA DSM-5-TR (2022), pp265–270. Paraphrased criteria.

OCD distilled - ICD-10 ICD-11 DSM-5-TR criteria. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Anxiety, OCD and related disorders.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 22.5

OCD: treatment, dose and duration

Indian Psychiatric Society (IPS) 2025 update, published 2026 • gold = IPS • resistant pathway: 25.3

ASSESS → CHOOSE SETTING / FIRST LINE • IPS 2026

• Assess suicide risk, bipolarity, depression, insight, tics, accommodation and covert rituals; follow Yale–Brown Obsessive Compulsive Scale (Y-BOCS) and function. **High risk / self-neglect → intensive or inpatient care.**
• **Severe OCD:** selective serotonin reuptake inhibitor (SSRI) + cognitive behavioural therapy (CBT) with exposure and response prevention (ERP). Mild–moderate: CBT/ERP alone when feasible, or SSRI by preference/access. No SSRI is consistently superior.

ADULT ORAL AGENT	START → IPS TARGET / DAY
Sertraline	50 mg → **150–200 mg**
Fluoxetine	20 mg → **60–80 mg**
Fluvoxamine	50 mg at night → **200–300 mg**, divided as needed
Clomipramine • second line	25 mg → **150–225 mg**; poorer tolerability
Escitalopram • off-label OCD	5–10 mg → IPS 20–30 mg; **>20 mg exceeds usual label ceiling**

TARGET ≠ START; LABEL LIMITS STILL MATTER

Starts are prescribing-reference context; targets are IPS Table 7. Lower / slower if sensitive. IPS lists citalopram 40–60 mg/day, but US label ceiling is **40 mg/day** (20 mg in specified high-risk groups) because of QT prolongation: **do not use 60 mg routinely**.

HIGHER DOSE → BENEFIT / HARM

IPS 2026: higher SSRI doses improve average efficacy but increase adverse-effect dropouts. Lower tolerated doses may still work. This is **not proof of "greater serotonin deficiency"**.

LONGER TRIAL → AVOID EARLY SWITCH

IPS 2026: titrate over **4–6 weeks**, then hold effective / maximum tolerated range **6–8 weeks**; generally **≥12 weeks total**. Continue if improving; early benefit can occur.

Mechanism (supplementary explanation): early transporter blockade → slower adaptation/plasticity and network change are plausible; no single proven explanation for the clinical lag.

SAFETY DURING TITRATION

• Monitor activation, suicidality, sleep, sexual / gastrointestinal effects; sodium and bleeding risks when relevant. **Bipolar disorder → stabilise mood first.** Avoid monoamine oxidase inhibitor combinations; use drug-specific washouts.
• **Clomipramine:** electrocardiogram, pulse / blood pressure, cardiac, anticholinergic and seizure risks; overdose toxicity. Drug levels when indicated. Routine SSRI + clomipramine combination is not recommended.

Adequate trial: useful response?

YES NO

YES → MAINTAIN • IPS 2026

Continue effective dose **≥1–2 years after remission**; ERP practice / boosters. Residual symptoms or previous relapse favour longer care. If stopping → gradual taper + relapse monitoring.

NO / PARTIAL → AUDIT • IPS 2026

Check dose, duration, adherence, interactions, comorbidity and actual ERP. Partial benefit → augment; no benefit / intolerance → switch. **See 25.3.** Harms → slow, reduce or change treatment.

Sources: IPS OCD update (2026), pp48–57 / Tables 7–9; DailyMed adult labels. Mechanistic explanations are qualified synthesis. All doses educational, not patient-specific.

22.5 • WEAVE 100+ / PAPER II • DISTILLED • A4 / 300 DPI • Paraphrased educational reference

OCD distilled - Management doses rationale and maintenance. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Anxiety, OCD and related disorders.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 22.6

Obsessive–compulsive rating scales

Yale–Brown scale architecture · severity · response and remission · interpretation

Yale–Brown Obsessive Compulsive Scale · Y-BOCS

Original adult clinician-rated instrument, Goodman et al. (1989). Establish target obsessions and compulsions using interview / checklist, then rate their severity over the past week. The symptom checklist describes content; it is not added to the severity total.

Obsessions	Compulsions
5 severity items **0–4 each** **Subtotal 0–20**	**5 severity items** **0–4 each** **Subtotal 0–20**

Total = obsession subtotal + compulsion subtotal = 0–40

Dimensions summarised: time occupied, interference, distress, resistance and control, considered for both symptom groups. Follow the original anchors; mental rituals count, and low resistance is not automatically poor insight.

Traditional adult bands · conventional, not diagnostic cut-offs

0–7 / 8–15	Subclinical range / mild symptom severity.
16–23 / 24–31	Moderate / severe symptom severity.
32–40	Extreme severity. Empirical boundaries differ; state your framework.

Response is not remission · international consensus 2016

Response: ≥35% reduction in (C)Y-BOCS plus Clinical Global Impression–Improvement (CGI-I) 1 or 2, for at least 1 week.
Remission: no longer meets OCD diagnostic criteria; if no diagnostic interview is feasible, (C)Y-BOCS ≤12 plus Clinical Global Impression–Severity (CGI-S) 1 or 2, sustained ≥1 week. Recovery requires remission for ≥1 year.
Example: 32 → 20 is a 37.5% fall: potential response with CGI confirmation, but not remission.

Versions are not interchangeable

Y-BOCS-II: revised adult severity scale, 10 items scored 0–5, total 0–50; includes revised handling of avoidance / resistance. Do not apply original 0–40 bands directly.
Children's Yale–Brown scale (CY-BOCS): developmentally adapted clinician assessment, using child and caregiver information. Specify original versus revised version.

Add what the total misses

Assess avoidance, family accommodation, insight, depression, suicidality, tics and functioning directly. Obsessional content may be concealed; establish rapport. Severe avoidance can reduce contact with triggers and make apparent symptom frequency misleading. A symptom subtype does not itself determine the severity score.

Use in practice and in an exam

Identify the version / rater / period → describe the two subscales and range → report baseline → repeat with trained ratings → interpret score change together with function and global improvement.
Y-BOCS is an OCD severity tool, not a standalone diagnostic test, and not a generic rating of compulsive behaviours across all disorders.

Sources: OCD-area supplement Q19/Q22/Q25. Goodman et al. (1989), doi:10.1001/archpsyc.1989.01810110048007. Baer & Blais (2010), Ch3. Mataix-Cols et al. (2016), doi:10.1002/wps.20299 (response/remission). Storch et al. Y-BOCS-II; primary benchmarking PMC13112087 distinguishes traditional bands from empirical alternatives. Original summaries; no full item form reproduced.

Q22 supplement · (C)Y-BOCS = adult / Children's Yale–Brown scale · 17 Sep 2026 A4 · 300 dpi

OCD rating scales and YBOCS. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Anxiety, OCD and related disorders.

Credits and references: retained within the illustration.

Obsessive–compulsive and related disorders

Classify the driver, then assess syndrome, impairment and exclusions • OCRD exam map

ENTRY MAP

Obsessions/rituals → OCD • Appearance → dysmorphia • Possessions → hoarding • Hair/skin urges → body-focused repetition
Odour/illness → additional named ICD-11 disorders. Assess distress/function; exclude substances, medical causes and better explanations.

Syndrome	Discriminator / diagnostic logic	System difference / assessment / exclusions
OCD	Unwanted intrusive thoughts/images/urges; behavioural or mental compulsions. Obsessions OR compulsions; >1 h/day (example) OR significant distress/impairment.	DSM-5-TR / ICD-11: no fixed minimum duration. ICD-10: most days ≥2 weeks. Assess insight, tics, depression/suicide risk, avoidance, family accommodation; Yale-Brown severity scale. Poor insight ≠ automatic psychosis.
Hoarding	Persistent difficulty discarding + need to save + discarding distress → clutter compromises living-space use. Third-party clearing may conceal severity; DSM excessive acquisition is optional specifier.	Often chronic; insight varies. Attachment, indecision, fear of waste/loss. Check fire/falls, exits, sanitation, dependants, animals. Distinguish collecting, poverty, OCD rituals, depression, dementia, psychosis.
Body dysmorphic disorder	Preoccupation with absent/slight appearance defect + checking, comparison, grooming/camouflage. DSM: repetitive behaviour/mental acts at some point; ICD-11 also allows marked avoidance.	DSM muscle-dysmorphia specifier. Assess insight, suicide risk, cosmetic consultations, functional avoidance. Exclude eating-disorder weight/fat concerns. Delusional appearance conviction can remain within diagnosis.
Hair pulling / skin picking	Recurrent hair pulling (trichotillomania) → hair loss; recurrent skin picking (excoriation) → lesions. Both: repeated attempts to reduce/stop + distress/impairment. Automatic/focused; tension/relief and obsessions not required.	Examine alopecia/skin; exclude dermatological disease, stimulants and appearance rituals. Hair swallowing → bezoar; picking → infection/scars.
Olfactory reference disorder	Offensive-odour belief/preoccupation; odour absent/barely noticeable. Checking, washing, masking, reassurance/avoidance; referential ideas (“covering noses because of me”).	ICD-11 named OCRD + insight qualifiers; DSM other-specified example. Check true odour/medical causes, depression/suicide risk; distinguish social anxiety / primary psychosis.
Hypochondriasis / health anxiety	Persistent serious-illness fear/preoccupation + checking/reassurance OR maladaptive avoidance. ICD-11: OCRD.	DSM illness anxiety: somatic-symptom group; absent/mild symptoms; ≥6 months preoccupation. Prominent symptoms + excessive response may fit somatic symptom disorder. Medical illness and health anxiety can coexist.
Secondary / residual	DSM: substance/medication-induced OR another medical condition—causal/temporal evidence, not coincidence; not exclusively delirium.	Both: other specified (state unmet criteria) / unspecified. ICD-11 also residual body-focused categories (e.g. impairing nail/lip/cheek biting). DSM other-specified example: obsessional jealousy.
Tourette / traps	ICD-11: cross-listed, primarily nervous-system tic disorder; DSM: neurodevelopmental. Multiple motor + ≥1 vocal tic, not necessarily together; onset <18; DSM course >1 year.	Premonitory urge ≠ obsession. Obsessive-compulsive personality disorder is not OCRD; anankastic rigidity/perfectionism ≠ intrusions. Repetition alone does not make gambling / compulsive sexual behaviour OCRDs.

TREATMENT ANCHORS • IPS 2020 / 2026

- OCD: ERP ± serotonin reuptake inhibitor → reassess.
- Dysmorphia: tailored CBT → review checking/avoidance.
- Hair/skin: habit reversal—awareness → competing response + stimulus control → review.
- Distinct protocols; no universal OCD drug ladder for all OCRDs.

SUPPLEMENTARY CARE

- Hoarding: home risk → collaborative sorting/discarding + non-acquisition → home follow-up. Forced clearance alone is inadequate.
- Odour/health: medical assessment → formulation-based psychological care → review.
- Drug evidence varies; no universal medicine/dose. Insight varies; comorbidity can coexist.

Sources: ICD-11 CDDR (2024); DSM-5-TR (2022), OCRD / illness-anxiety chapters; IPS CBT (2020); IPS OCD update (2026).

OCD = obsessive–compulsive disorder; CBT = cognitive behavioural therapy; ERP = exposure and response prevention • Q27

OCRD family - Exam overview. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Anxiety, OCD and related disorders.

Credits and references: retained within the illustration.

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

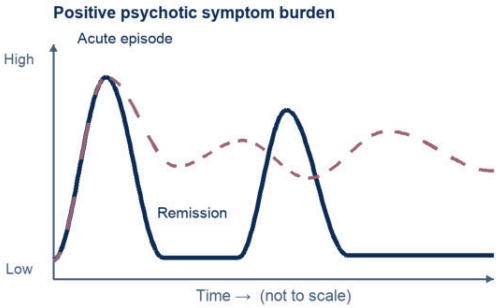
Day 8, Schizophrenia: aetiology, phenomenology, classification and course

Course of schizophrenia, bipolar disorder and obsessive–compulsive disorder

SCHEMATIC EXAMPLES • No patient data, probabilities or individual predictions

Schizophrenia

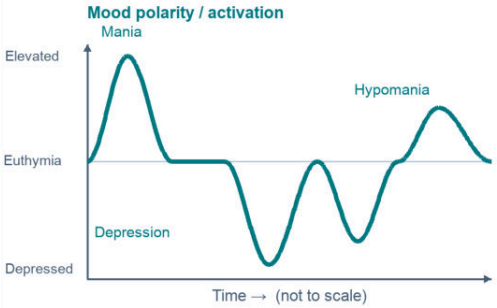
Variable episodic or persistent illness



Negative symptoms and cognitive difficulties may persist after psychosis improves. Neither relapse nor progressive deterioration is inevitable.

Bipolar disorder

Episodes differ in polarity and timing

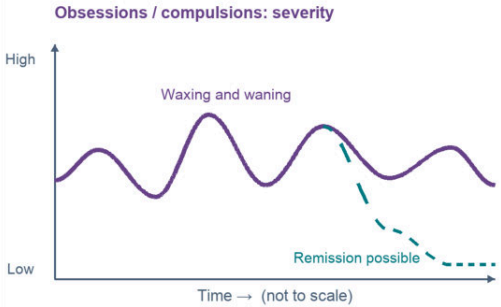


Example containing mania: bipolar I.
Bipolar II: hypomania + major depression; no history of a manic episode.

Long euthymic intervals are possible; residual symptoms and functional difficulties may persist. Episodes need not alternate; mixed features occur.

Obsessive–compulsive disorder

Commonly chronic and fluctuating



Solid: continuing symptom fluctuations.
Dashed: an alternative course with remission.
Remission is possible; chronic ≠ unremitting.

Symptoms can improve, persist or recur. Chronic does not mean continuously severe. Remission is possible; relapse can follow.

Response, remission, relapse and recovery

Response
Clinically meaningful improvement from baseline.
Symptoms may still be present; response is not remission.

Remission
Symptoms absent or below a defined clinically significant threshold, maintained for the duration required by that definition.
Symptom remission does not guarantee functional recovery.

Relapse
Return or significant worsening of symptoms after improvement or remission, according to the operational definition.
Some definitions distinguish relapse from a new recurrence.

Recovery
Sustained improvement; clinical definitions may combine symptom remission with restored functioning. Personal recovery emphasises agency and meaning, even with symptoms. It is not guaranteed cure.

Thresholds and durations vary by disorder and definition. Assess functioning separately.

Schematics: curve heights and durations cannot be compared across disorders.

Sources: Indian Psychiatric Society: schizophrenia (2026), obsessive–compulsive disorder (2025 update; published 2026).
Bipolar: CANMAT / ISBD (2018); outcome terms: ISBD (2009), OCD guideline Table 8; personal recovery: SAMHSA. • 28.1

Course - Schizophrenia bipolar disorder and obsessive-compulsive disorder. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-28.

Credits and references: retained within the illustration.

Cross-disorder landmark studies

Schizophrenia and bipolar disorder • Design → result → limits

Study / publication	Design / population	Endpoint / finding	Interpretation / limitation
[1] International Study of Schizophrenia (ISoS) Harrison et al., 2001	Cohort: 18 treated cohorts International follow-up: 15 / 25 years	• ≈75% traced • ≈50% of survivors: favourable outcome • 16% of early unremitting cases: late recovery	• Centre variation; survivor/attrition bias • “Favourable” ≠ universal recovery definition
[2] Madras Longitudinal Study Rangaswamy, 2012	Cohort: Chennai 90 first-episode schizophrenia patients 25 years	• 47/90 fully assessed; 32/47 partial/total remission • Assessed outcomes: 13 good, 25 intermediate, 9 poor	• 25 died; 18 lost • Assessed subset ≠ original cohort • No causal country comparison
[3] Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) Lieberman et al., 2005	Randomised: 1,493 adults Chronic schizophrenia Drug comparison; ≤18 months	• 74% (1,061/1,432 treated) stopped assigned drug • Olanzapine retention > quetiapine / risperidone	• Discontinuation: efficacy + tolerability + preference • Olanzapine: more weight/metabolic harm
[4] Recovery After an Initial Schizophrenia Episode – Early Treatment Program (RAISE-ETP) Kane et al., 2016	Cluster randomised: 34 clinics 404 with first-episode psychosis Age 15–40	• 2 years: NAVIGATE > community care for quality of life, symptoms and work/school participation	• Service package, not one drug • Mixed psychosis diagnoses • Greater benefit: shorter untreated psychosis
[5] Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD) Perlis et al., 2006	Cohort: 1,469 initially symptomatic 858 subsequently recovered	• ≤2 years: 416/858 (48.5%) recurrence • 34.7% depressive; 13.8% manic/hypomanic/mixed	• Naturalistic: predictors = associations • Denominator = recovered subset, not all entrants
[6] STEP-BD antidepressant trial Systematic Treatment Enhancement Program for Bipolar Disorder Sachs et al., 2007	Randomised: 366 adults Bipolar depression Mood stabiliser + bupropion/paroxetine OR placebo	• Durable recovery: 23.5% (42/179) antidepressant vs 27.3% (51/187) placebo • No significant difference	• Recovery = 8 consecutive euthymic weeks; treatment ≤26 weeks • Not monotherapy; no class-wide safety inference
[7] Bipolar Affective disorder: Lithium/Anticonvulsant Evaluation (BALANCE) Geddes et al., 2010	Open randomised: 330, age ≥16 Bipolar I; combination run-in ≤2 years	• New mood-episode intervention: combination 59/110; lithium 65/110; valproate 76/110 • Combination > valproate	• Combination not significantly > lithium • Open treatment; selected run-in tolerators

CBT = cognitive behavioural therapy • Study endpoints and populations differ: do not rank percentages across trials.

Original study names, years and linked reports retained in source ledger • All estimates refer to the stated populations

Cross-disorder landmark studies - Schizophrenia and bipolar disorder. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-28.

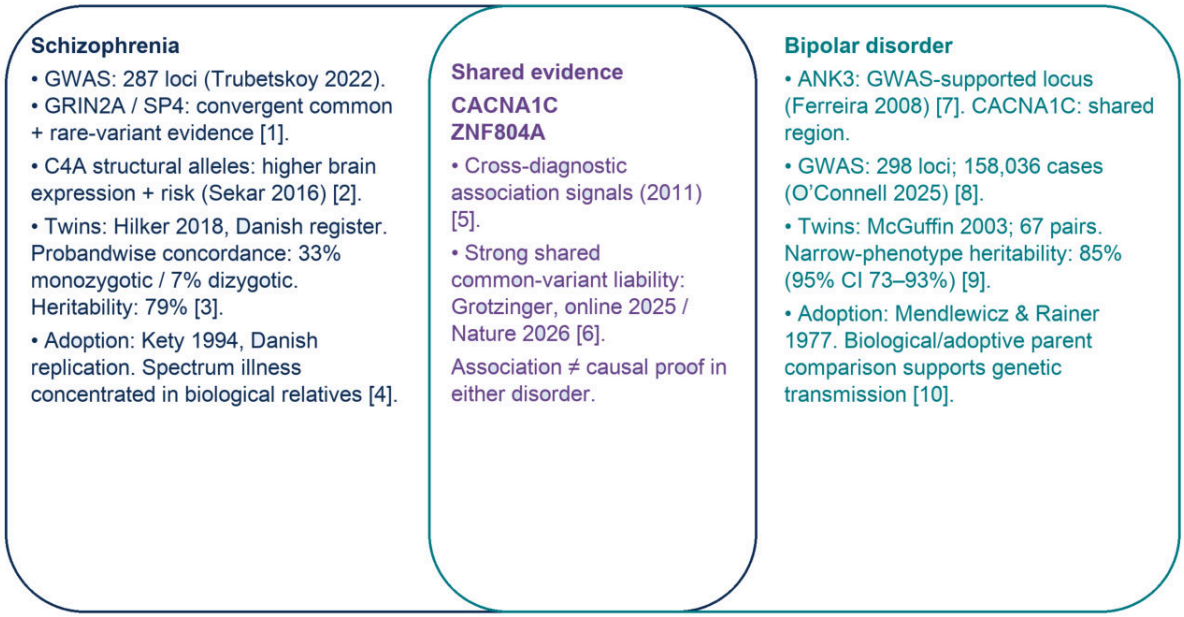
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WEAVE 100 · PAPER II · 28.4

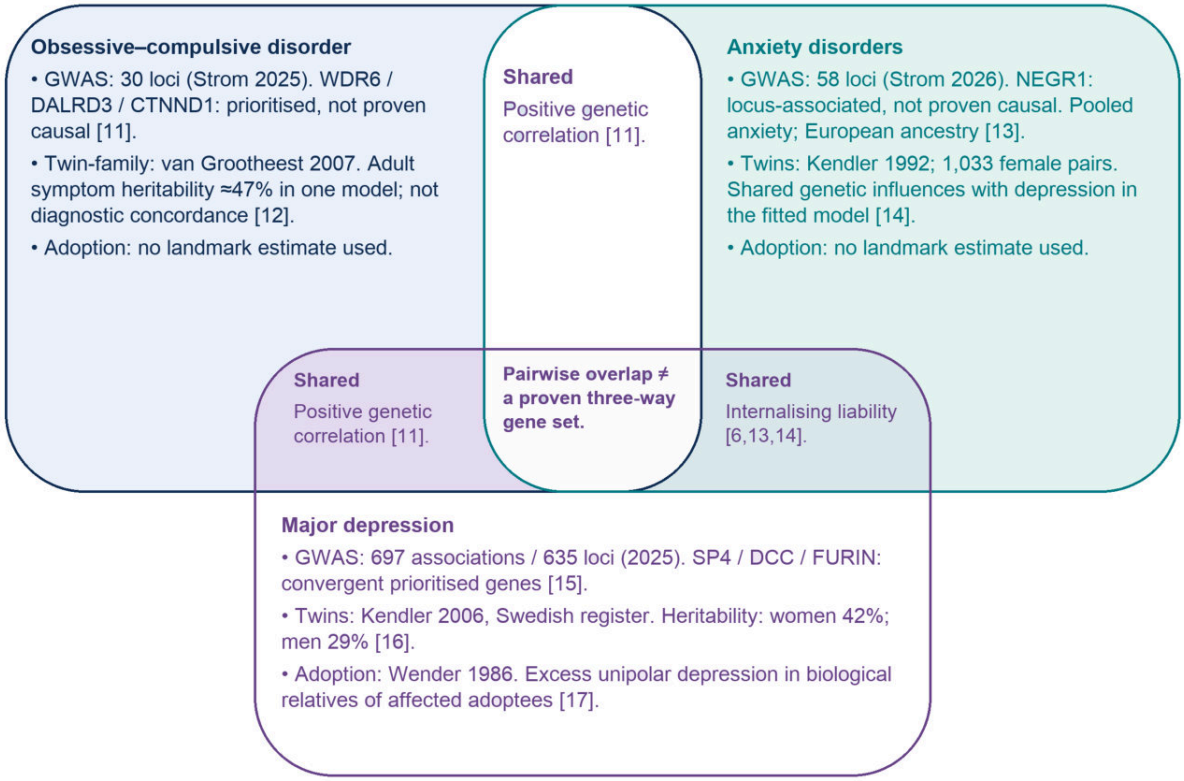
Genetics across five psychiatric disorders

Unique areas = evidence discussed under that disorder, not biological exclusivity.

Overlaps = selected shared evidence; shapes are conceptual, not proportional or exhaustive.



Obsessive–compulsive disorder, anxiety and depression



GWAS = genome-wide association study; a locus is a DNA region, not necessarily one causal gene.

Monozygotic = identical twins; dizygotic = fraternal twins (≈50% shared segregating DNA variants).

Concordance = co-twin illness probability; heritability = genetic share of population liability variance.

Estimates depend on phenotype, sample and method. Linked sources [1–17] in ledger • MD Psychiatry • 28.4

Comparative genetics - Schizophrenia bipolar obsessive-compulsive depression and anxiety. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-28.

Credits and references: retained within the illustration.

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

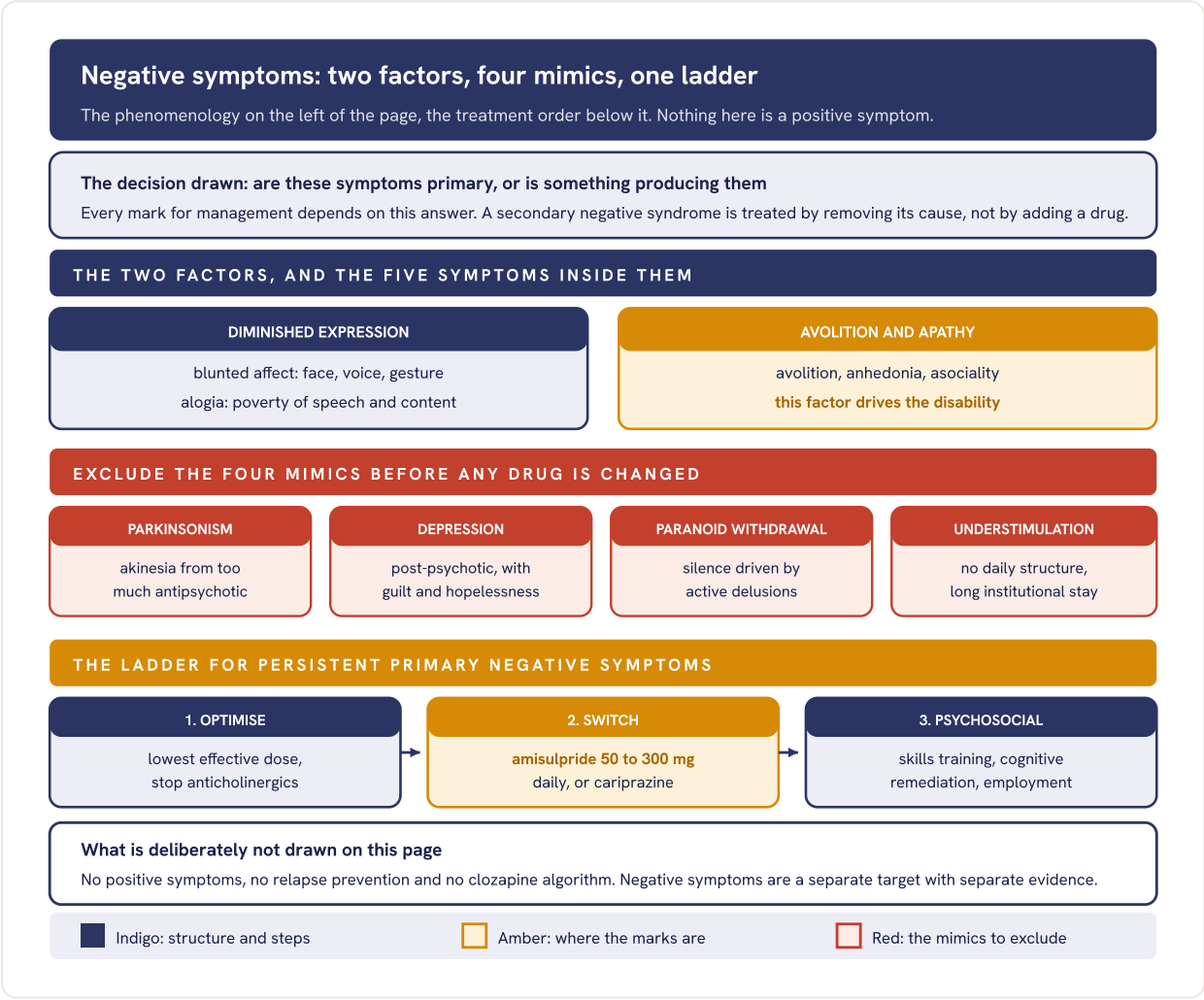


Fig 029.1 The two-factor structure of negative symptoms, the four secondary causes that must be excluded before the syndrome is called primary, and the three-step treatment ladder that follows.

- **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

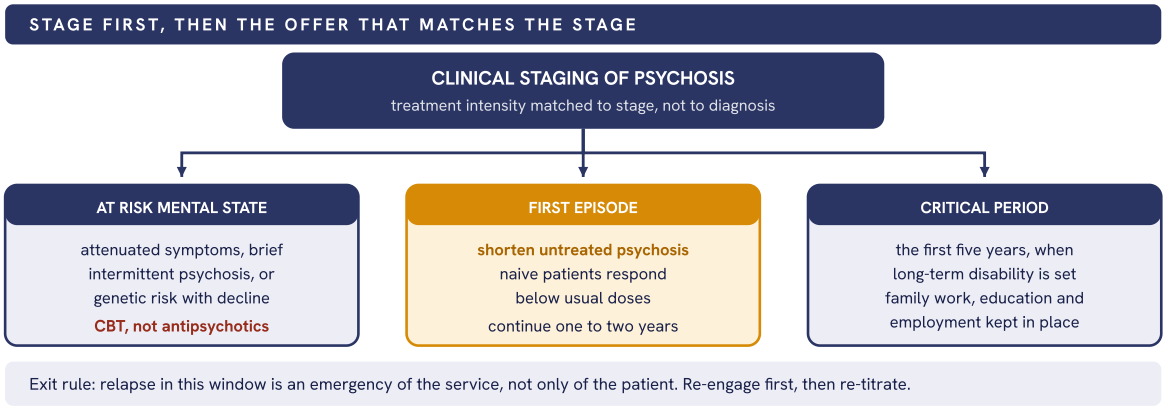


Fig 030.1 Early psychosis as three stages, each with the offer that belongs to it, and the first five years marked as the window where effort pays.

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

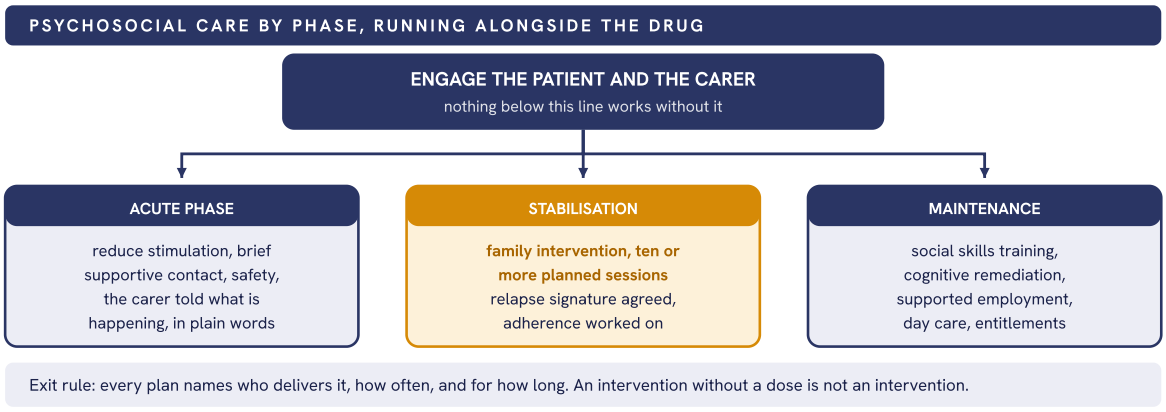


Fig 031.1 Psychosocial management by phase of illness, with the interventions that carry trial evidence placed where they are actually delivered.

- **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

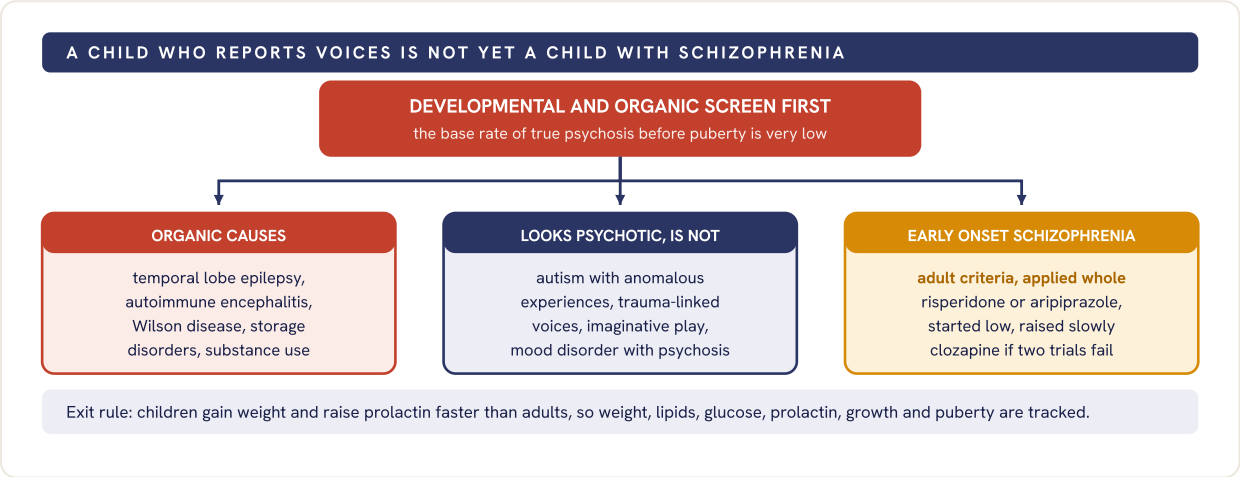


Fig 032.1 The order of work in a child with apparent psychosis: exclude the organic causes, separate the mimics, then treat as early onset schizophrenia.

- **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

Biomarkers across five psychiatric disorders

No standalone routine diagnostic biomarker • Group differences overlap substantially with healthy people

Reference = typical matched-control response, not a universal cut-off • ↑ higher/greater; ↓ lower/smaller

Schizophrenia

Measure	Reference	Finding • status • limits
P50 gating • EEG	Second click suppressed: S2 < S1	Suppression ↓; S2/S1 ↑ Candidate trait; relatives affected [1]
Prepulse inhibition	Weak prepulse → smaller startle	Startle inhibition ↓ Candidate trait; nonspecific; protocol-sensitive [2]
Mismatch negativity • EEG	Auditory deviant → negative EEG signal	Amplitude magnitude ↓ (less negative) Robust illness finding; weaker relative evidence [1,2]
P300 • EEG	Target detection → positive peak	Amplitude ↓; latency often ↑ Candidate trait; shared with bipolar disorder [1,2]
Eye movements	Smooth tracking; suppress cue-directed gaze	Pursuit gain ↓; corrective saccades ↑; antisaccade errors ↑ Candidate traits [2]
Cognitive testing	Working/verbal memory + attention	Performance ↓; persists beyond episodes; relatives affected Candidate traits [2]
Structural MRI	Ventricular/hippocampal volumes	Mean lateral ventricles ↑; hippocampus ↓ Neither specific nor universal [3]
Dopamine PET	Striatal presynaptic function	Mean synthesis/release ↑ Heterogeneous; not every patient [4]
Niacin skin response	Challenge → visible vasodilatory flush	Blunted flush / sensitivity ↓ Subgroup; research only [5]

Bipolar disorder

Measure	Reference	Finding • status • limits
P300 / cognition	Target peak; memory + attention	P300 amplitude ↓; memory/executive deficits may persist in euthymia Candidate traits [6,7]
Blood BDNF	Peripheral BDNF concentration	↓ in mania/depression; no clear pooled euthymic change State-associated [8]
Structural MRI	Cortical thickness	Mean frontal/temporal thinning Medication + illness effects [9]

Obsessive–compulsive disorder

Measure	Reference	Finding • status • limits
Error-related negativity	Performance error → negative EEG signal	More negative; relatives affected Candidate trait; shared [10]
Stop-signal / set shifting	Fast response inhibition; rule shifting	Stopping time ↑; flexibility ↓ Patients + some relatives; candidate traits [11]
Functional imaging	Task/rest circuit activity	Orbitofrontal/anterior cingulate hyperactivity in symptom studies Striatal direction varies [12]

Anxiety disorders — specify the phenotype

Measure	Reference	Finding • status • limits
Carbon dioxide • panic	May cause distress; less frequent panic	Panic ↑; some relatives sensitive Research probe [13]
Heart-rate variability	Panic/generalised anxiety: beat-to-beat variability	Mean resting vagal variability ↓ Medical/drug confounders [14]
Threat imaging	Social anxiety/specific phobia: threat activation	Amygdala/insula reactivity ↑ Task-dependent; not all anxiety disorders [15]

Major depression

Measure	Reference	Finding • status • limits
Dexamethasone suppression	Dexamethasone → cortisol ↓	Reduced/absent suppression in subset; especially psychotic depression State-sensitive [16]
Sleep recording	Usual rapid eye movement (REM) timing	REM latency ↓; density ↑ Medication/comorbidity-sensitive [17]
Structural MRI	Hippocampal volume	Volume ↓; especially recurrent illness Small effect; no individual diagnosis [18]
Blood BDNF	Serum BDNF concentration	↓ in untreated depression Treatment-sensitive; shared with bipolar episodes [19]
Inflammatory blood markers	C-reactive protein / interleukin-6	Mean ↑; raised only in subset Nonspecific research association [20]

EEG = electroencephalography • MRI = magnetic resonance imaging • PET = positron emission tomography • BDNF = brain-derived neurotrophic factor

Endophenotypes also require heritability, state independence and familial evidence • Routine tests assess alternatives/safety • Sources [1–20] retained • Q34

Comparative biomarkers - Reference responses and research findings. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-34.

Credits and references: retained within the illustration.

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

Cross-disorder landmark studies

Depression, obsessive–compulsive disorder and anxiety • Design → result → limits

Study / publication	Design / population	Endpoint / finding	Interpretation / limitation
[8] Sequenced Treatment Alternatives to Relieve Depression (STAR*D) **Rush et al., 2006**	Sequential treatment 3,671 analysed: adults with nonpsychotic major depression	• Self-rated remission, steps 1–4: 36.8%, 30.6%, 13.7%, 13.0% • Later steps: more follow-up relapse	• 67% cumulative remission was theoretical: assumed similar dropout outcomes • Not observed remission in 67% of entrants
[9] Treatment for Adolescents With Depression Study (TADS) **March et al., 2004**	Randomised: 439 Age 12–17; major depression 12 weeks	• Response: fluoxetine + CBT 71.0%; fluoxetine 60.6%; CBT 43.2%; placebo 34.8%	• Response ≠ remission • Therapy arms unblinded • Selected adolescents; not adults
[10] Antidepressants to prevent relapse in depression (ANTLER) **Lewis et al., 2021**	Randomised: 478 well primary-care adults Long-term antidepressants: maintain OR taper/stop	• Relapse by 52 weeks: maintain 92/238 (39%); discontinue 135/240 (56%) • Hazard ratio 2.06	• Withdrawal/crossover complicate interpretation • Does not establish lifelong treatment for everyone
[11] Pediatric Obsessive-Compulsive Disorder Treatment Study (POTS) **POTS Team, 2004**	Randomised: 112, age 7–17 CBT / sertraline / both / placebo 28 per arm	• 12-week remission: both 53.6%; CBT 39.3%; sertraline 21.4%; placebo 3.6%	• Combined: best symptom-score improvement • Remission not significantly > CBT alone
[12] Brown Longitudinal Obsessive Compulsive Study (BLOCS) **Eisen et al., 2013**	Cohort: 213 treatment-seeking adults 5 years; mean illness duration ≈22 years	• 39% experienced remission: 22.1% partial, 16.9% full • Improvement possible despite chronicity	• Selected chronic clinical sample; naturalistic care • Definitions/chronicity affect estimates
[13] Child/Adolescent Anxiety Multimodal Study (CAMS) **Walkup et al., 2008**	Randomised: 488, age 7–17 Separation anxiety / generalised anxiety / social phobia 12 weeks	• Response: CBT + sertraline 80.7%; CBT 59.7%; sertraline 54.9%; placebo 23.7%	• Response = much/very much improved • Combination > each monotherapy • Not adult / panic trial
[14] Child/Adolescent Anxiety Multimodal Extended Long-term Study (CAMELS) **Ginsburg et al., 2014**	Cohort: 288 former CAMS participants Mean 6 years after randomisation	• 134/288 (46.5%): no study-entry anxiety disorder at follow-up • Acute response predicted better outcome	• Only 59% of original 488 assessed • Later care naturalistic • Point remission ≠ continuous remission
[15] Coordinated Anxiety Learning and Management (CALM) **Roy-Byrne et al., 2010**	Randomised: 1,004 adults, 18–75 Panic / generalised or social anxiety / post-traumatic stress disorder	• 12-month response: collaborative care 63.66% vs usual care 44.68% • Remission: 51.49% vs 33.28%	• Flexible therapy/medication in primary care • Pooled outcomes ≠ equal benefit per disorder

CBT = cognitive behavioural therapy • Study endpoints and populations differ: do not rank percentages across trials.

Original study names, years and linked reports retained in source ledger • All estimates refer to the stated populations

Cross-disorder landmark studies - Depression obsessive-compulsive disorder and anxiety. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Schizophrenia: aetiology, phenomenology and course.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 28.6

Psychosis rating scales

Positive and Negative Syndrome Scale · Brief Psychiatric Rating Scale · change over time

Purpose and administration

These clinician-rated instruments quantify symptom severity and treatment change; neither establishes schizophrenia on its own. Use trained interview / observation with collateral information and a defined time window. Record the version and distinguish symptoms from medication effects, substances or medical illness.

Positive and Negative Syndrome Scale · PANSS · 30 items

Each item is scored 1–7: 1 means absent, so the total cannot be zero. Use the manual's anchors and interview protocol (commonly the previous week).
Positive: 7 items, range 7–49. Negative: 7 items, range 7–49. General psychopathology: 16 items, range 16–112. Total: 30–210.

7 positive **7 negative** **16 general psychopathology**

What PANSS adds — and misses

Balanced assessment of positive, negative and general symptoms; report subscales as well as total. Similar totals can hide different clinical profiles.
Negative ratings may reflect depression, positive symptoms, medication-induced parkinsonism or sedation. Investigate these before calling them primary negative symptoms. PANSS is not a comprehensive measure of cognition, disability or quality of life.

Percentage improvement: account for the PANSS floor

When reporting floor-corrected total-score percentage reduction:
100 × (baseline − follow-up) / (baseline − 30).
Example: 90 → 60 = 100 × 30 / 60 = 50%, rather than 33% using the uncorrected baseline. State the method; undefined at baseline 30. Raw score differences themselves do not need floor subtraction.

Brief Psychiatric Rating Scale · BPRS · specify the edition

Broad psychiatric symptom severity: affective, positive, negative and activation-related features. Interview-based and observed items are both used.
Common 18-item form, each 1–7 → total 18–126. Expanded 24-item form, each 1–7 → 24–168. Some forms use 0–6: their totals and floors differ. Do not mix versions, treat "not assessed" as absent, or compare raw totals without specifying the form.
BPRS is shorter / broader than PANSS; its total is not specific to schizophrenia.

Targeted additions when the clinical question needs them

Psychotic Symptom Rating Scales (PSYRATS): dimensions of voices and delusions, including distress, conviction and disruption; useful alongside a global symptom scale.
For prominent negative symptoms consider a dedicated instrument such as the Brief Negative Symptom Scale (BNSS) or Clinical Assessment Interview for Negative Symptoms (CAINS). Assess function and adverse effects separately.

Report a useful result

"PANSS total, positive / negative / general subscores; assessment period; baseline and current values; scoring method; clinical and functional change."
Add adherence, adverse effects, patient priorities and observed functioning. A lower total does not by itself establish symptomatic remission or recovery. Remission criteria also require sustained low severity in specified symptoms, not simply a total-score cut-off.
Use the same version and trained rating approach at each visit; discrepancies between informants and observation are clinical information, not numbers to average automatically.

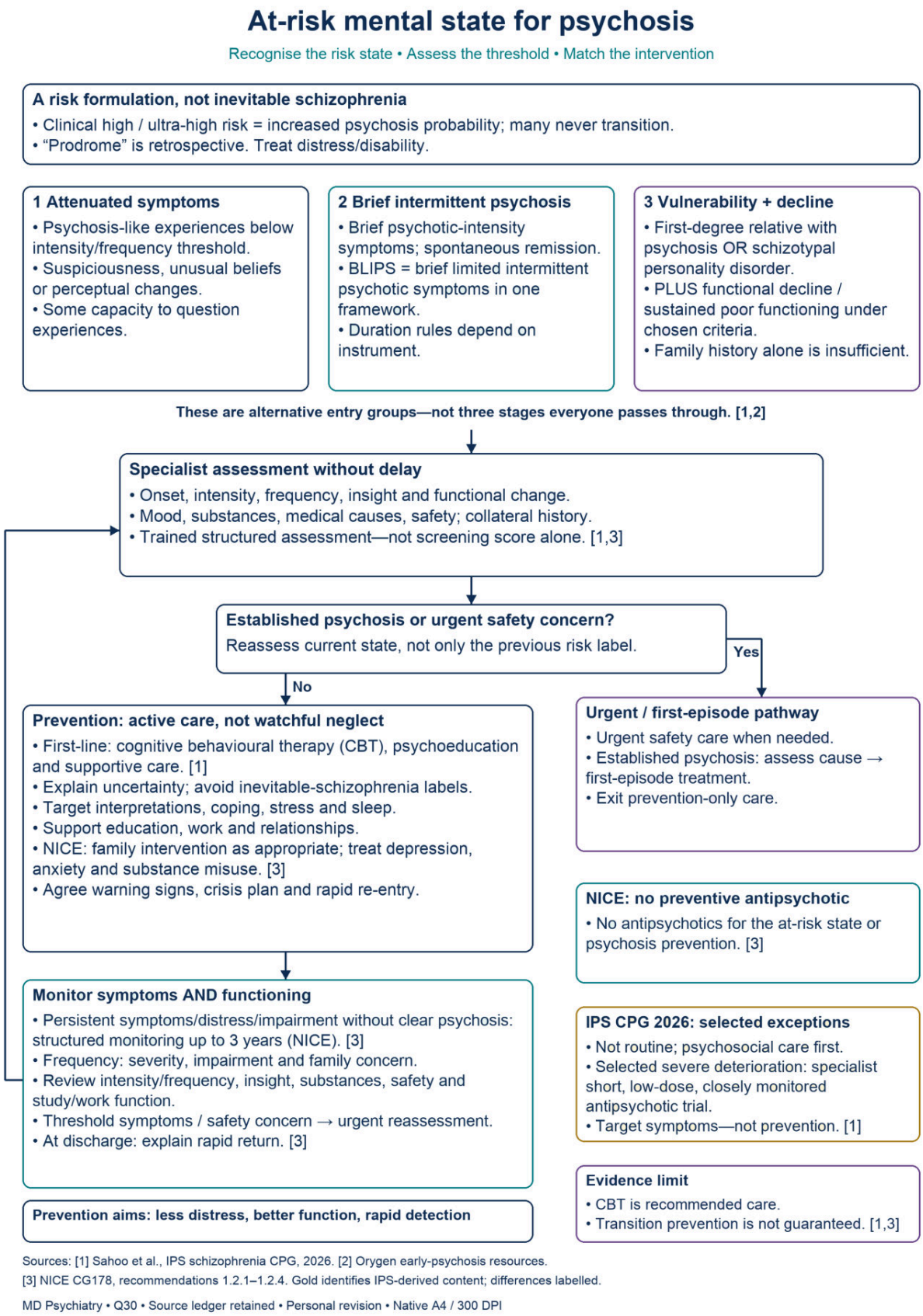
Sources: psychosis-area supplement Q28–31/Q44. Kay, Fiszbein & Opler PANSS (1987), doi:10.1093/schbul/13.2.261; Baer & Blais (2010), Ch10. PANSS primary methods PMC4270028; floor correction analysis PMC2879676. Overall & Gorham BPRS; Ventura et al. expanded BPRS manual v4.0, UCLA / UW ADAI. Scale summaries only; use authorised administration manuals.

Q28 supplement · PANSS minimum 30, not zero · 17 Sep 2026 A4 · 300 dpi

Psychosis rating scales PANSS and BPRS. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Schizophrenia: aetiology, phenomenology and course.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 30.1



At-risk mental state - Recognition assessment and management. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Schizophrenia: aetiology, phenomenology and course.

Credits and references: retained within the illustration.

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

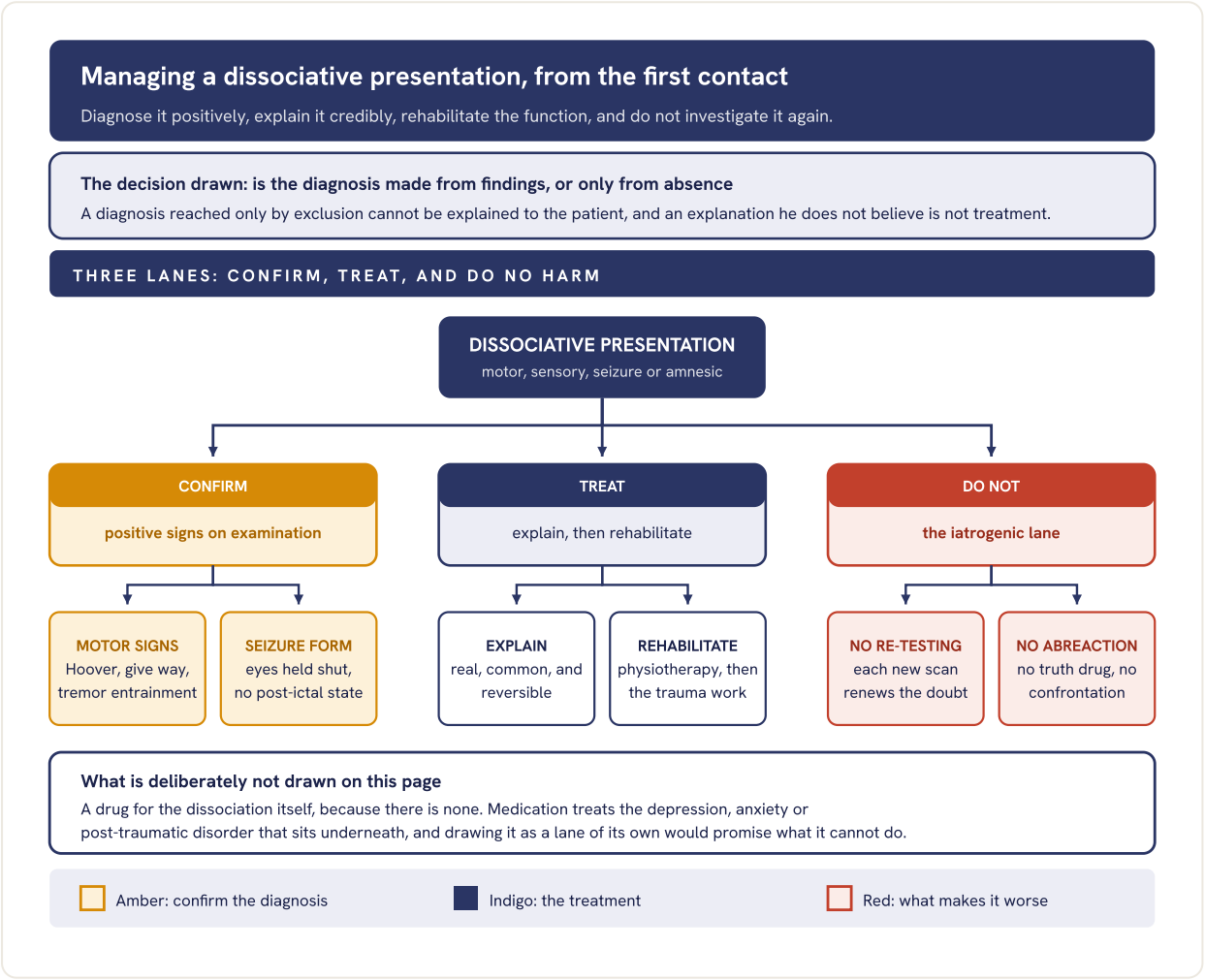


Fig 036.1 Confirmation drawn as a lane of its own, because the explanation given to the patient can only be as convincing as the findings it rests on.

- **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

WEAVE 100 · PAPER II · 36.1

Dissociative disorders: management

Safety → a positive shared explanation → syndrome-specific rehabilitation → review

1 Stabilise and assess · IPS dissociative emergencies CPG 2023

Check airway, physiology, glucose, injury, altered awareness and neurological red flags. Investigate according to findings. Assess self-harm, substances, safeguarding, psychiatric comorbidity and supports; admit when medical / diagnostic uncertainty or safety needs require it.

During an event: protect from injury, calm the setting, use simple reassurance / grounding and observe. Avoid humiliating confrontation, painful stimulation and unnecessary escalation.

2 Explain and agree goals · IPS psychological interventions 2020

Give a clear, nonjudgmental explanation and validate the experience. Discuss relevant positive findings, understandable mechanisms and the potential for improvement. Agree goals for daily function; involve family with consent and align the messages of the treating team.

Unexplained symptoms are not automatically dissociation; avoid promises of certain recovery.

Motor / sensory FND	Functional seizures	Amnesia / identity / DPDR
Specialist physiotherapy / occupational or speech therapy as appropriate. Retrain automatic movement, reduce excessive symptom-focused attention and practise graded functional tasks. Neurology and rehabilitation remain involved.	Explain the diagnosis and create an event / safety plan. Offer suitable psychological treatment; include carers if agreed. Evaluate epilepsy. Taper antiseizure medication only with the prescriber if no epilepsy / other indication.	Safety, grounding, regulation and functional restoration. Specialist psychotherapy for persistent identity / amnesic presentations. Explore trauma sensitively only when relevant; do not force recall or presume hidden trauma.

3 Match the intervention to evidence · named supplements

Motor FND: Nielsen et al. physiotherapy consensus (2015). Functional seizures: AAN (2025/2026) supports psychological treatment; no benzodiazepines or antiseizure drugs for functional seizures alone.

Treat comorbid depression, anxiety, PTSD or pain on their own indications. No medication treats dissociation as a single syndrome [IPS 2020/2023].

"Gain" · formulation concepts, not tests of honesty

Primary gain: a classical psychodynamic account of relief from internal conflict / distress through the symptom. Secondary gain: external consequences such as care, escape from demands or financial support. Neither is required for diagnosis; benefit does not prove conscious production.

Factitious disorder: intentional falsification without obvious external reward. Malingering: intentional production for external incentive. Both require evidence of intentional deception. FND / dissociation must not be inferred to be feigning.

4 Review function and safety · a continuing care plan

Coordinate follow-up; reinforce independence without withdrawing care. Track function, episodes and participation. Address maintaining stressors collaboratively. Avoid redundant tests, but reassess new or changed neurological signs and medical red flags.

Sources: Weave Q36 pp54–55, Q43 p62 (absolute "no re-testing" / universal trauma-work wording corrected). IPS psychological interventions (2020), S280–289; IPS dissociative emergencies (2023). Nielsen et al., JNNP (2015). AAN functional-seizure guideline (2025/2026). WHO CDDR / DSM-5-TR for diagnostic distinctions.

Q36 · FND = functional neurological disorder; DPDR = depersonalisation / derealisation · 17 Sep 2026 A4 · 300 dpi

Dissociative disorders management and gain. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-36.

Credits and references: retained within the illustration.

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

WEAVE 100 · PAPER II · 37.1

Depression rating scales

Choose the rater, population and purpose · record the exact version · follow change

What a score can and cannot do

Severity and screening overlap: PHQ-9 can do both. None independently establishes a depressive diagnosis, excludes bipolarity or replaces a suicide assessment. Compare the same version, language, time window and scoring method at follow-up.

Five core instruments · original summaries, not administration forms

Hamilton Depression Rating Scale HAM-D / HDRS-17	Clinician-rated severity; usually past week. Seventeen scored items, mixed 0–2 / 0–4 anchors; common total 0–52. Specify the form: 21/24-item versions and some anchor variants differ. Somatic / sleep weighting and rater variability are limitations. Common trial conventions: ≥50% reduction = response; ≤7 = remission, with clinical review.
Montgomery–Åsberg Depression Rating Scale MADRS	Clinician-rated change in depressive symptoms; commonly past week. Ten items × 0–6 = 0–60. Developed for sensitivity to treatment-related change. A score ≤10 is a common remission convention, not a universal diagnostic boundary; symptoms, function and trial definition still matter.
Beck Depression Inventory–II BDI-II	Self-report; 21 items × 0–3 = 0–63; past 2 weeks, including today. Conventional bands: 0–13 minimal, 14–19 mild, 20–28 moderate, 29–63 severe. Do not transfer BDI-I / IA cut-offs. Literacy, insight and somatic comorbidity affect interpretation; use the authorised form.
Patient Health Questionnaire–9 PHQ-9	Self-report, past 2 weeks; 9 items × 0–3 = 0–27. Bands: 0–4 minimal, 5–9 mild, 10–14 moderate, 15–19 moderately severe, 20–27 severe. ≥10 commonly prompts diagnostic assessment. Any self-harm response requires direct follow-up; a zero response does not rule out risk.
Geriatric Depression Scale GDS-15 / GDS-30	Older-adult screening, yes/no responses, usually past week. Totals 0–15 or 0–30: label the version. PhenX GDS-15 protocol: >5 warrants comprehensive assessment. Reduced somatic emphasis helps in older adults; cognition, communication and setting affect accuracy. Not a dementia test.

HAM-D-17: describe one scale in an exam

Purpose → clinician interview / observation → 17 scored items → mixed anchors and summed total → baseline and repeated follow-up. Covers mood, guilt, suicidality, sleep, activity, psychomotor and somatic / anxiety features. Explain somatic and sleep confounding, limited atypical-symptom coverage and the value of a structured interview / rater training.

Useful population-specific additions

Edinburgh Postnatal Depression Scale: perinatal screening. Cornell Scale for Depression in Dementia: clinician integrates patient and informant information. Hospital Anxiety and Depression Scale: separate anxiety / depression subscales in medical settings. Use each validated manual and local language version.

Interpret change, then act

A lower total is not automatically recovery. Check residual symptoms, function, adverse effects and safety. Never compare numerical totals across different scales as if the units were identical.

Sources: Weave Q37 p56 read / inspected; placement also cross-links depression area Q52/Q62. Baer & Blais handbook (2010), Ch2, actual local text; HAM-D anchor/range inconsistency corrected with primary study PMC3750710. PHQ-9: Kroenke et al. (2001), PMID11556941. BDI-II: Beck et al. (1996), Pearson; validation doi:10.2196/12051. GDS: Stanford/PhenX121704. MADRS: Montgomery & Åsberg (1979).

Q37 · depression scales · thresholds are version- and purpose-dependent · 17 Sep 2026 A4 · 300 dpi

Depression rating scales. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-37.

Credits and references: retained within the illustration.

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

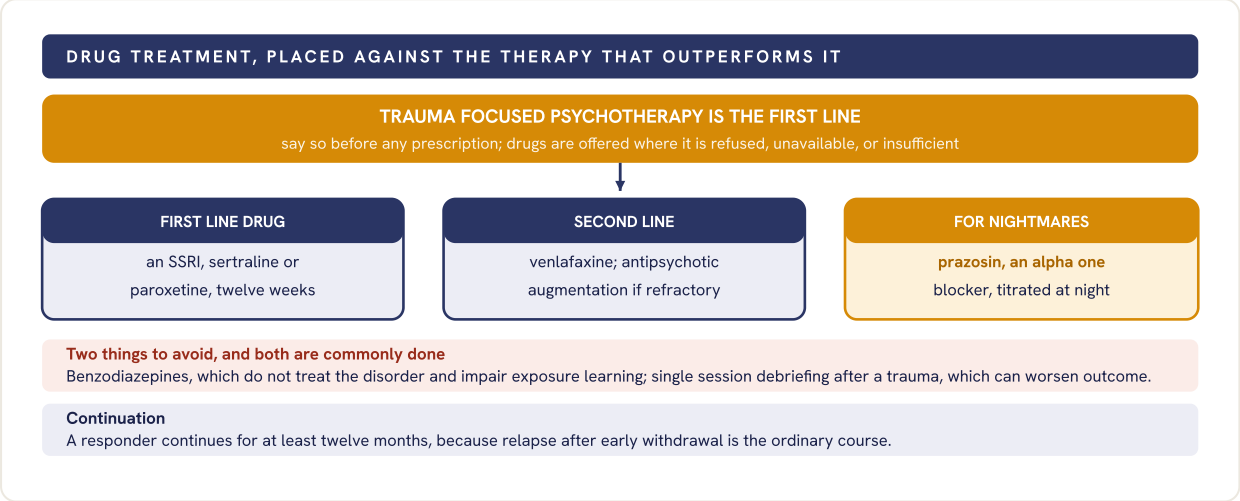


Fig 038.1 The drug lanes drawn beneath the therapy that outranks them, with the two avoidable harms carried as a red strip.

- **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.

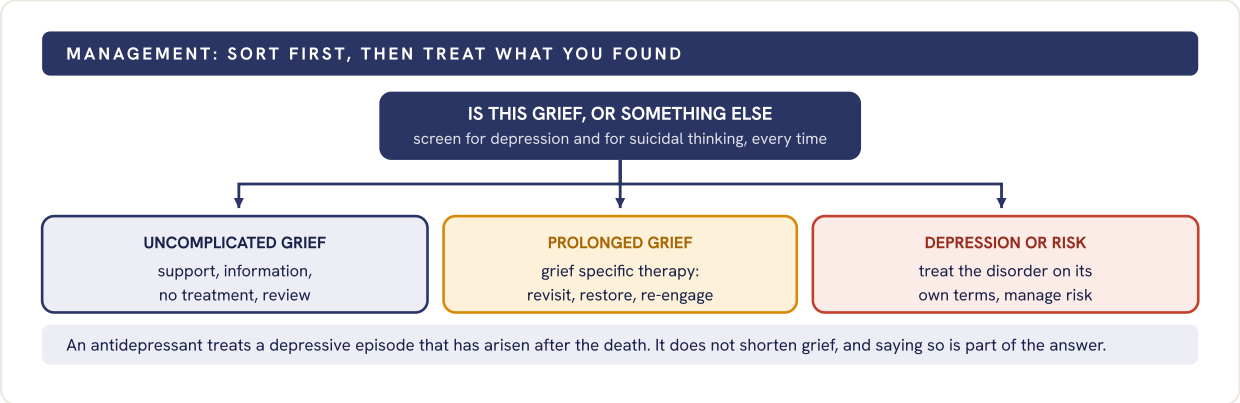


Fig 039.1 One sorting question with three destinations, drawn because the commonest error is treating all three the same way.

- **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.

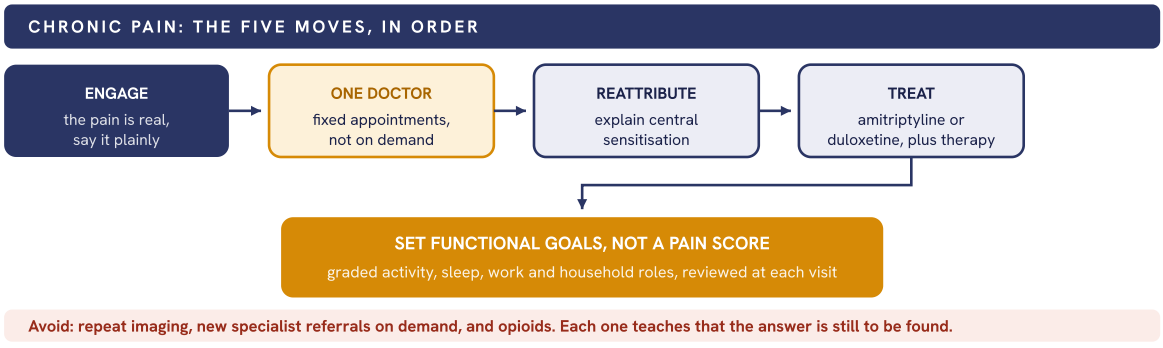


Fig 041.1 Five moves in sequence, ending in the goal that replaces cure, with the three iatrogenic traps carried along the foot.

- **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.

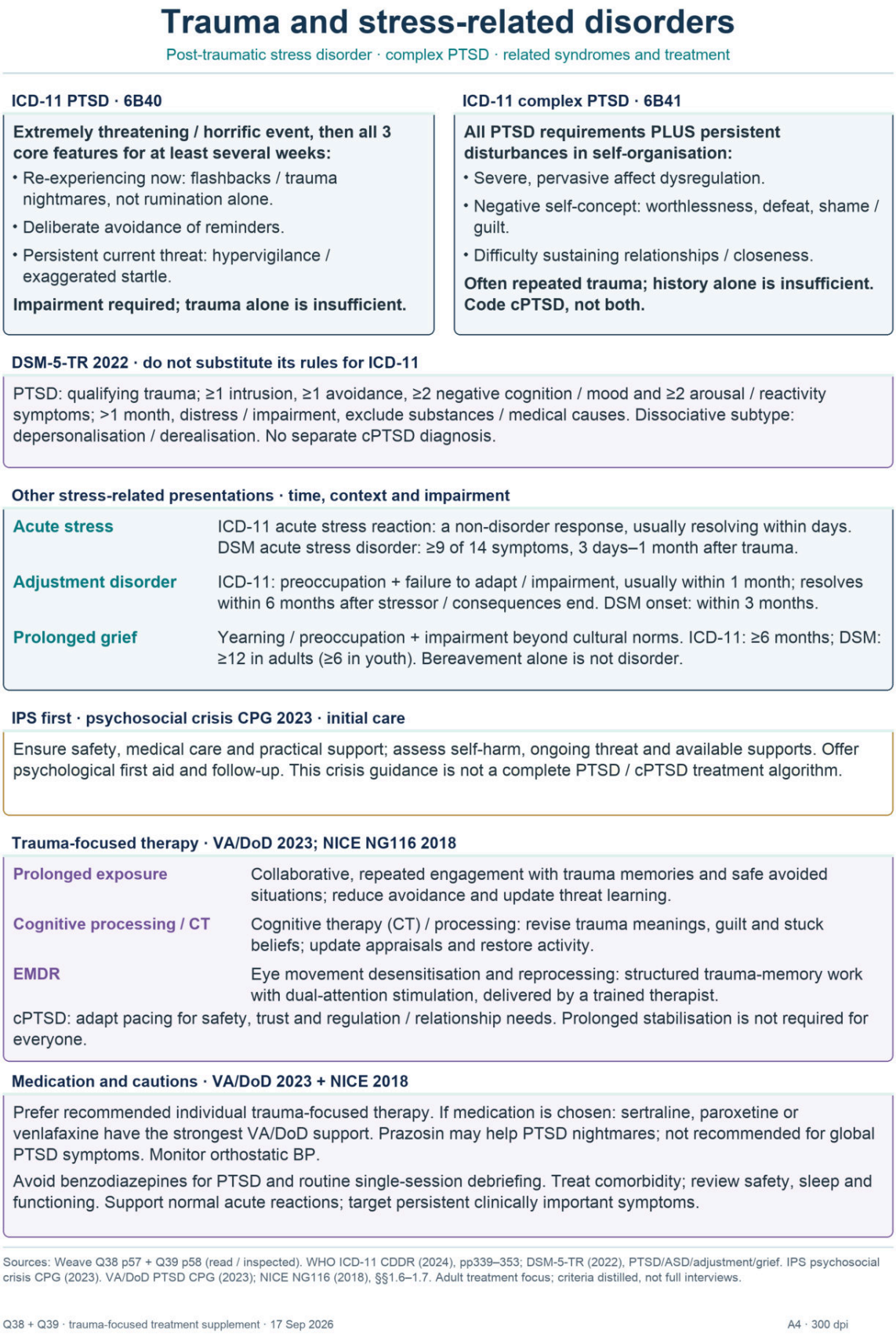
- **What the signs do not prove.** They support a positive diagnosis, they do not exclude coexisting disease, and none of them says anything about motivation. La belle indifference is unreliable and should not be offered as a sign.
- **How to use them.** Demonstrate one to the patient. A sign he can 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

WEAVE 100 · PAPER II · 38.1



Trauma PTSD complex PTSD and therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Trauma, dissociative and somatic symptom disorders.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 43.1

Dissociation: signs and differential

Positive evidence of altered function · no single sign proves a functional diagnosis

Nosology and examination frame

ICD-11 6B60: dissociative neurological symptom disorder; DSM-5-TR: functional neurological symptom disorder (conversion disorder). Diagnose from compatible positive findings, not simply normal tests. Symptoms are involuntary; trauma / a stressor is not required in every case.

IPS 2023: assess medical differentials and comorbidity; disease can coexist.

Positive functional signs · IPS 2023 + neurological consensus 2015/2020

Hoover sign	Weak voluntary hip extension improves during opposite hip flexion against resistance. Interpret with pain, effort, cognition and the whole neurological examination.
Hip abductor sign	Weak hip abduction improves during resisted abduction of the opposite hip; supports inconsistency between voluntary and automatic movement.
Tremor	Distractibility / entrainment: tremor changes or follows a rhythm tapped by the other limb. Specialist assessment may be needed.
Weakness / drift	Collapsing weakness or drift without pronation can support FND; pain / fatigue confound. Normal reflexes alone do not establish FND.
Sensation / vision	Reproducible non-anatomical inconsistency, e.g. a tubular visual field; limited specificity. Midline splitting can also occur in neurological disease.
Gait / balance	Inconsistency across tasks, knee buckling despite preserved strength or improvement with distraction. Bizarre appearance alone is insufficient.
Straight-leg raise	A radicular-tension test, not a test confirming dissociation. Compare function across safe tasks; do not use painful “proof” or deceptive provocation.

Psychogenic nonepileptic seizures (PNES) versus epileptic seizures

May favour functional	Fluctuating / prolonged episodes, asynchronous movements, eye closure, or retained responsiveness during bilateral movements. None is diagnostic alone.
May favour epileptic	Stereotyped evolution, ictal onset from sleep, postictal confusion or lateral tongue injury; absence does not exclude epilepsy.
Other mimics	Syncope can include jerks; assess posture, prodrome and cardiovascular cause. Also panic, sleep disorders, movement disorders, hypoglycaemia and intoxication.

Incontinence, injury, eye closure or a normal EEG alone cannot decide the diagnosis.

AAN functional-seizure guideline · released 2025; journal 2026

Use history, witness / video evidence and, where feasible, typical-event video-EEG with expert interpretation. Normal interictal EEG does not exclude epilepsy; some epileptic seizures lack a clear scalp EEG correlate.

Assess coexisting epilepsy; investigate diagnostic uncertainty appropriately.

Other dissociative presentations · identify the syndrome

Amnesia / fugue	Autobiographical memory loss beyond ordinary forgetting; fugue includes travel / wandering. Exclude neurological, substance and sleep-related causes.
Depersonalisation / derealisation	Detachment from self / unreality of surroundings, with reality testing intact; assess distress, substances, panic and neurological illness.
Identity / trance	Disruption of identity and agency ± memory gaps; DID / partial DID need careful specialist assessment. Do not pathologise accepted cultural or religious practices.

Sources: Weave Q43 p62 and Q36 pp54–55 (read / inspected; absolute signs corrected). WHO ICD-11 CDDR (2024), dissociative disorders; DSM-5-TR (2022). IPS dissociative emergencies CPG (2023), Table11. Nielsen et al., JNNP (2015); Nonnekes et al., Neurology (2020). AAN functional seizures guideline (Dec2025 / Jan2026).

Q43 · signs support diagnosis; disease can coexist · 17 Sep 2026 A4 · 300 dpi

Dissociation positive signs and seizure differential. Read across the labelled contrasts to distinguish the related concepts. Section companion: Trauma, dissociative and somatic symptom disorders.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 43.2

Gaits in psychiatric practice

Observe the pattern → localise / review medicines → treat the cause and rehabilitate

Start with medical assessment · do not attribute gait to psychiatry by default

Observe initiation, base, stride, arm swing, turns and balance with fall protection. Examine power, tone, reflexes, sensation, coordination and cognition; review medicines and intoxication. Acute onset, focal deficit, fever, marked confusion or inability to stand needs urgent medical assessment.

SCHEMATIC FOOTFALLS	PATTERN / DISCRIMINATOR	CAUSE-BASED REMEDIATION
	**Parkinsonian** Short shuffling steps, reduced arm swing, en-bloc turns; bradykinesia / rigidity. Drug-induced or degenerative.	Review dopamine-blocking drugs; adjust with prescriber. Neurology, physiotherapy and cueing; assess falls / orthostasis.
	**Akathisia** Pacing / shifting plus inner restlessness; not a specific gait disorder. Ask about distress and medication timing.	Review causative drug, reduce / switch if appropriate; treat confirmed akathisia. Do not escalate antipsychotic for “agitation” reflexively.
	**Cerebellar ataxic** Broad base, variable steps / veering, impaired tandem gait; may have nystagmus or dysmetria.	Assess intoxication, toxicity, stroke / cerebellar disease; treat cause, balance training and suitable aids.
	**Sensory ataxic** Stamping / watching feet; worse without visual input, impaired proprioception, positive Romberg.	Investigate neuropathy / posterior columns (including B12); treat cause, lighting, foot care and gait rehabilitation.
	**Spastic / hemiparetic** Stiff circumduction on one side; bilateral spastic gait may scissor. Upper motor-neuron signs guide localisation.	Stroke / cord assessment as indicated; rehabilitation, orthoses and targeted spasticity management.
	**Frontal / magnetic** Start hesitation, feet “stuck”, short broad-based steps; frontal signs ± cognitive / urinary symptoms.	Assess vascular / frontal disease or normal-pressure hydrocephalus; selected specialist treatment and falls rehabilitation.
	**Steppage** High hip / knee lift to clear foot drop; foot slap. Check ankle dorsiflexion and peripheral localisation.	Identify peroneal / root / neuropathic cause; physiotherapy and ankle-foot orthosis when appropriate.
	**Functional / astasia-abasia** Inconsistent balance / walking despite preserved movement in other tasks. Astasia: inability to stand; abasia: inability to walk.	Positive-sign diagnosis, shared explanation and motor retraining. Astasia-abasia can also occur with organic disease; appearance alone is insufficient.

IPS first where applicable · scope and evidence

IPS schizophrenia CPG (2026): identify / manage medication-related movement effects. IPS dissociation CPGs (2020/2023): assessment and rehabilitation. No single IPS gait algorithm found; neurological pattern guidance and FND physiotherapy consensus supplement these.

Sources: Q43 supplement (no dedicated Weave gait question). IPS schizophrenia CPG (2026); IPS dissociation (2020/2023). Stanford Medicine25 gait examination; Pirker & Katzenschlager, Wien Klin Wochenschr (2017); Nonnekes et al., Neurology (2020); Nielsen et al., JNNP (2015). Footfalls are schematic aids, not diagnostic tests.

Q43 supplement · wider / smaller steps illustrated, not patient trajectories · 17 Sep 2026 A4 · 300 dpi

Gait patterns and remediation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Trauma, dissociative and somatic symptom disorders.

Credits and references: retained within the illustration.

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

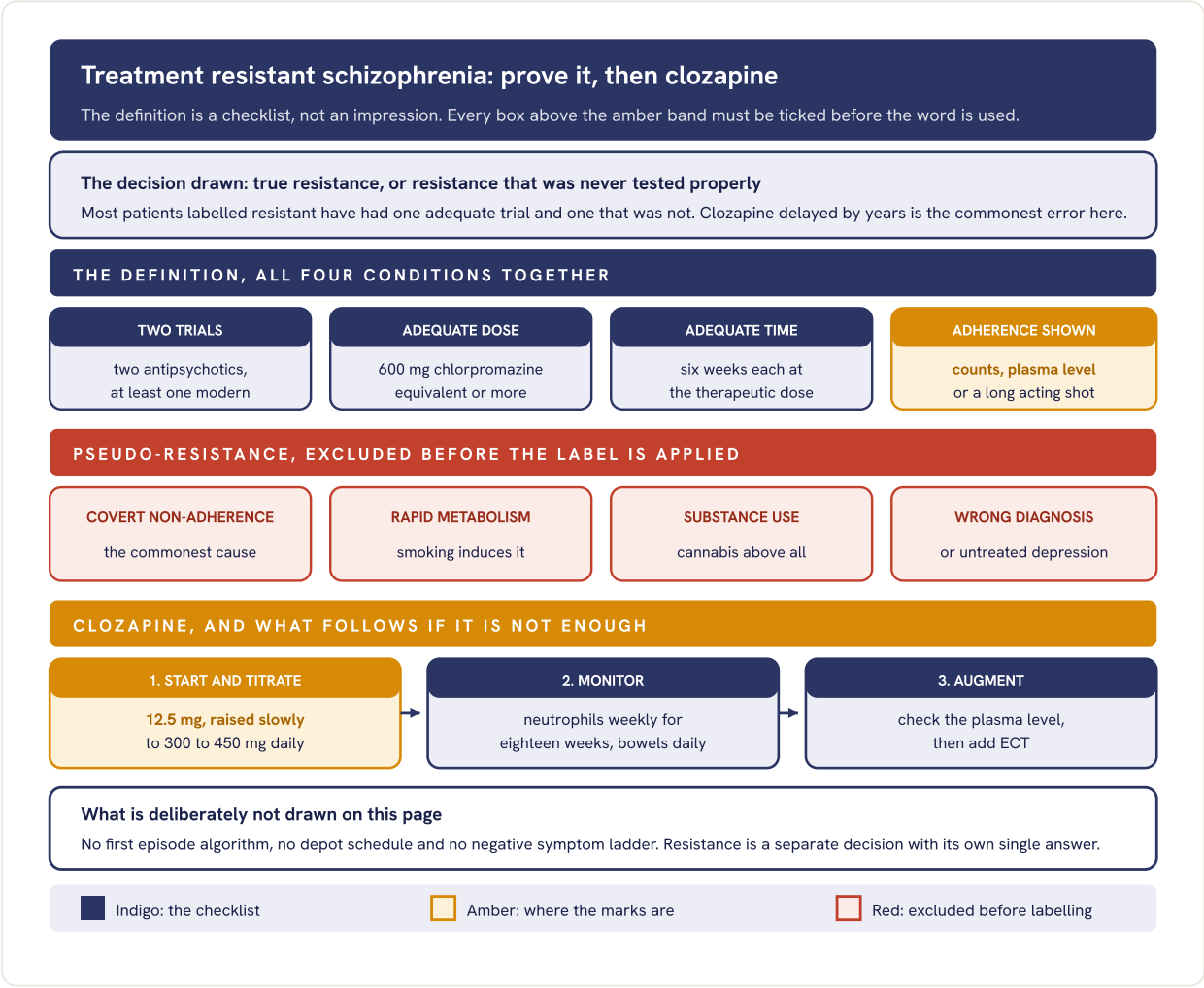


Fig 044.1 The four conditions that define resistance, the four causes of apparent resistance that are excluded first, and the clozapine sequence that follows once the label is earned.

- **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

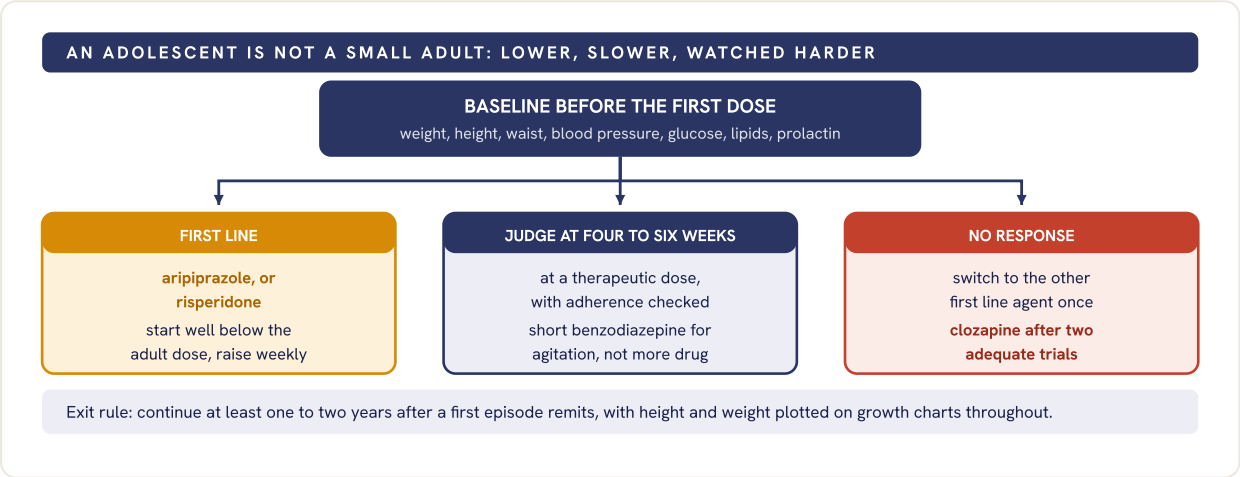


Fig 045.1 First episode pharmacology in an adolescent: the baseline that precedes the first dose, the two first line agents, and the point at which clozapine becomes the answer.

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.

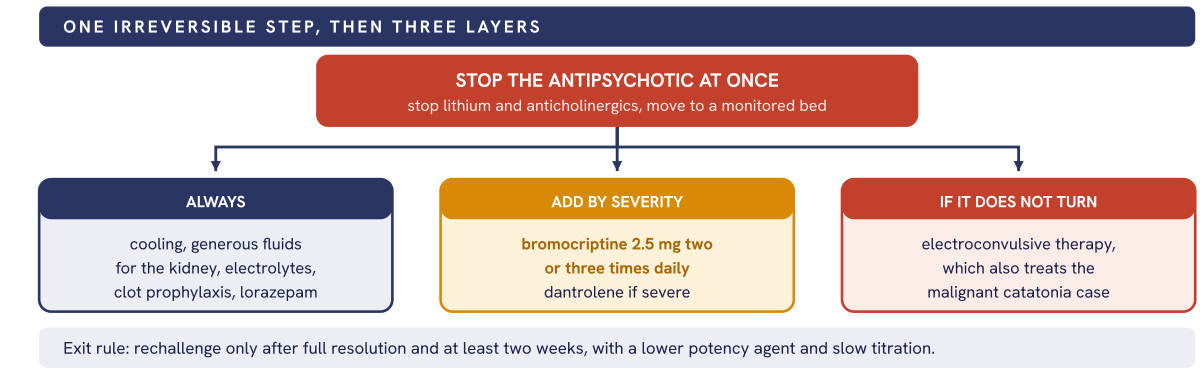


Fig 046.1 One irreversible action, then three layers: always, by severity, and if it does not turn.

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

NMS, serotonin toxicity and malignant catatonia

Exposure + motor examination + course | Overlap is common; no single laboratory test settles the diagnosis

EMERGENCY FIRST — assessment and treatment run together

- Stop suspected drugs; airway/breathing/circulation; urgent medical/critical-care review. Active cooling + monitored benzodiazepines.
- Fluids/electrolytes; temperature, ECG, urine output, CK, potassium, renal function; aspiration + thrombosis prevention.
- Severe hyperthermia, rigidity or organ failure → ICU. Never wait for criteria or laboratory confirmation.

Clue	NMS [1,3,A]	Serotonin toxicity [2,4,5]	Malignant catatonia [3,A]
Trigger / tempo	Dopamine antagonist (antipsychotic/antiemetic) OR dopaminergic withdrawal Usually days after start/escalation	Serotonergic addition / dose ↑ / overdose / interaction Usually hours; often <24 h; delays possible MAOI + reuptake inhibitor: high risk	Drug exposure NOT required Mood, psychotic or medical illness Catatonic prodrome may precede deterioration
Motor / mental pattern	Generalized lead-pipe rigidity Reflexes often normal/reduced Atypical cases: little rigidity or fever	Clonus + hyperreflexia (legs > arms) Tremor, agitation, sweating, diarrhoea Severe rigidity may mask clonus	Excitement OR stupor; mutism, posturing, negativism Fever + autonomic deterioration Rigidity overlaps with NMS

NMS: Gurrera 2011 weights [1]

Total 100; not an all-or-none checklist.
- Dopamine antagonist / agonist withdrawal within 72 h: 20.
- Oral temperature >38°C ≥2 times: 18.
- Rigidity: 17; altered consciousness: 13.
- CK ≥4 × ULN: 10.
- Sympathetic lability: 10; ≥2 of:

BP ↑≥25% above baseline; BP swing ≥20 diastolic / ≥25 systolic mmHg in 24 h; sweating; incontinence.
- HR ↑≥25% AND RR ↑≥50% above baseline: 5.
- Alternative-cause work-up negative: 7.

Suspicion can precede the complete pattern.

Hunter 2003: ANY ONE branch [2,5]

Serotonergic exposure + one of:
1. Spontaneous clonus.
2. Inducible clonus + (agitation OR sweating).
3. Ocular clonus + (agitation OR sweating).
4. Tremor + hyperreflexia.
5. Hypertonia + temperature >38°C + (ocular OR inducible clonus).

Derived in overdose; caution elsewhere. Exclude other causes. Onset clock / absent visible clonus alone cannot exclude severe toxicity.

Catatonia + malignant features [3]

DSM-5-TR: ≥3 of 12 signs
- Stupor • Catalepsy • Waxy flexibility
- Mutism • Negativism • Posturing
- Mannerism • Stereotypy • Grimacing
- Agitation unaffected by external stimuli
- Echolalia • Echopraxia

Malignant: hyperthermia + autonomic dysfunction; no separate universal numerical criteria.
Track Bush–Francis score. Lorazepam response supports, not confirms; a negative challenge does not exclude.

NMS treatment [3]

- Stop dopamine blockers + anticholinergics; supportive care.
- Selected severe cases: bromocriptine/amantadine or dantrolene; limited evidence.
- Persistent/severe or catatonic overlap → consider ECT.
- Withdrawal syndrome → specialist restoration of dopaminergic therapy.

Serotonin toxicity treatment [4]

- Stop serotonergic drugs; benzodiazepines + cooling.
- Severe hyperthermia/rigidity → airway control + non-depolarizing paralysis in ICU.
- Cyproheptadine oral/NG: optional adjunct; outcome benefit uncertain; never delay resuscitation.
- Antipyretics do not correct muscular heat.

Malignant catatonia: BAP 2023 [3]

- Withhold dopamine blockers; treat cause.
- Lorazepam 8 mg/day → up to 24 mg/day by response/tolerance; respiratory/sedation monitoring.
- Arrange ECT early. Partial/no response within 48–72 h → bilateral ECT; earlier if deteriorating.
- No prolonged medication trial.

SHARED TRAPS [1,3,A]

- CK ↑, leukocytosis, low iron: nonspecific. NMS–catatonia relationship: debated. Repeat drug history + motor examination.
- Exclude infection/encephalitis, seizures, heat illness, anaesthetic malignant hyperthermia, anticholinergic toxicity and withdrawal.

[1] Gurrera 2011 [2] Dunkley 2003 [3] BAP 2023 [4] Chiew & Isbister 2025 [5] Isbister 2007 [A] Weave "Heat and Iron", critically checked

CK = creatine kinase • ULN = upper limit of normal • HR/RR = heart/respiratory rate • ECT = electroconvulsive therapy • Q46

NMS serotonin toxicity and malignant catatonia - Differential and emergency management. Read across the labelled contrasts to distinguish the related concepts. Read with II-46.

Credits and references: retained within the illustration.

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.

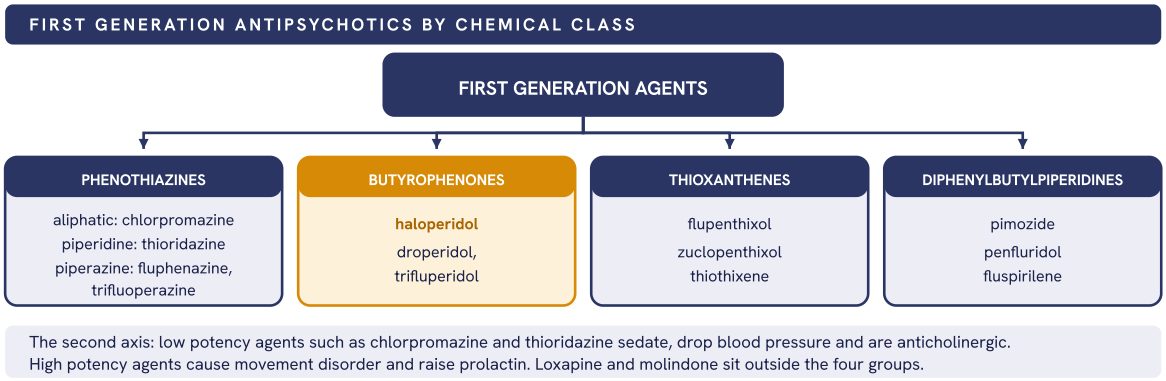


Fig 047.1 One axis, four branches, with the potency axis carried as a strip along the foot.

- **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.

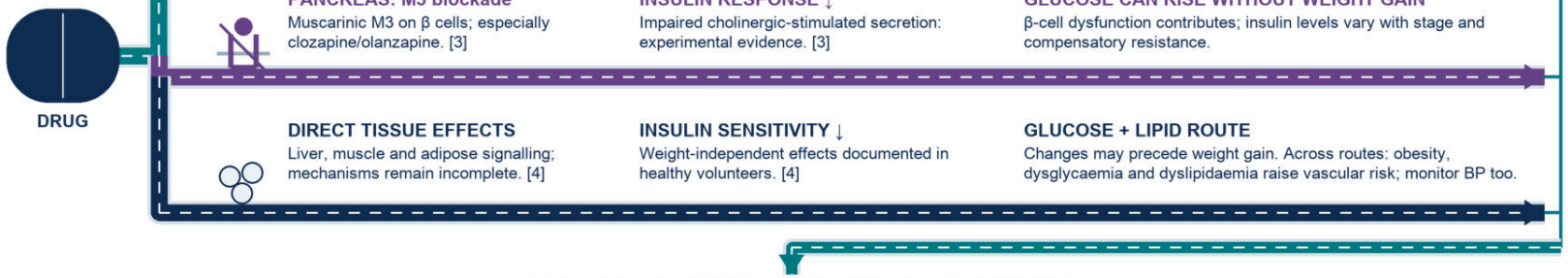
WHAT EARNS THE MARKS

- **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

Antipsychotics: the roads to metabolic harm

Receptor effects → appetite, insulin and tissue metabolism → prevention from the first prescription | Adult clinical pathway



PREVENT → MEASURE → ACT EARLY → REASSESS

Risk [5]	Antipsychotic
Higher	Clozapine • Olanzapine
Intermediate	Quetiapine • Risperidone • Paliperidone
Lower	Aripiprazole • Lurasidone • Ziprasidone • Cariprazine

1 PREVENT from the first prescription

- Risk varies by outcome; lower ≠ zero.
- Efficacy / relapse risk: retain effective clozapine where appropriate.
- Diet, activity, sleep, smoking support; affordable plan.
- Review other weight-promoting drugs.

IPS schizophrenia CPG 2026 [6]

- Baseline / follow-up: weight + BMI.
- Metabolic checks: 1–3 months; annually; sooner if abnormal.
- Structured lifestyle + medical care; assign results/action.

2 BASELINE — NICE CG178 [7]

- Weight/BMI • Waist • Pulse/BP
- Fasting glucose OR HbA1c • Lipids
- Personal/family history + lifestyle

Follow-up [7]	Timing
Weight	Weekly ×6 weeks; 12 weeks; 1 year; annually
Waist	Annually
Pulse/BP, glucose OR HbA1c, lipids	12 weeks; 1 year; annually

ACT EARLY — do not wait for 7%

- More checks: rapid change, abnormal tests/symptoms; reassess after switching.
- Track % change from baseline: >3% first-year gain = metformin-prevention threshold [8]; not universal diagnostic cutoff.
- Act on glucose/lipid/BP abnormalities despite stable weight.

3 METFORMIN — PREVENT EARLY [8]

- 2025 consensus (online 2024): co-start with clozapine/olanzapine.
- Medium risk: co-start if hypertension, dyslipidaemia, diabetes/prediabetes, BMI >25 OR age 10–25.
- Any drug: start if >3% gain in year one. Strong recommendation; low-certainty evidence.
- Off-label: e.g. 500 mg/day with food → 1–2 g/day as tolerated.
- Renal check: avoid eGFR <30 mL/min/1.73 m²; adjust for impairment.
- Monitor GI effects/B12; assess lactic-acidosis risks.

ESCALATE WITH CLINICAL STABILITY

- Joint switching review; treat diabetes, lipids and BP to medical guidelines.
- Eligible obesity/diabetes: consider GLP-1 agonist; RCT benefit in selected antipsychotic-treated groups.
- Check contraindications, tolerability, access. [7,9]

URGENT EXIT — possible hyperglycaemic crisis

- Polyuria/thirst + vomiting, abdominal pain, dehydration, deep breathing or confusion → urgent glucose/ketones + emergency assessment. Do not wait for HbA1c or the next scheduled review.

[1–4] Mechanistic studies [5] Pillinger 2020 [6] IPS 2026 [7] NICE CG178, amended 2021 [8] Carolan 2025 [9] Semaglutide RCTs 2025 • Source ledger

Original pathway illustration • Preclinical mechanisms qualified; receptor affinity alone does not predict individual risk • Q50

Antipsychotic metabolic effects - Mechanism roads prevention and management. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with II-50.

Credits and references: retained within the illustration.

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.

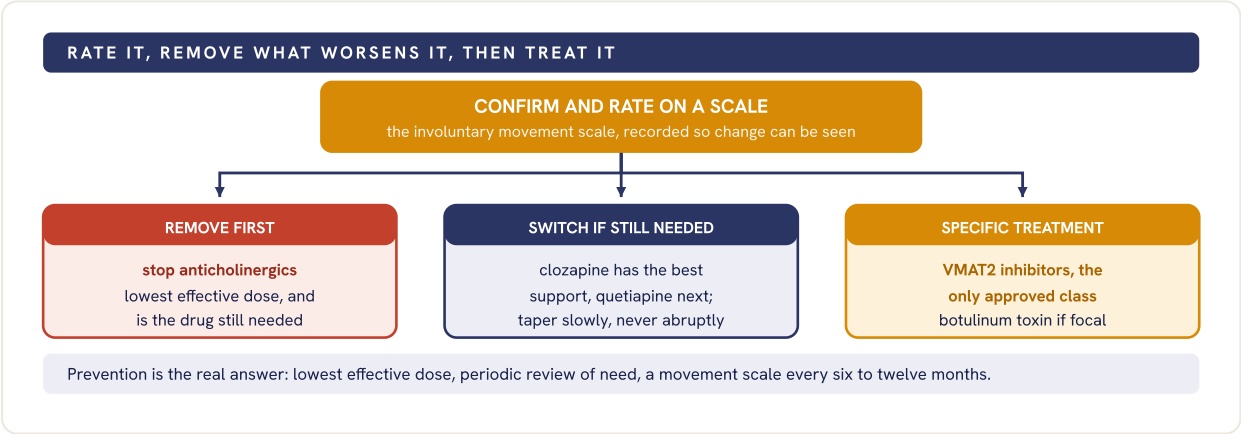


Fig 051.1 Management in order: rate the movements, remove what worsens them, switch if an antipsychotic is still required, then treat.

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

Treatment resistance: definitions and adequate trials

Schizophrenia • Unipolar major depression • Mania | Name the source; intolerance is not an adequate efficacy trial

Exclude pseudo-resistance first — IPS 2026 [4–6]

- Confirm diagnosis/current polarity; measure symptoms + function.
- Audit drug, dose, duration, adherence, interactions and levels; address substances, medical causes, adverse effects and psychosocial barriers.

SCHIZOPHRENIA: THREE DIFFERENT CRITERION SETS

Domain	Original Kane 1988 [1]	Modified Kane: 2016 [2]	TRRIP 2017 [3]
Trial history	≥3 trials; ≥2 chemical classes; previous 5 years	≥2 sequential different antipsychotics	≥2 different antipsychotics
Dose + duration	Each ≥6 weeks; ≥1000 mg/day CPZ	Each ≥6 weeks; ≥400 mg/day CPZ	Each ≥6 weeks; therapeutic dose Table benchmark ≥600 mg/day CPZ
Symptoms + function	No significant relief / good function for 5 years BPRS ≥45; CGI ≥4 ≥4 on ≥2: conceptual disorganization, suspiciousness, hallucinations, unusual thought content	≥Moderate on ≥1 PANSS positive item GAF <59 No symptom/function improvement ≥6 months	≥Moderate symptoms ≥12 weeks ≥Moderate functional impairment
Verification	Prospective haloperidol ×6 weeks: up to 60 mg/day or higher (historical) Response: ≥20% BPRS reduction AND (CGI ≤3 OR BPRS ≤35); responders excluded	Adherence: interview + pharmacy / medical records This study's "modified Kane"; no universal modified rule	Adherence ≥80%; ≥2 methods + ≥1 plasma level Optimal: prospective ≥6 weeks with <20% improvement + ≥1 LAI trial, generally ≥4 months
Interpretation	Historical doses ≠ current targets	Name Mouchlianitis 2016 explicitly	Research benchmark; do not delay indicated clozapine

UNIPOLAR TRD — IPS 2026 + APAC 2019 [5,7]

- Persistent MDD after ≥2 adequate antidepressants; document this episode + adherence.
- APAC: adequate drug-specific dose ×6–8 weeks per trial; brief/low-dose exposure does not count.
- Validated symptom scale + function; partial response ≠ remission; definitions vary.
- Screen past mania/hypomania: not bipolar-depression criteria.

MANIA — no universal definition [8,9]

- CINP 2020: inadequate response after 8–10 weeks of evidence-based antimanic treatment; assess mania AND emerging/worsening depression. Phase-specific consensus.
- 2024 proposal: DSM-5 mania ≥6 weeks; YMRS ≥18 OR MRS ≥13; ≥1 traditional mood stabilizer AND ≥1 antipsychotic, sequential/concurrent. Each therapeutic level/registration-trial dose ≥3 weeks. Proposal ≠ universal consensus.

Record before escalating — IPS bipolar "2025 update", published January 2026 [6]

- Each trial: dates; target/achieved dose or level; adherence evidence; tolerability; symptom + functional change; criterion set used.
- IPS broad TRBD: ≥2 adequate evidence-based agents; exclude pseudo-resistance. Urgent severe illness → treat now; do not wait for research thresholds.

[1] Kane 1988 [2] Mouchlianitis 2016 [3] TRRIP 2017 [4–6] IPS 2026 [7] APAC 2019 [8] CINP 2020 [9] TRM proposal 2024

CPZ = chlorpromazine equivalents • Full source ledger retained • Gold = verified IPS guidance • Q44/52/57

Treatment resistance - Definitions and adequate trial criteria. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Schizophrenia: treatment and antipsychotics.

Credits and references: retained within the illustration.

Treatment resistance: management pathways

Verify → optimize → reassess | Short decision nodes; urgent treatment can enter at any stage

COMMON ENTRY — safety + a verified treatment history [4–6]

- Suicide/violence risk, catatonia, exhaustion, dehydration, capacity → hospital care when indicated.
- Correct pseudo-resistance; measure symptoms/function; agree goals. Psychological care, family support and rehabilitation throughout.

SCHIZOPHRENIA

Two adequate failures → clozapine [4]

- Verify adherence + disabling psychosis.
- IPS: ≥1 trial of amisulpride, olanzapine or risperidone.
- Offer clozapine promptly.

Start + optimize clozapine [4]

- Shared decision; gradual titration.
- ANC; myocarditis; constipation/ileus; seizures; metabolic health.
- Smoking, infection, drug interactions.

Adequate exposure? TRRIP [3]

- Trough ≥350 ng/mL.
- ≥3 months AFTER therapeutic level.
- Individual response + tolerability govern dose.

Persistent symptoms [4]

- Specialist reassessment.
- Consider ECT / selected drug augmentation.
- Drug-combination evidence limited: define target; stop ineffective additions.

Maintain gains [4]

- Effective medication + monitoring.
- CBT for psychosis; family intervention; supported work; cognitive rehabilitation.

UNIPOLAR MAJOR DEPRESSION

Optimize initial treatment [5,10]

- Tolerated therapeutic dose + duration.
- Include psychotherapy.

SWITCH — No benefit / poor tolerance

AUGMENT — Partial response + tolerance

Choose the next step [5,10]

- Switch antidepressant OR augment.
- Aripiprazole / lithium / supported alternative.
- CANMAT 2023: brexpiprazole also first-line adjunctive. Monitor specific harms.

Persistent burden [5,10]

- Shared choice: rTMS, ECT or supervised ketamine/esketamine as appropriate/available.
- Ketamine: cardiovascular + dissociation monitoring; continuation plan.

URGENT / PSYCHOTIC route [5]

- Severe suicidality, catatonia, rapid-response need → consider EARLY ECT.
- Psychotic depression: antidepressant + antipsychotic OR ECT.

Continuation + relapse prevention [5]

- Effective treatment + psychotherapy + relapse plan.
- Track function / quality of life.
- Difficult-to-treat depression is broader than medication failure.

MANIA

Remove contributors + optimize [6]

- Stop antidepressants in mania.
- Address substances + sleep loss.
- Verify antimanic dose/adherence and lithium/valproate levels.

Monotherapy inadequate? [6]

- Lithium/valproate + evidence-based antipsychotic (add either to the other).
- Severe mania: combination often appropriate FROM THE OUTSET.

Still not responding [6,8]

- Recheck diagnosis + levels.
- Switch/optimize supported combination.
- Specialist clozapine for refractory mania; evidence weaker than TRS.

ECT — early when urgent [6]

- Resistant mania.
- Earlier if dangerous, catatonic, medically exhausting or rapid control needed.
- Do not wait through every drug step.

Stabilize + maintain [6]

- Prior response + predominant polarity + safety.
- Lithium: renal/thyroid/level monitoring.
- Valproate: reproductive, hepatic, hematological risks.

[3] TRRIP 2017 [4] IPS schizophrenia 2026 [5] IPS depression 2026 [6] IPS bipolar 2026 [8] CINP 2020 [10] CANMAT 2023 update

Evidence and safety qualifications preserved • Urgent nodes apply at any stage • Q44/52/57

Treatment resistance - Management flowcharts. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Schizophrenia: treatment and antipsychotics.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 44.3

Clozapine

Treatment-resistant schizophrenia · start safely · monitor beyond the blood count

Indications · IPS schizophrenia 2026

Treatment resistance after ≥2 adequate antipsychotic trials; verify adherence. Selected patients with persistent suicidality or aggression.

Therapeutic mechanism · not settled

Weak / transient D2 blockade plus serotonergic and other receptor actions.

Unique efficacy in treatment resistance is not mechanistically explained.

Start, titrate, restart · adult example

- Start 12.5 mg at night; increase 12.5–25 mg every 1–3 days if tolerated. Check BP, pulse, temperature and sedation before each increase.
- Often 200–450 mg/day; Indian / Asian patients may need less. Titrate slower when frail, in community or during inflammation.
- Gap >48 h: restart 12.5 mg once / twice on day 1, then re-titrate. Lost hypotension tolerance makes the old full dose unsafe.
- Clozapine alone: ~350–600 ng/mL reference range. Sample ~12 h post-dose after ≥5 days on a stable dose; response varies.
- Smoking induces CYP1A2. Stopping smoking, infection, fluvoxamine or ciprofloxacin can raise levels → review dose / level.

Monitoring · local programme governs

- Baseline: full blood count / absolute neutrophil count (ANC), weight, BP, glucose / HbA1c, lipids, ECG, renal / liver tests, bowel history; C-reactive protein (CRP) and troponin.
- UK example [Maudsley 2025]: weekly 18 weeks → fortnightly to 1 year → monthly. Follow Indian programme / low-baseline-ANC rules.
- Fever / sore throat → urgent ANC. Falling ANC needs prompt review; earlier action thresholds vary. ANC <500/µL → stop and seek urgent haematology care.
- Early treatment: daily vitals / symptoms; weekly CRP + troponin for 4 weeks [Maudsley]. Follow weight and bowels; metabolic tests at ~3 months, then 6–12 monthly.

Adverse-effect mechanisms · distinguish established actions from hypotheses

Agranulocytosis	Idiosyncratic immune / reactive-metabolite injury proposed; exact mechanism unproven. Not a predictable dose-dependent effect.
Constipation → ileus	Muscarinic blockade → gut hypomotility. Daily bowel record; preventive stimulant ± osmotic laxative. Avoid extra anticholinergics.
Myocarditis / heart failure	Early myocarditis: immune / hypersensitivity hypothesis, not fully established. Cardiomyopathy can emerge months to years later.
Sedation / orthostasis	H1 blockade → sedation; α1 blockade → postural hypotension. Sialorrhoea is multifactorial; with sedation it increases aspiration risk.
Metabolic / neurological	H1 / 5-HT2C pathways contribute to appetite / weight gain; diabetes is multifactorial. Seizure risk rises with dose / plasma exposure.

Urgent red flags · do not attribute these to routine titration

Chest pain, dyspnoea, persistent tachycardia or fever → urgent myocarditis work-up. CRP >100 mg/L OR troponin >2× upper limit: stop clozapine and urgently assess [Maudsley protocol].

Abdominal pain / distension, vomiting or overflow diarrhoea → urgent assessment for obstruction / ileus; withhold clozapine if suspected. Bulk-forming laxatives are unsuitable for severe hypomotility.

Landmarks · primary trials, not IPS guidance

Kane 1988: 268 refractory patients, 6-week double-blind trial; response 30% with clozapine vs 4% with chlorpromazine. Established efficacy in a selected resistant group; too short for long-term safety.

InterSePT 2003: 980 high-risk patients, 2 years; open-label, masked outcome ratings. Suicidal behaviour lower than with olanzapine (HR 0.76; 95% CI 0.58–0.97). Suicide mortality was not significantly different.

Sources: Meyer & Stahl, The Clozapine Handbook (first published 2020), pp 68–71, 91–99, 125, 141–148, 224–228; Maudsley 15e (2025), pp 234–238, 255, 868–869. IPS schizophrenia CPG (2026), pp 98–99, 113. Kane: Arch Gen Psychiatry 1988;45:789–796. InterSePT: 2003;60:82–91.

Q44 supplement · UK schedule labelled; Indian local protocol prevails · 17 Sep 2026 A4 · 300 dpi

Clozapine - Mechanisms titration and monitoring. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Section companion: Schizophrenia: treatment and antipsychotics.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 51.2

Movement adverse-effect scales

Match the syndrome: parkinsonism → Simpson–Angus; akathisia → Barnes; dyskinesia → AIMS

Extrapyramidal symptoms (EPS): start with examination

Review medicine exposure, timing, dose changes and movement type; record a baseline. A scale grades findings, not causation, and cannot exclude neurological disease. Acute dystonia, airway compromise or fever with rigidity / instability needs urgent assessment.

Simpson–Angus Scale · SAS · parkinsonian signs

Clinician examination, 10 items scored 0–4. Raw sum 0–40; traditional mean = sum / 10, range 0–4. State whether you report sum or mean. The scale is strongly weighted toward rigidity, with other motor and salivation findings.
A mean >0.3 is a conventional screening boundary; Janno et al. (2005) found a higher optimal threshold (about 0.65) in their sample. Neither is a universal diagnostic cut-off.
Useful for drug-induced parkinsonism severity; not a complete measure of all EPS. Consider underlying Parkinson disease and other neurological or musculoskeletal causes.

Barnes Akathisia Rating Scale · BARS / BAS

Clinician observes the seated and standing patient and directly asks about inner restlessness / distress. Separate motor restlessness from anxiety, agitation and restless legs.
Three component ratings: observed movement, subjective awareness and associated distress, each 0–3. A separate global clinical rating runs 0–5. The global score is a clinical judgement, not the arithmetic sum of components.
Global ≥2 supports clinically significant akathisia in the Barnes framework. Record objective and subjective findings; visible pacing alone is insufficient, and severe subjective distress can occur with subtle movement.

Abnormal Involuntary Movement Scale · AIMS · dyskinesia

Standard 12-item form: 7 regional movement ratings, 3 global / awareness ratings, and 2 dental-status questions. Regional items use 0–4; dental questions are yes/no. Complete the structured examination, including activation manoeuvres.
Trials often report the motor sum of items 1–7: 0–28. This excludes global and dental items; it is not a sum of all 12. Clinically, retain regional scores, impairment and the movement description.
Mild movements in ≥2 regions or moderate movements in ≥1 can support a tardive-dyskinesia case definition only with exposure, duration and differential assessment. AIMS alone cannot establish the diagnosis; ordinary tremor is not scored as dyskinesia.

The totals are not interchangeable

SAS sum 8 = mean 0.8, not mean 8. BARS global 3 is not a summed total. AIMS motor 8 can represent very different regional patterns.
Akathisia, parkinsonism and tardive dyskinesia may coexist. Match each instrument to its syndrome, and keep acute dystonia / other urgent movement presentations in the clinical assessment.

Follow-up that helps care

Use the same standard examination and version; document medication exposure, discomfort, falls, eating / speech difficulty and functional impact. Review changes with the prescriber. A reduction in movement score must be weighed against psychiatric response and the person's priorities.

Exam distinction

SAS = parkinsonism; BARS = akathisia; AIMS = dyskinesia. None is a single universal "EPS total". Use the original manual / validated translation and distinguish raw sums from mean or global ratings.

Sources: Q51 tardive-dyskinesia / medication-adverse-effect supplement. Simpson & Angus (1970); Janno et al. (2005), PMC555761. Barnes (1989), PMID2574607; Barnes revisited (2003). Guy ECDEU manual (1976), AIMS form hosted by University of Washington; AIMS motor-sum use in KINECT trials. Baer & Blais (2010), Ch10, supplemented where scoring prose is imprecise.

Q51 supplement · educational scale summaries, not copied item forms · 17 Sep 2026 A4 · 300 dpi

EPS and movement rating scales. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Schizophrenia: treatment and antipsychotics.

Credits and references: retained within the illustration.

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

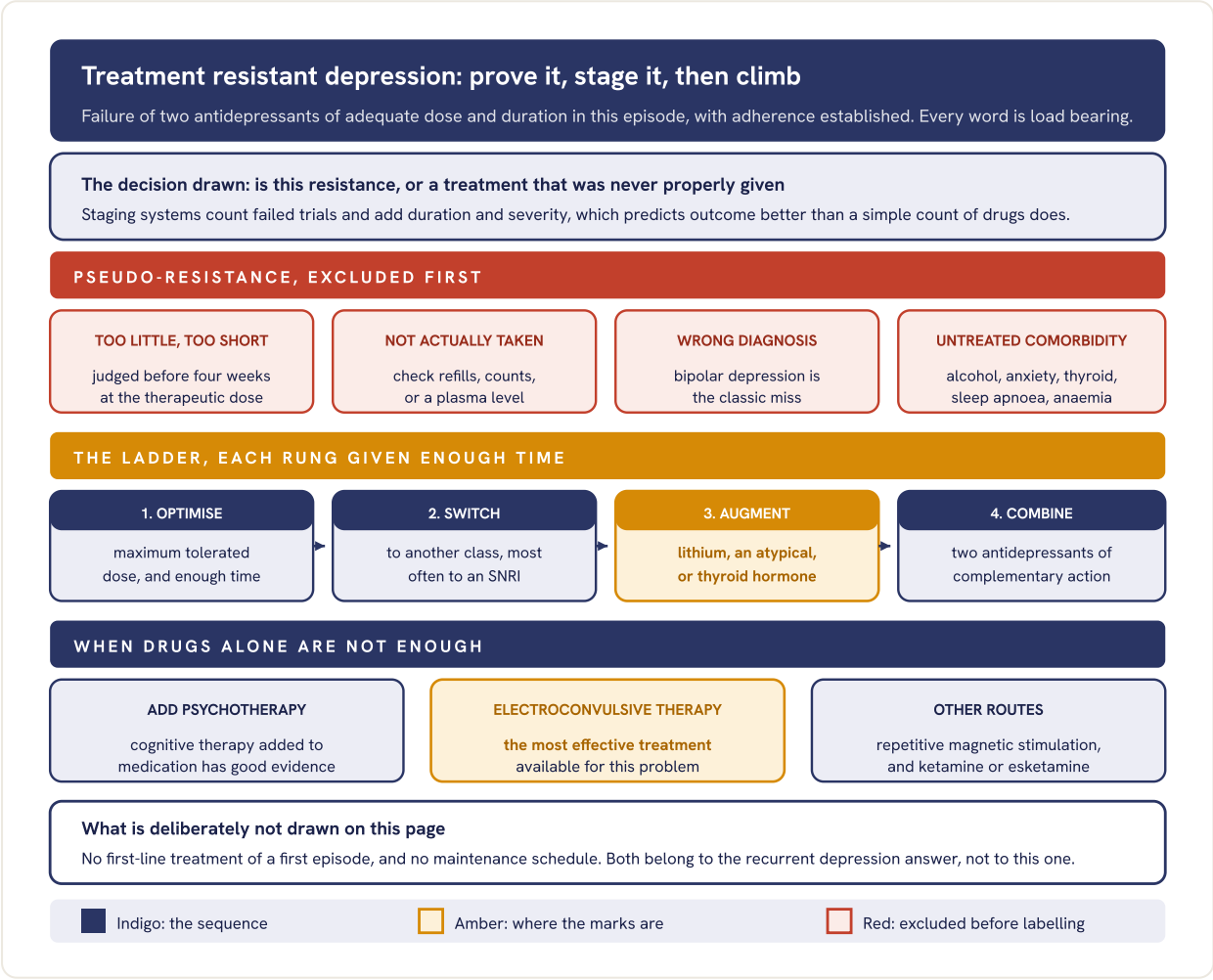


Fig 052.1 The four causes of apparent resistance excluded first, then the drug ladder, then what is done when the ladder runs out.

- **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.

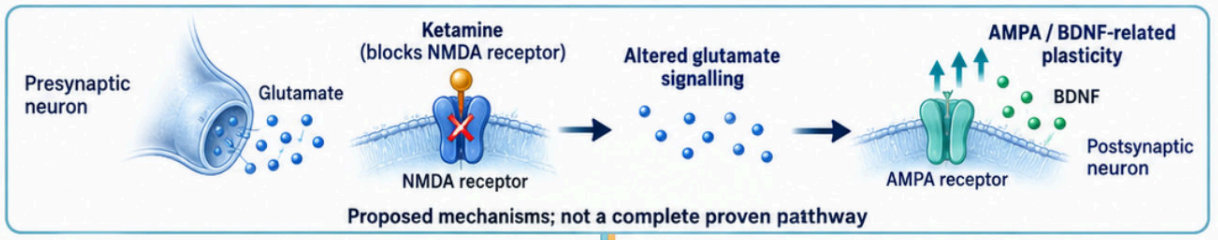
WHAT EARNS THE MARKS

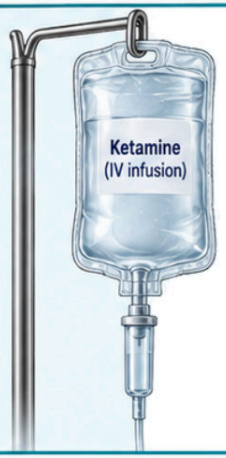
- **The definition unpacked,** with adequate dose, adequate duration and confirmed adherence each spelled out.
- **Pseudo-resistance excluded before the ladder starts.**
- **The ladder in order, with augmentation agents named.**
- **Electroconvulsive therapy identified as the most effective option,**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

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

Ketamine therapy in treatment-resistant depression

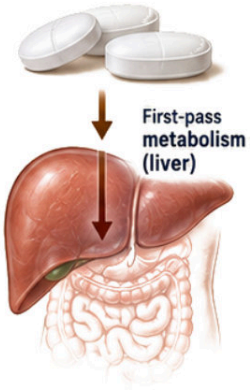
Racemic ketamine: IV and oral routes require different evidence and protocols





INTRAVENOUS (IV)

- Common studied regimen: 0.5 mg/kg over 40 minutes
- Specialist monitored infusion; never a rapid bolus
- Rapid benefit can begin within hours; relapse is common
- Repeat treatment and maintenance: individualise and reassess



ORAL

- First-pass metabolism; lower, variable parent-drug exposure
- No simple IV-to-oral dose conversion
- 2019 trial: 1 mg/kg three times weekly for 21 days (n=41)
- Study regimen, not a standard prescription
- 2024 R-107: benefit in an enriched responder population
- 2026 KET01: primary day-21 endpoint not significant
- Evidence is mixed and formulation-specific

1 BEFORE
Confirm genuine resistance; review bipolarity, psychosis, substance use, medicines and cardiovascular risk

2 DURING / AFTER
Monitor blood pressure, pulse, oxygenation, sedation and dissociation; discharge only when stable with an escort

3 FOLLOW-UP
Track symptoms, function, suicidality, urinary symptoms, cognition and misuse; plan relapse prevention

Racemic ketamine ≠ intranasal esketamine | Rapid symptom relief ≠ proven suicide prevention

Adult specialist education | Sources: CANMAT 2021; Domany 2019; Glue 2024; Walter 2026; FDA safety communication

Ketamine therapy - IV and oral in resistant depression. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-52.

Credits and references: retained within the illustration.

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

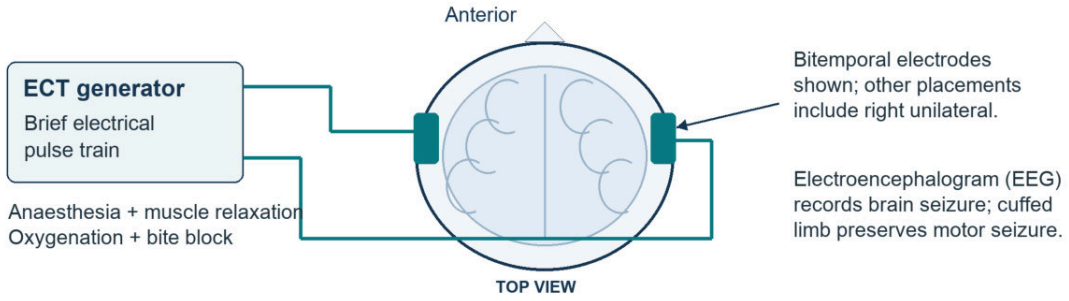
- **Four named mechanisms with the honest closing line,** rather than one confident theory.
- **Consent handled under the statute,** including the prohibition on unmodified treatment.
- **Seizure adequacy described as something measured,** with what is done when it is inadequate.
- **Cognitive monitoring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

WEAVE 100 · PAPER II · 54.1

Electroconvulsive therapy

Modified ECT · controlled generalised seizure under anaesthesia



Electrical stimulus → **Generalised seizure** → **Repeated network changes**

Mechanism: four hypotheses; no single established explanation

Neurotransmitters	Altered serotonin / dopamine signalling and receptor sensitivity.
Anticonvulsant	Threshold often rises across sessions; greater inhibitory tone proposed.
Neuroendocrine	Hypothalamic–pituitary–adrenal stress-axis modulation proposed.
Neuroplasticity	Neurotrophic signalling and structural change; causality remains unclear.

Clinical use · IPS CPG 2023 / 2026

- Severe unipolar or bipolar depression: resistance, psychosis, or rapid response needed.
- Urgent use: high suicide risk, poor intake or severe debilitation; malignant catatonia.
- Also severe / resistant mania; selected schizophrenia, including clozapine augmentation.
- Continuation medication ± ECT after response; acute benefit is not relapse prevention.

Preparation and monitoring

- Consent / capacity; anaesthetic and cardiac assessment; baseline cognition.
- Fasting: clear liquids ≥2 h; light meal / milk ≥6 h; fatty meal >8 h. [IPS 2023]
- Review lithium, benzodiazepines and anticonvulsants individually—not blanket withdrawal.
- Oxygen saturation, electrocardiogram, blood pressure, brain + motor seizure recordings; quality > fixed duration.
- Track symptoms and cognition. Confusion / retrograde amnesia can occur.

Landmark · Sackeim et al., JAMA 2001 · non-IPS primary trial

Randomised, double-blind continuation trial: 84 unipolar-depression remitters after ECT; 24 weeks.

Placebo	Nortriptyline	Nortriptyline + lithium
84% relapse	**60% relapse**	**39% relapse**

Meaning: plan active continuation treatment from the outset.

Limits: small, selected remitter sample; wide uncertainty; tests relapse prevention, not acute ECT efficacy or its mechanism.

Sources: IPS ECT CPG (2023), pp 258–269; IPS depression CPG (2026), pp 88–90. Maudsley 15e (2025), pp 384–385. Weave II Q54, p77. Sackeim et al. JAMA 2001;285:1299–1307. DOI: 10.1001/jama.285.10.1299.

Q54 · educational revision · evidence checked 17 Sep 2026 A4 · 300 dpi

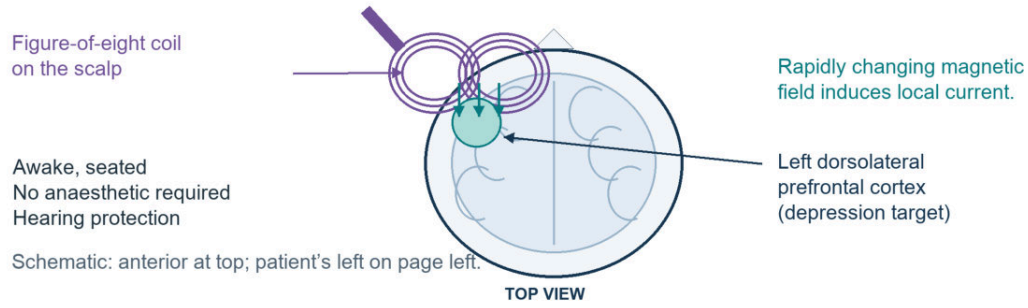
Electroconvulsive therapy - Setup mechanisms and evidence. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with II-54.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 54.2

Repetitive transcranial magnetic stimulation

rTMS · magnetic induction produces a cortical electric field



Pulsed coil current → **Changing magnetic field** → **Induced cortical current**

Mechanism: physical induction is established; therapeutic pathway is not

- Induced electric field can depolarise cortical axons; repeated stimulation changes excitability.
- High-frequency left stimulation / intermittent theta burst usually facilitate; low-frequency right stimulation usually inhibits. Effects depend on protocol and brain state.
- Long-term potentiation / depression-like plasticity and frontolimbic network modulation are proposed—not proof of a single antidepressant mechanism.

Clinical use · IPS CPG 2023 / 2026

- Best-established psychiatric use: major depression, particularly after inadequate treatment response.
- Depression alternatives: high-frequency left, low-frequency right, or intermittent theta burst.
- Other protocol-specific uses: obsessive-compulsive disorder; selected schizophrenia symptoms.
- Evidence varies by target / coil / diagnosis. Persistent hallucinations and negative symptoms are distinct targets.

Dose example and safety

- Conventional depression protocol: left prefrontal cortex, 10 Hz, 120% resting motor threshold; 3,000 pulses/session.
- Usually 5 sessions/week for 4–6 weeks. Calibrate threshold; use protocol-specific train intervals.
- Intermittent theta burst: 600 pulses in ~3 minutes; use its specific protocol.
- Screen seizures / medicines and magnetic-sensitive implants. Headache common; seizures rare; monitor mood switch.

Landmark · THREE-D · Blumberger et al., Lancet 2018 · non-IPS

414 adults; multicentre randomised non-inferiority trial. Intermittent theta burst versus 10-Hz rTMS; stable antidepressants; 4–6 weeks.

~3 min vs 37.5 min: theta burst was non-inferior for symptom change.

Meaning: much shorter sessions can retain benefit under the studied protocol.

Limits: treatment open-label, assessors masked; no sham arm; primary per-protocol analysis. Does not establish identical long-term outcomes.

Sources: IPS rTMS CPG (2023), pp 270–288; IPS depression CPG (2026), pp 88–90, Table 17. THREE-D: Lancet 2018;391:1683–1692. DOI: 10.1016/S0140-6736(18)30295-2; PMID 29726344 (corrected abstract).

Q54 supplement · not an original exam question · checked 17 Sep 2026 A4 · 300 dpi

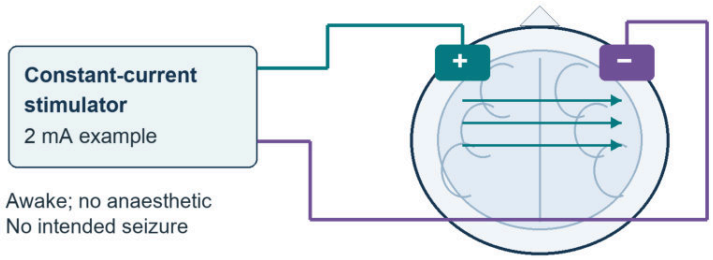
Repetitive transcranial magnetic stimulation - Setup mechanisms and evidence. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with II-54.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 54.3

Transcranial direct current stimulation

tDCS · weak constant current shifts cortical excitability



Depression montage: dorsolateral prefrontal cortex; anterior at top, patient's left at page left.

Weak electric field → **Membrane polarisation** → **Modulated firing probability**

Mechanism: modulation of ongoing activity, not a triggered seizure

- Weak current shifts membrane potential; conventional tDCS does not directly trigger action potentials.
- Anode often facilitates; cathode often inhibits. A heuristic, not a rule: orientation, intensity, duration and brain state alter effects.
- Lasting effects may involve N-methyl-D-aspartate receptor plasticity, gamma-aminobutyric acid and network changes. Clinical mediation remains uncertain.

Clinical use · IPS CPG 2023 / 2026

- Major depression: a selected neuromodulation option, often adjunctive; benefits are protocol-dependent.
- Other studied uses: persistent hallucinations in schizophrenia; craving; obsessive-compulsive disorder.
- These uses do not share equal evidence. IPS 2023 stresses limited short-term evidence and weak long-term evidence.
- Consent: explain uncertain benefit.

Dose example and safety

- Depression example: 2 mA for 20–30 min; pads 25–35 cm²; anode left / cathode right prefrontal cortex. [IPS 2026]
- Course schedule varies; supervised montage, skin preparation and contact checks matter.
- Tingling, itching, erythema and headache; poor contact can cause burns. Monitor mood switch.
- Screen scalp lesions, neurological injury, seizures and implants. Home treatment requires a structured supervision pathway.

Landmark · ELECT-TDCS · Brunoni et al., NEJM 2017 · non-IPS

245 adults with unipolar depression; double-blind, three-arm trial; 10 weeks. tDCS (2 mA, 30 min, 22 sessions) vs escitalopram vs placebo.

tDCS beat placebo, but non-inferiority to escitalopram was NOT shown.

Meaning: modest benefit does not establish antidepressant equivalence.

Limits: single centre, selected low-suicide-risk patients, one protocol; skin effects and two new-onset mania cases in the tDCS arm.

Sources: IPS tDCS CPG (2023), pp 289–296; IPS depression CPG (2026), pp 89–90. ELECT-TDCS: N Engl J Med 2017;376:2523–2533. DOI: 10.1056/NEJMoa1612999; PMID 28657871. Mechanism statements remain qualified.

Q54 supplement · not an original exam question · checked 17 Sep 2026 A4 · 300 dpi

Transcranial direct current stimulation - Setup mechanisms and evidence. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with II-54.

Credits and references: retained within the illustration.

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

WEAVE 100 · PAPER II · 55.1

Lithium

Indications · therapeutic monitoring · adverse-effect mechanisms

Clinical indications · IPS + handbook

Acute mania; bipolar maintenance / relapse prevention. Also used in bipolar depression and resistant unipolar depression augmentation [Handbook].

Therapeutic mechanisms · proposed

Inhibits inositol recycling and glycogen synthase kinase-3β; alters signalling and plasticity.

Clinical benefit has no single proven mechanism.

Adverse effects: connect the organ effect to the mechanism

Kidney · polyuria / thirst	Entry via epithelial sodium channels → less aquaporin-2 → renal vasopressin resistance (nephrogenic diabetes insipidus).
Kidney · chronic injury	Long-term tubulointerstitial injury / fibrosis may reduce filtration. Polyuria alone is not proof of chronic kidney disease.
Thyroid / parathyroid	Reduced thyroid hormone release → hypothyroidism / goitre. Higher calcium-sensing set-point → raised PTH and calcium.
Brain / gastrointestinal	Fine tremor, cognitive dulling, nausea / diarrhoea: often exposure-related; specific mechanisms are incompletely defined.
Weight / skin / heart	Weight gain is multifactorial; acne, psoriasis exacerbation, hair thinning. Sinus-node dysfunction can occur; assess bradycardia.

12-hour serum targets · mmol/L

Acute mania: 0.8–1.0 [IPS 2026 Table 5]; Handbook: 1.0–1.2. Select by response, age and tolerability.

Bipolar maintenance: 0.6–0.8 usual; 0.4–0.6 if effective but poorly tolerated; 0.8–1.0 for breakthrough episodes if tolerated.

Unipolar augmentation: 0.4–0.6 [Handbook]. Older age / renal impairment usually require lower exposure.

Sample 12 h after the last dose. Record dose, formulation and sample time; mmol/L = mEq/L for lithium.

Treat hypothyroidism when indicated; it need not mean stopping lithium.

Dose, timing and organ monitoring

- Start low (often 300 mg at night); titrate to level and response. Renal function, age and formulation alter dose.
- Baseline: weight, renal function / electrolytes, thyroid-stimulating hormone (TSH), calcium, blood count; pregnancy status; ECG if cardiac risk.
- Level ~5–7 days after starting / changing dose; repeat until stable. Then every 3 months in year 1; every 3–6 months thereafter.
- Renal function, TSH and calcium every 6 months; closer checks with declining renal function, illness or interacting drugs.

Important risks · toxicity and pregnancy

Dehydration / sodium loss, thiazides, ACE inhibitors / ARBs and NSAIDs can raise lithium. Keep salt and fluid intake consistent; recheck after illness or interactions.

Coarse tremor, ataxia, dysarthria, vomiting or confusion → WITHHOLD lithium; urgent level, electrolytes / renal tests and medical assessment. Toxicity can occur despite a "therapeutic" level.

Pregnancy: first-trimester exposure raises cardiac-malformation risk; specialist preconception counselling.

Landmarks · primary trials, not IPS guidance

Bowden 1994: 179 manic inpatients, 21-day double-blind trial. ≥50% improvement: lithium 49%, divalproex 48%, placebo 25%. Acute efficacy; not a maintenance trial.

BALANCE 2010: 330 bipolar-I patients, open-label, up to 2 years. New intervention: lithium + valproate 54%, lithium 59%, valproate 69%. Combination beat valproate; superiority over lithium was unproven. Active run-in limits generalisability.

Sources: Meyer & Stahl, The Lithium Handbook (2023), pp 78–80, 107–109, 241–243, 277–281, 299; Maudsley 15e (2025), pp 279–283. IPS bipolar CPG (2026), Tables 5–6; NHS SPS lithium monitoring (2024). Bowden: JAMA 1994;271:918–924. BALANCE: Lancet 2010;375:385–395. Pregnancy: Patorno et al., NEJM 2017;376:2245–2254.

Q55 supplement · clinician revision · checked 17 Sep 2026 A4 · 300 dpi

Lithium - Mechanisms adverse effects and monitoring. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with II-55.

Credits and references: retained within the illustration.

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

WEAVE 100 · PAPER II · 59.1



Mania and bipolar rating scales. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-59.

Credits and references: retained within the illustration.

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.

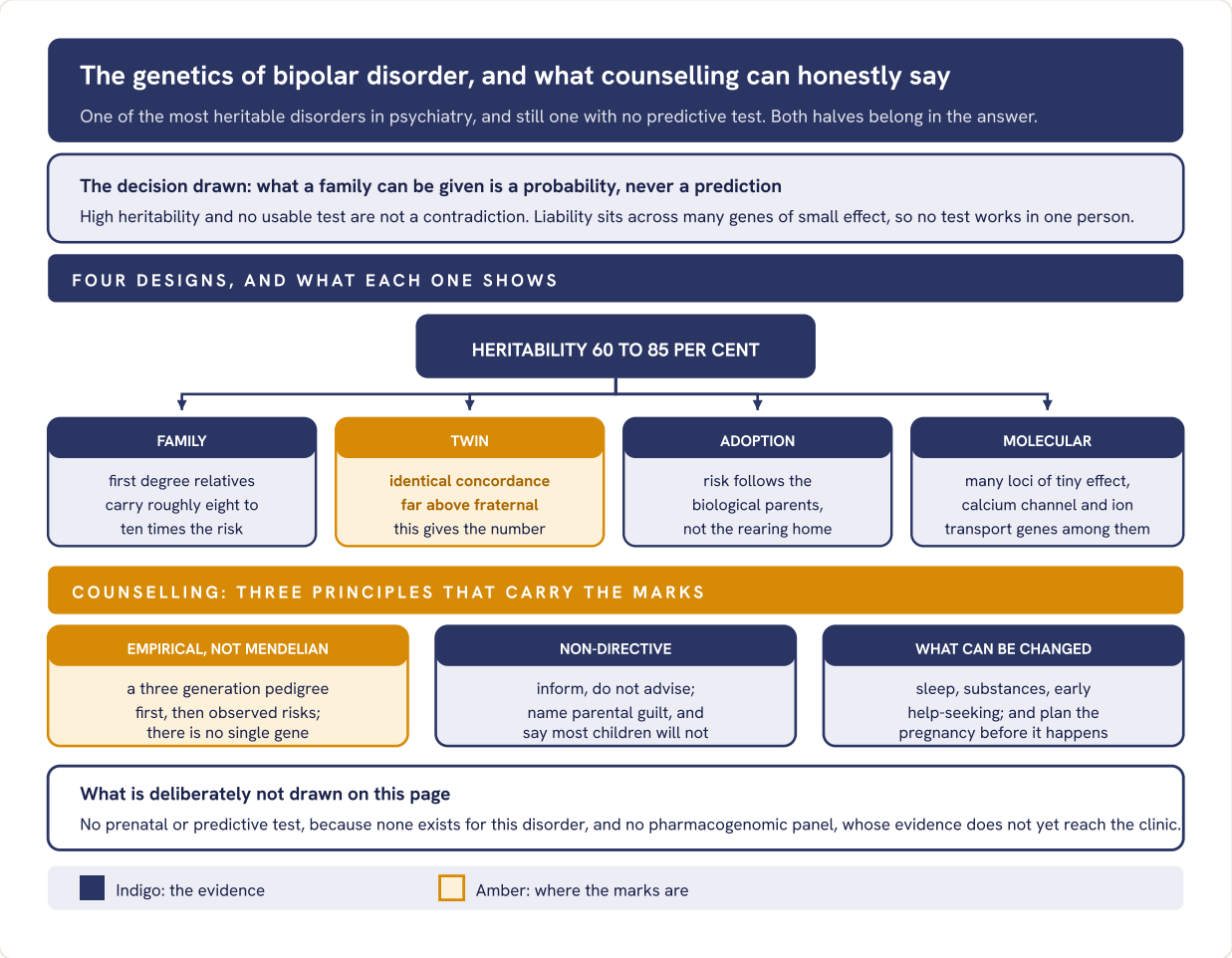


Fig 060.1 The four study designs as one tree, with the counselling principles carried below them because each principle follows from what the design above it can and cannot show.

- **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.

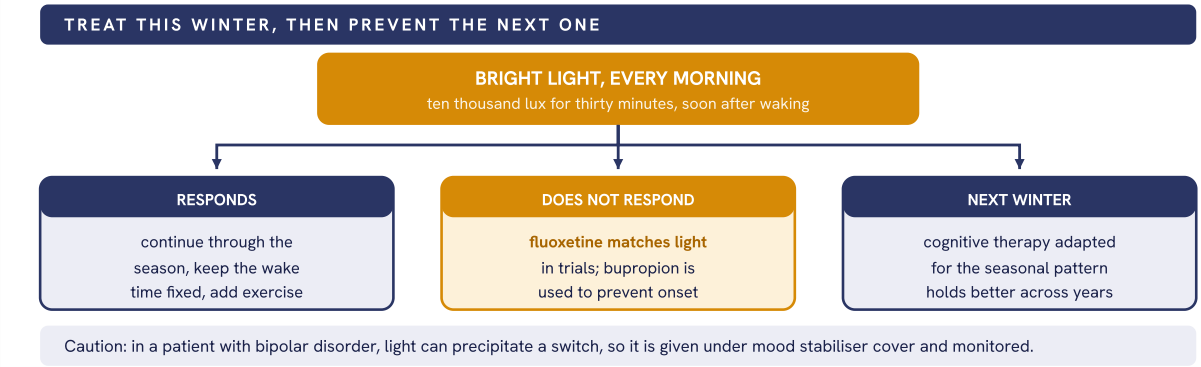


Fig 061.1 Management of the seasonal pattern, with prevention of the following winter treated as part of the same plan.

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

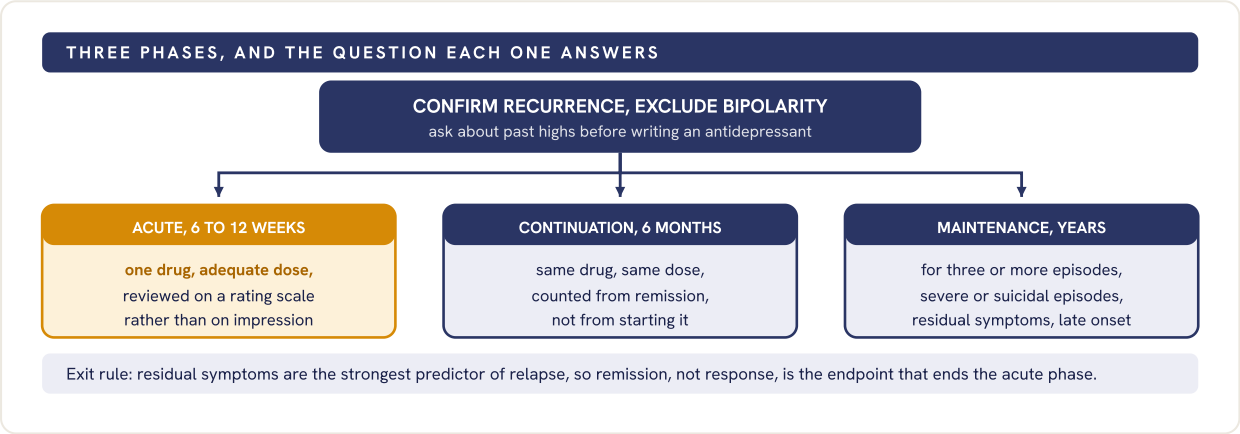


Fig 062.1 The three treatment phases, with the criteria that decide who moves from continuation into indefinite maintenance.

- **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

WEAVE 100 · PAPER II · 62.1

Recurrent depressive disorder

Episode treatment → sustained recovery → relapse prevention · seasonal pattern below

Diagnosis and course · classify before treating

≥2 depressive episodes. ICD-10 CDDG (F33) / ICD-11 (6A71): several months without significant mood disturbance between episodes. DSM-5-TR recurrent MDD: ≥2 consecutive months below full episode criteria.

Screen lifetime mania / hypomania, mixed symptoms, substances and medical causes. A bipolar history changes the diagnosis and treatment pathway.

IPS depression CPG · published 2026 · treatment pathway

1 ACUTE	Mild / low risk: psychotherapy, or medication according to need, preference and access. Moderate: antidepressant and/or psychotherapy. Severe: medication; add psychotherapy when feasible.
2 CONTINUATION	After response, retain the effective antidepressant dose for 16–24 weeks (IPS prose). Continue psychological treatment and restore function; residual symptoms predict relapse.
3 MAINTENANCE	IPS describes 6–12 months after full remission, extending to ≥2 years with recurrent, severe or chronic illness, residual symptoms, comorbidity, continuing stress or poor support.

Nonresponse → recheck diagnosis, adherence, dose and comorbidity → optimise → switch / augment or add psychotherapy. Psychotic depression: antidepressant + antipsychotic. Life-threatening severity: urgent hospital / ECT assessment.

NICE NG222 · 2022 · relapse prevention

Higher relapse risk: continue the effective antidepressant dose, group CBT / mindfulness-based cognitive therapy (MBCT), or both, by shared choice. Review maintenance medication at least every 6 months.

IPS phase intervals overlap; do not add them mechanically into a fixed total course.

SEASONAL PATTERN · a course qualifier, not automatically a separate disorder

Unipolar or bipolar course. DSM-5-TR: seasonal onset and remission / polarity switch for 2 years, no nonseasonal episode of that polarity during that interval; seasonal episodes predominate over the lifetime.

Exclude a recurring seasonal stressor. ICD-11 6A80.4 uses regular seasonality and predominance; do not import the DSM two-year rule into every classification.

Unipolar winter depression

IPS 2026: no specific antidepressant has demonstrated superiority for seasonal patterns.

CANMAT 2023 update (2024): light therapy is first-line monotherapy for winter seasonal MDD: 10,000 lux white light, 30 min each morning, shortly after waking.

NICE 2022 is more cautious: explain uncertainty about light efficacy. Discuss antidepressants / psychotherapy and plan ahead of the usual relapse season.

Bipolar seasonal depression

IPS 2026: bright light is a third-line adjunct; antidepressant monotherapy is not recommended.

ISBD 2025: clinician-supervised light, often starting 15 min/day with gradual titration; morning or midday. Bipolar I: antimanic treatment for ≥2–4 weeks before and during light.

Avoid light in current / recent mania or hypomania, mixed symptoms or rapid cycling. New activation → stop / reduce light and obtain prompt review.

Light: clock mechanism and precautions

Retinal light input → suprachiasmatic clock → circadian phase adjustment. Use a UV-filtered therapeutic device at its specified distance; check retinal disease / photosensitising drugs and monitor sleep, mood and activation.

Sources: Weave Q62 p87 + Q61 p86. ICD-10 CDDG; ICD-11 CDDR (2024); DSM-5-TR (2022). IPS depression (2026), pp74,77–78; IPS bipolar update (2026). NICE NG222 (2022), §§1.8,1.4.39; CANMAT 2023 update (2024), light therapy; ISBD light recommendations (2025). Exact evidence / URLs in companion ledger.

Q62 + Q61 · clinical revision · checked 17 Sep 2026 A4 · 300 dpi

Recurrent depression and seasonal pattern. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-62.

Credits and references: retained within the illustration.

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

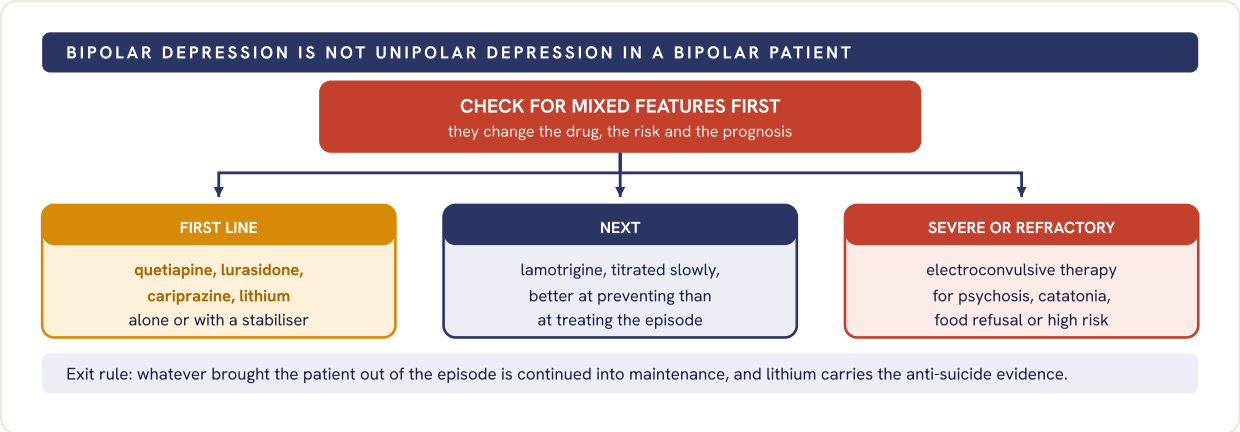


Fig 063.1 The treatment sequence for a depressive episode in bipolar disorder, entered through the mixed-features check because that answer changes everything below it.

- **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

Rapid-cycling bipolar disorder

Definition • Risk correlates • Four-step Maudsley strategy | 15th edition (2025), pp316–317, Table 2.8

DEFINITION

≥4 mood episodes in 12 months: mania, hypomania or depression.
An episode pattern; not ordinary within-day mood shifts.

RISK CORRELATES [2]

- Female sex
- Bipolar II
- Mixed features
- Childhood maltreatment

Associations, not proof of causation or inevitable rapid cycling.

POTENTIALLY MODIFIABLE [1,2]

- Antidepressant exposure
- Alcohol / other substances
- Thyroid dysfunction
- External stressors

Review these before adding more drugs.

HOW TO READ THE SEQUENCE [1]

- Limited data; few drug comparisons.
- No universal first-choice agent or combination.
- Prior response + current polarity + patient factors determine choice.
- Rapid cycling can remit or recur.
- Higher depressive burden / suicide risk: assess safety throughout.

CLINICAL GUARDRAILS

- Taper/withdraw antidepressants under supervision.
- Combinations need interaction, level and adverse-effect review.
- Lamotrigine: slow, interaction-adjusted titration; not acute antimanic treatment.
- High-dose thyroxine: specialist use + thyroid/cardiac monitoring.
- Urgent severe illness may require earlier ECT.

1 Withdraw antidepressants

Maudsley recommends withdrawal in all rapid cyclers; it notes controversial evidence supporting SSRI continuation in selected cases.

↓

2 Evaluate possible precipitants

Alcohol • thyroid dysfunction (including antithyroid antibodies) • external stressors.

↓

3 Optimize mood stabilizer treatment

Use plasma levels. Consider combinations: lithium + valproate; lithium + lamotrigine; valproate + carbamazepine. Alternatively, proceed to Step 4.

↓

4 Consider other, usually adjunctive, options

Source list in alphabetical order; NOT an efficacy ranking. Doses below are the table's reported regimens, not universal targets.

Option	Source regimen
Aripiprazole	15–30 mg/day
Clozapine	Usual doses
ECT	—
Lamotrigine	Up to 225 mg/day
Levetiracetam	Up to 2000 mg/day
Lurasidone	40–120 mg/day
Nimodipine	180 mg/day

Option	Source regimen
Olanzapine	Usual doses
Quetiapine	300–600 mg/day
Risperidone	Up to 6 mg/day
Thyroxine	150–400 micrograms/day
Topiramate	Up to 300 mg/day
rTMS	—

Evidence notes from Maudsley [1]

- Quetiapine: probably best supporting data; similar efficacy to aripiprazole/olanzapine.
- Levetiracetam, nimodipine, thyroxine, topiramate: relatively limited evidence.
- Clozapine: role in treatment-resistant bipolar disorder, which may include rapid cycling.

Akiskal & Pinto 1999 [3]	Historical prototypes • not DSM/ICD diagnoses
I: full mania	I½: depression + protracted hypomania
II: depression + spontaneous hypomania	II½: major depression + cyclothymic background
III: antidepressant-associated hypomania	III½: bipolarity masked/unmasked by stimulant misuse
IV: depression + hyperthymic temperament	1999 version; later extensions excluded

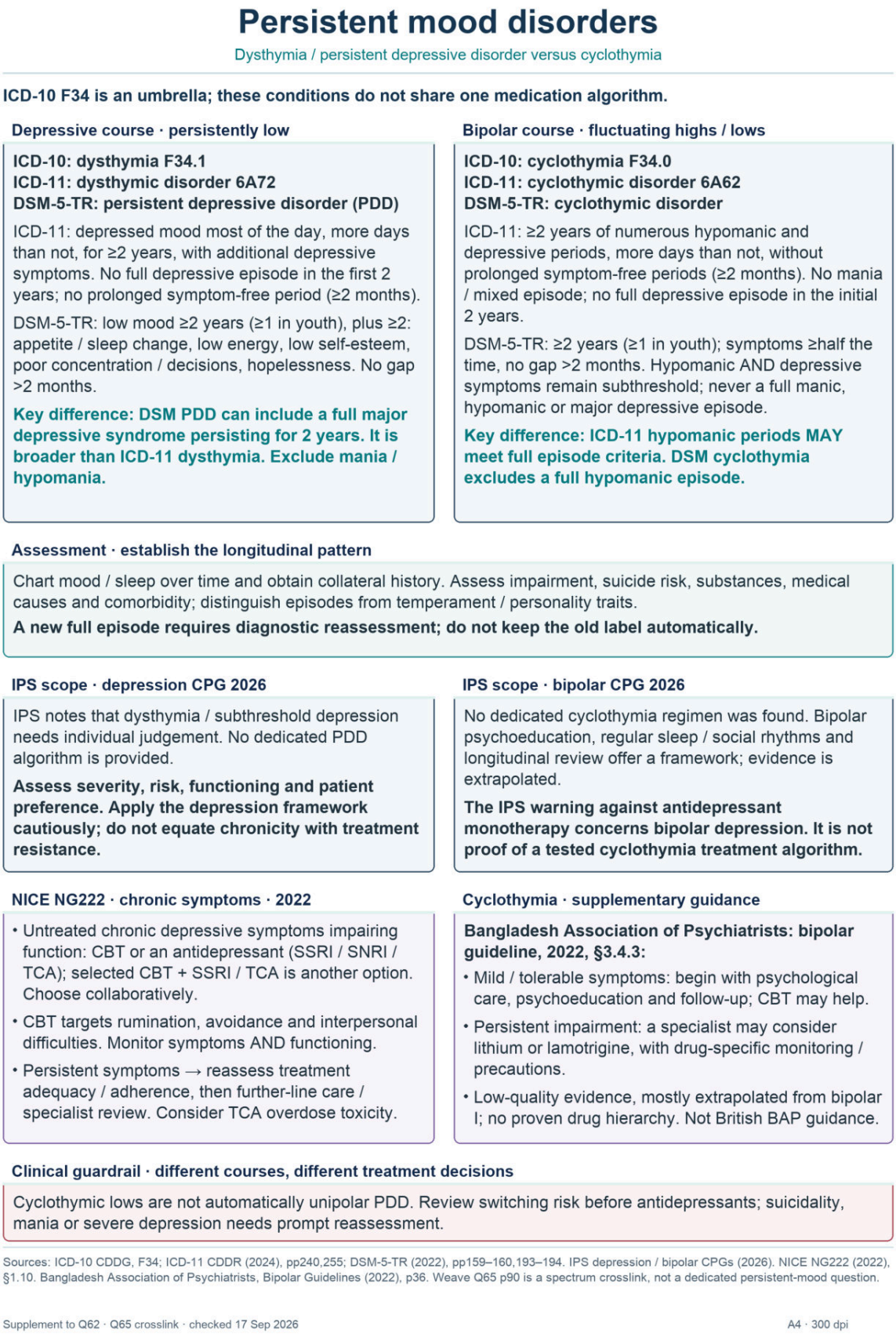
[1] Taylor, Barnes & Young: Maudsley Prescribing Guidelines, 15th ed., 2025, pp316–317 / Table 2.8 [2] Miola et al., 2023 [3] Akiskal & Pinto 1999

Four-step sequence paraphrased from the verified local edition • Guardrails are clinical context • Akiskal retained as historical context; detailed formal criteria omitted

Rapid cycling bipolar disorder - Management types and Akiskal spectrum. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-65.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 62.2



Persistent mood disorders. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Mood disorders: syndromes and nosology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 63.1

Valproate

Head-to-toe adverse effects · mechanisms · a compact prescribing framework

Clinical indications · IPS + Maudsley

Acute mania / mixed presentations; maintenance. Weaker evidence in bipolar depression.

Also epilepsy; migraine prevention (product dependent).

Therapeutic mechanisms · multiple

Enhances GABA inhibition; affects sodium channels, intracellular signalling and gene expression.

No single proven mood-stabilising pathway.

ADVERSE-EFFECT MAP

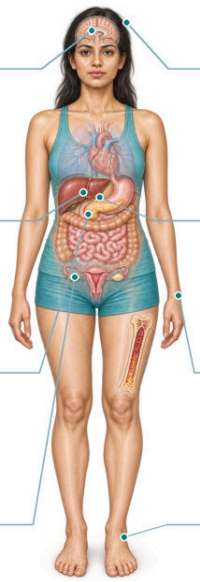
Proposed mechanisms are labelled; connector lines locate organs.

Brain · sedation / tremor

CNS effects are often dose-related; exact adverse-effect pathways are uncertain. Reversible parkinsonism can occur.

Hair · reversible alopecia

Usually reversible and exposure-related; the mechanism is uncertain. Do not assume a biotin or trace-element deficiency.



Ammonia · encephalopathy

Mitochondrial / carnitine effects and impaired urea-cycle function can raise ammonia, even with normal liver tests.

Liver · rare severe injury

Mitochondrial dysfunction → impaired fatty-acid oxidation / microvesicular injury. Risk is high with POLG disease.

Gut / pancreas

Nausea is common. Rare pancreatitis has an uncertain mechanism; severe abdominal pain / vomiting needs urgent review.

Blood · platelets / bleeding

Dose-related count reduction and platelet dysfunction; immune / production pathways remain unsettled. Bruising merits a count.

Weight / ovaries

Appetite / metabolic changes → weight gain. Insulin resistance and altered ovarian steroid pathways may contribute to PCOS.

Peripheral / long-term

Oedema: mechanism uncertain. Bone-health risk with prolonged antiseizure therapy is multifactorial; assess individual risks.

Dose and level · IPS / Maudsley

- Divalproex: start 250 mg twice / three times daily; titrate to 20–30 mg/kg/day as tolerated. Review formulation switches.
- Acute-mania total level: 50–125 µg/mL (= mg/L); no universal maintenance target. Clinical response remains central.
- Predose trough ~5 days after a change; 24 h only for once-daily extended release. Low albumin raises the free fraction: consider a free level.

Monitoring, contraindications, interactions

- Baseline: weight, blood count / platelets, liver tests, pregnancy status. Liver tests early; blood count / liver tests every 6–12 months once stable.
- New lethargy, vomiting or confusion: check ammonia; not a routine screening test. Avoid in significant liver disease, urea-cycle or known POLG disorders.
- Valproate raises lamotrigine exposure / rash risk; carbapenems sharply lower valproate; topiramate increases hyperammonaemia risk.

Reproductive harm · major prescribing constraint

Fetal malformations / developmental harm: histone-deacetylase inhibition is one proposed pathway. WHO 2023: avoid in women / girls of childbearing potential; specialist alternatives + contraception.

Male fertility may fall. UK precaution: contraception during treatment + 3 months after; paternal risk causality uncertain. No abrupt cessation without a treatment plan.

Landmark · Bowden et al., JAMA 1994 · non-IPS primary trial

179 manic inpatients; 21-day double-blind trial. ≥50% improvement: valproate 48%, lithium 49%, placebo 25%. Supports acute efficacy; not long-term prevention.

Sources: Maudsley 15e (2025), pp 289–291, 868–869; IPS bipolar CPG (2026), Tables 5 / 7; NIH LiverTox: Valproate; current US Depakote label. Reproductive advice: WHO 2023; UK MHRA 2024–2025 (not Indian regulation). Mechanisms: PMID 8058141, 28879549, 14576182, 11473107. Bowden: JAMA 1994;271:918–924.

Q63 broader pharmacotherapy supplement · not an original exam question · 17 Sep 2026 A4 · 300 dpi

Valproate - Head-to-toe adverse effects and mechanisms. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Section companion: Mood disorders: syndromes and nosology.

Credits and references: retained within the illustration.

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.

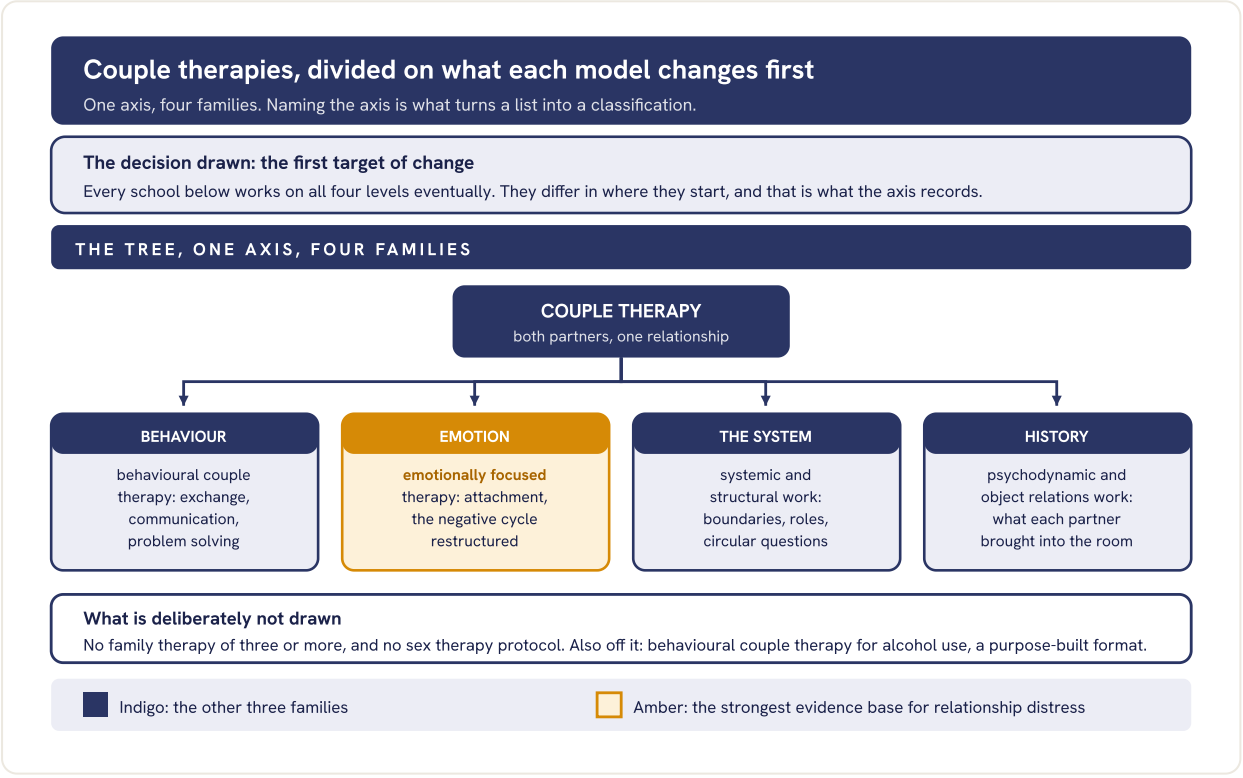


Fig 068.1 One axis, the first target of change, dividing the field into four families, with the emotionally focused branch marked because it carries the largest trial base.

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

WEAVE 100 · PAPER III · A01

WEAVE

100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION

MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

CT / COGNITIVE-BEHAVIOURAL

Cognitive Therapy

Treat interpretations as hypotheses. Change grows through guided discovery and lived experiments, not compulsory positive thinking.

MODEL PRACTICE EVIDENCE



Aaron T. Beck

Founder of cognitive therapy

THE CLINICAL IDEA

Ask: What maintains the problem, and what observation would test that explanation?

AI-ILLUSTRATED PORTRAIT

A01

Aaron T. Beck • Collaborative empiricism • Present-focused formulation

SECTION 01 THE MODEL

1. Situation
2. Meaning
3. Emotion + body
4. Action

Automatic thoughts, assumptions and core beliefs shape responses. Behaviour and attention can then maintain the original belief; intervention can begin at any point.

SECTION 02 THE TECHNIQUES

- **Formulate together.** Map a recent episode: trigger, thought, emotion, bodily response and action. Identify a maintaining cycle.
- **Guided discovery.** Use Socratic questions, evidence review and alternative explanations; do not debate the person into agreement.
- **Test predictions.** Design a behavioural experiment with a specific prediction, observable outcome and learning review.
- **Consolidate learning.** Use thought records, graded tasks and between-session practice; develop a personalised relapse plan.

SECTION 03 IN THE ROOM

Fictional example: After one correction, a trainee predicts universal rejection. They ask for specific feedback and compare the actual response with the prediction.

SECTION 04 WHAT THE EVIDENCE SAYS

Disorder-specific cognitive and CBT protocols have substantial evidence, including for depression and anxiety. Evidence for a whole protocol does not establish that cognitive restructuring alone produces all benefit. [1,2]

CLINICAL CAUTION

Validate adversity before examining appraisals. Poverty, abuse and discrimination are not cognitive distortions. Adapt homework to attention, literacy and available time.

SELECTED REFERENCES
1. Beck AT et al. Cognitive Therapy of Depression. Guilford; 1979.
2. NICE. Depression in adults: treatment and management. NG222; 2022. nice.org.uk/guidance/ng222

WEAVE 100+ / CLINICIAN APPENDIX

Narrative evidence appraisal • Fictional examples • White-background magazine edition

Cognitive therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A02

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

CLASSICAL FRAMEWORK / CLINICAL ADJUNCT

Yoga

Read Patanjali on his own terms; evaluate modern yoga programmes on their own clinical evidence.



Patanjali
Traditional depiction • Yoga Sutras traditionally attributed to him

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

Classical purpose, modern protocol and clinical outcome are three different questions.

TRADITIONAL DEPICTION

Patanjali • Yoga Sutra • Modern programmes vary in content and intensity

A02

SECTION 01 THE MODEL

Ethical discipline → Body + breath
Concentration ← Sense withdrawal

Eight limbs (II.29): yama (restraints), niyama (observances), asana (posture), pranayama (breath regulation), pratyahara (sense withdrawal), dharana (concentration), dhyana (meditation), samadhi (absorption). This is a classical framework, not a medical outcome scale. [1]

SECTION 02 THE TECHNIQUES

- **Philosophical frame.** I.2 concerns the stilling of mental fluctuations; practice and non-attachment appear in I.12. Avoid equating these directly with diagnostic remission.
- **Specify the intervention.** Record posture, breathing, meditation, session dose, instructor competence and home practice. Yoga is not a uniform treatment.
- **Adapt safely.** Use accessible postures and comfortable breathing; omit painful movements, forceful breathwork or prolonged retention when unsuitable.
- **Measure benefit.** Track the actual target: anxiety, mood, function, pain or sleep. Record adherence and adverse experiences.

SECTION 03 IN THE ROOM

Fictional example: A person with anxiety chooses gentle yoga alongside care. Progress is judged by functioning and symptoms, not by achieving a spiritual state.

SECTION 04 WHAT THE EVIDENCE SAYS

In GAD, Kundalini yoga beat stress education; noninferiority to CBT was not established. For chronic nonspecific low-back pain, benefits over no exercise were small and clinically unimportant; function was similar to other back-focused exercise. Adverse events (mainly back pain) increased versus no exercise. [2,3]

CLINICAL CAUTION

Offer as a preference-sensitive adjunct. Modify for physical illness, pregnancy and trauma sensitivity. Spiritual participation must be voluntary; yoga does not replace indicated treatment.

SELECTED REFERENCES
1. Patanjali. Yoga Sutra I.2, I.12, II.29; translations differ.
2. Simon NM et al. JAMA Psychiatry. 2021;78:13-20. doi:10.1001/jamapsychiatry.2020.2496
3. Wieland LS et al. Yoga for chronic non-specific low back pain. Cochrane; 2022. doi:10.1002/14651858.CD010671.pub3

WEAVE 100+ / CLINICIAN APPENDIX
Narrative evidence appraisal • Fictional examples • White-background magazine edition

Yoga. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A03



Couple therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A04

WEAVE
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THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

DELIVERY FORMAT / CRITICAL APPRAISAL

Mobile-Based Psychotherapy

A phone can deliver therapy. The device, interface or chatbot is not itself a validated therapeutic model.



Gerhard Andersson
Pioneer of internet-delivered CBT

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

Ask: Which intervention, for whom, against what comparator, with what human support?

AI-ILLUSTRATED PORTRAIT

A04

Guided digital CBT • Teletherapy • Apps • Ecological momentary support

SECTION 01 THE MODEL

1. Assess + consent
2. Choose programme
3. Practise + support
4. Review + escalate

Separate clinician-delivered teletherapy, structured digital programmes, self-help apps and generative chatbots. Evidence cannot be transferred automatically between these categories.

SECTION 02 THE TECHNIQUES

- **Match need and delivery.** Check diagnosis or target, severity, language, disability access, connectivity and preference; establish a human escalation route.
- **Make practice useful.** Use brief modules, behavioural tasks, reminders and diaries tied to a formulation; minimise notification burden.
- **Appraise the product.** Check the exact programme version, trial population, comparator, attrition, follow-up and whether outcomes were independently assessed.
- **Protect the alliance.** Agree response times and monitoring limits. Explain data collection, sharing, retention and what happens during a crisis.

SECTION 03 IN THE ROOM

Fictional example: A busy resident completes guided CBT tasks by phone. Persistent deterioration triggers a clinician review rather than more automated reminders.

SECTION 04 WHAT THE EVIDENCE SAYS

A 2024 meta-analysis of 176 RCTs of smartphone apps found small average benefits for depressive and generalised anxiety symptoms, with important heterogeneity. These findings do not validate every commercial app, teletherapy service or LLM chatbot. [1]

CLINICAL CAUTION

Watch for disengagement, digital exclusion, surveillance, commercial claims and untested updates. App access is not equivalent to crisis monitoring or confidential clinical care.

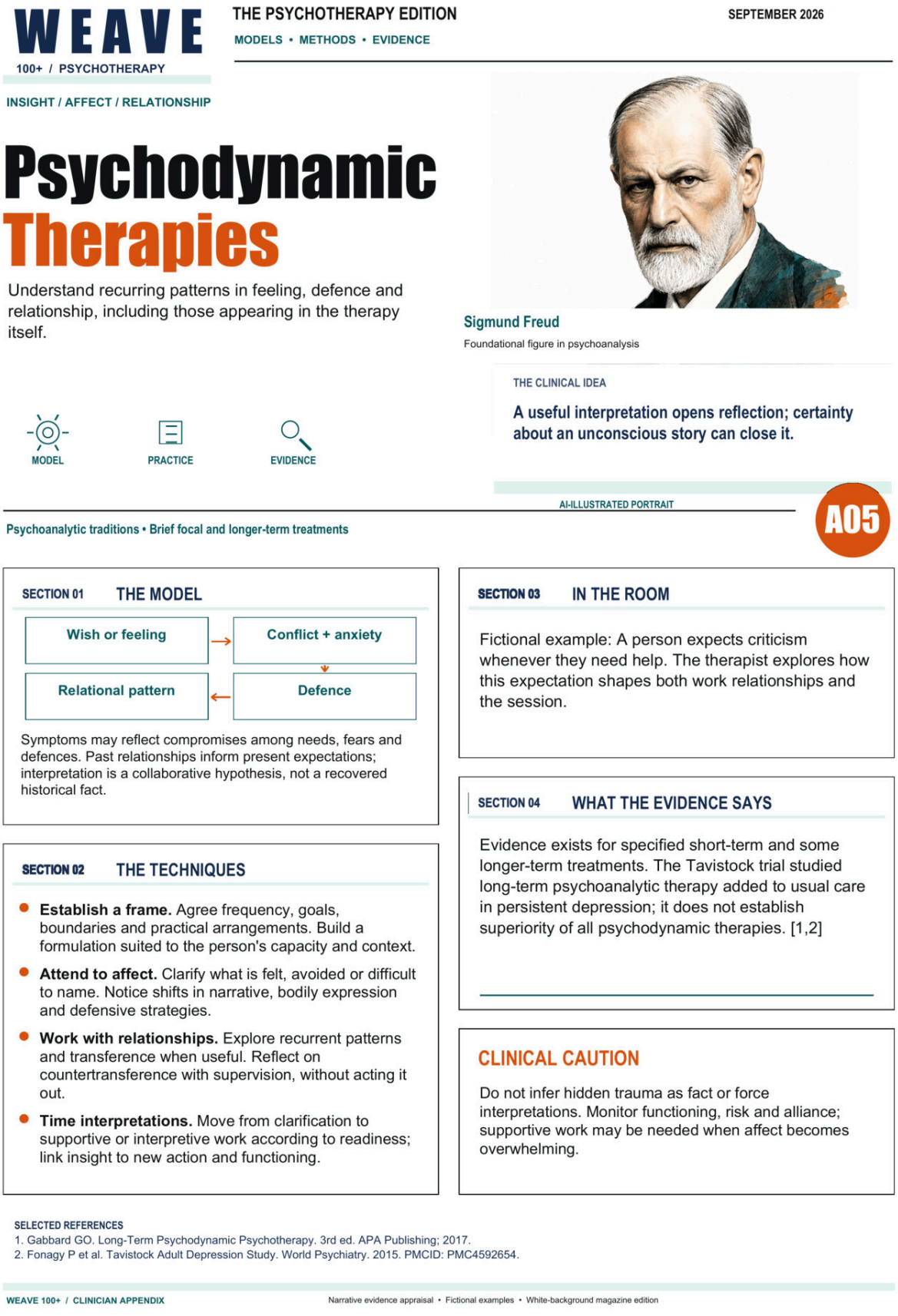
SELECTED REFERENCES
1. Linardon J et al. World Psychiatry. 2024;23:139-149. doi:10.1002/wps.21183
2. NICE. Depression in adults. NG222; guided self-help recommendations, 2022.

WEAVE 100+ / CLINICIAN APPENDIX
Narrative evidence appraisal • Fictional examples • White-background magazine edition

Mobile based psychotherapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A05



Psychodynamic therapies. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A06

WEAVE

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MODELS • METHODS • EVIDENCE

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EXTENDED FORMULATION-BASED WORK

Long-Term CBT

Longer treatment should deepen a testable formulation and build independence, not repeat the same sessions indefinitely.

MODEL PRACTICE EVIDENCE



Judith S. Beck

Major contributor to contemporary CBT

THE CLINICAL IDEA

More sessions are justified by a learning plan, measurable benefit and an exit strategy.

AI-ILLUSTRATED PORTRAIT

A06

No single standard protocol • Persistent problems • Regular review points

SECTION 01 THE MODEL

Shared formulation → Repeated experiments → Generalise skills → Taper + prevent relapse

Duration is a treatment parameter, not a separate school. Extend work when formulation, complexity, goals and response justify it; distinguish maintenance CBT from schema therapy or CBASP.

SECTION 02 THE TECHNIQUES

- **Reformulate stuck points.** Assess avoidance, rumination, safety behaviours, comorbidity, alliance and environmental obstacles before simply adding sessions.
- **Work on beliefs.** Use downward-arrow enquiry, continua, positive-data logs and experiments testing conditional rules and core beliefs.
- **Expand behavioural learning.** Use graded exposure, activity scheduling, problem-solving and interpersonal experiments; imagery rescripting requires suitable training and formulation.
- **Review and taper.** Track agreed functional goals and symptoms; schedule formulation reviews, spaced sessions, self-therapy practice and a relapse response plan.

SECTION 03 IN THE ROOM

Fictional example: Repeated reassurance briefly reduces doubt but maintains dependence. Treatment shifts toward uncertainty experiments and progressively self-directed practice.

SECTION 04 WHAT THE EVIDENCE SAYS

NICE supports CBT targeting maintaining processes in chronic depressive symptoms. That recommendation does not imply an unlimited dose or universal advantage of long-term over brief CBT. Match evidence to the specific disorder and protocol. [1,2]

CLINICAL CAUTION

A stalled course calls for review, not automatic extension. Consider diagnosis, formulation, accessibility, competing needs and another treatment when progress is limited.

SELECTED REFERENCES
1. NICE. Depression in adults. NG222, sections 1.8 and 1.10; 2022.
2. Beck JS. Cognitive Behavior Therapy: Basics and Beyond. 3rd ed. Guilford; 2021.

WEAVE 100+ / CLINICIAN APPENDIX

Narrative evidence appraisal • Fictional examples • White-background magazine edition

Long term CBT. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A07

WEAVE

100+ / PSYCHOTHERAPY

NEEDS / SCHEMAS / MODES

THE PSYCHOTHERAPY EDITION

MODELS • METHODS • EVIDENCE

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Schema Therapy

Connect enduring patterns to unmet needs, then build healthier ways of feeling, relating and acting.



Jeffrey E. Young

Founder of schema therapy

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

Schema = enduring theme. Mode = current state. Coping = what the person does with it.

AI-ILLUSTRATED PORTRAIT

A07

Jeffrey Young • Integrates cognitive, experiential and relational methods

SECTION 01 THE MODEL

1. **Unmet needs**
2. **Schema activation**
3. **Coping mode**
4. **Healthy Adult response**

Schemas are enduring themes; modes are moment-to-moment states. Surrender, avoidance and overcompensation can preserve schemas even when they temporarily reduce distress.

SECTION 02 THE TECHNIQUES

- **Build a mode map.** Identify triggers, vulnerable feelings, punitive or demanding messages and coping modes using concrete recent episodes.
- **Offer bounded repair.** Use empathic confrontation and limited reparenting within professional boundaries; meet needs without creating limitless availability.
- **Use experiential methods.** Imagery rescripting and chair dialogues access emotion, challenge internal criticism and strengthen compassionate protection.
- **Break behaviour patterns.** Rehearse new responses, set graded tasks, use flashcards or diaries, and strengthen Healthy Adult capacities outside sessions.

SECTION 03 IN THE ROOM

Fictional example: Feedback activates a Defectiveness schema and a Detached Protector response. The person practises receiving feedback without withdrawing from contact.

SECTION 04 WHAT THE EVIDENCE SAYS

RCTs support schema therapy for borderline personality disorder. Comparative findings depend on programme, setting and outcome; avoid calling it universally superior to DBT, MBT or TFP. The cited review concerns BPD, not all personality disorders. [1,2]

CLINICAL CAUTION

Experiential work can intensify affect. Agree pacing, grounding and consent. Limited reparenting is a technical stance with limits, not a parental relationship.

SELECTED REFERENCES
1. Young JE, Klosko JS, Weishaar ME. Schema Therapy: A Practitioner's Guide. Guilford; 2003.
2. Oud M et al. Specialized psychotherapies for BPD. Aust N Z J Psychiatry. 2018. doi:10.1177/0004867418791257

WEAVE 100+ / CLINICIAN APPENDIX

Narrative evidence appraisal • Fictional examples • White-background magazine edition

Schema therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A08

WEAVE

100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION

MODELS • METHODS • EVIDENCE

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TFP / OBJECT RELATIONS

Transference-Focused Psychotherapy

Use the here-and-now therapeutic relationship to examine and integrate polarized experiences of self and others.



Otto F. Kernberg

Principal originator of transference-focused psychotherapy

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

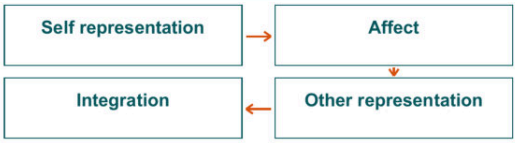
The target is integration of representations, not simply pointing out transference.

AI-ILLUSTRATED PORTRAIT

A08

Otto Kernberg • Frank Yeomans • John Clarkin • Structured psychodynamic treatment

SECTION 01 THE MODEL



An activated self-other dyad can flip rapidly: idealized helper becomes persecutor, or helpless self becomes attacking self. These shifts are explored as experiences, not taken as literal truths.

SECTION 02 THE TECHNIQUES

- **Contract first.** Agree treatment conditions, responsibilities, boundaries and how threats to safety or treatment will be managed.
- **Clarify the moment.** Explore what the person perceives, feels and expects in a specific exchange with the therapist.
- **Confront discrepancies.** Bring contradictory accounts or shifts into shared attention; confrontation here means careful comparison, not aggression.
- **Interpret and integrate.** Link split self-other representations and associated affects within the transference, while maintaining reflective attention and supervision.

SECTION 03 IN THE ROOM

Fictional example: A session ending is experienced as deliberate rejection. Therapist and patient examine that perception and its shift from the earlier idealized view.

SECTION 04 WHAT THE EVIDENCE SAYS

TFP has randomized-trial evidence in borderline personality disorder. It requires specialised training and a structured programme; comparative evidence does not establish one best therapy for every person or outcome. [1,2]

CLINICAL CAUTION

Technical neutrality does not mean emotional coldness or ignoring danger. Risk management and the treatment contract remain active throughout treatment.

SELECTED REFERENCES
1. Yeomans FE, Clarkin JF, Kernberg OF. Transference-Focused Psychotherapy for Borderline Personality Disorder. APA Publishing; 2015.
2. Oud M et al. Aust N Z J Psychiatry. 2018. doi:10.1177/0004867418791257

WEAVE 100+ / CLINICIAN APPENDIX

Narrative evidence appraisal • Fictional examples • White-background magazine edition

Transference focused psychotherapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A09

WEAVE
100+ / PSYCHOTHERAPY
DBT / ACCEPTANCE + CHANGE

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE
SEPTEMBER 2026

Dialectical Behaviour Therapy

Validate the person's experience while helping them replace behaviours that make life more dangerous or unworkable.



Marsha M. Linehan
Developer of dialectical behaviour therapy

THE CLINICAL IDEA

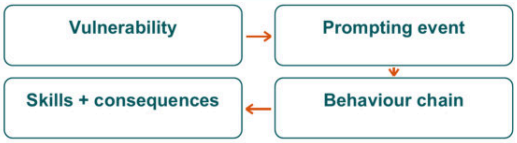
Validation explains why a response makes sense; change work asks whether it works.

AI-ILLUSTRATED PORTRAIT

A09

Marsha Linehan • Biosocial model • Behavioural analysis

SECTION 01 THE MODEL



Emotional vulnerability and invalidating transactions can reinforce dysregulation. The model is transactional; it does not reduce a person's difficulties to biology or family blame.

SECTION 02 THE TECHNIQUES

- **Follow the hierarchy.** Prioritise life-threatening behaviours, therapy-interfering behaviours, quality-of-life problems, then skills acquisition.
- **Analyse a chain.** Identify vulnerabilities, prompting event, linked thoughts/feelings/actions and consequences; add a solution analysis.
- **Teach four skill sets.** Mindfulness, distress tolerance, emotion regulation and interpersonal effectiveness; practise when calm and generalise to real situations.
- **Deliver the programme.** Standard comprehensive DBT includes individual therapy, skills training, between-session coaching and a therapist consultation team.

SECTION 03 IN THE ROOM

Fictional example: After an argument, a diary card identifies a crisis urge. Chain analysis locates an early point for paced responding and asking for help.

SECTION 04 WHAT THE EVIDENCE SAYS

DBT has evidence for reducing self-harm and related outcomes in BPD; certainty varies by outcome and comparator. NICE specifically recommends considering comprehensive DBT for women with BPD when recurrent self-harm is a priority. [1,2]

CLINICAL CAUTION

A skills group alone is not comprehensive DBT. Crisis skills supplement an individual safety plan; they do not replace risk assessment or urgent care.

SELECTED REFERENCES
1. Linehan MM. DBT Skills Training Manual. 2nd ed. Guilford; 2015.
2. NICE CG78, section 1.3.4; 2009. See also Oud et al., 2018, doi:10.1177/0004867418791257

WEAVE 100+ / CLINICIAN APPENDIX
Narrative evidence appraisal • Fictional examples • White-background magazine edition

Dialectical behaviour therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A10

WEAVE
100+ / PSYCHOTHERAPY

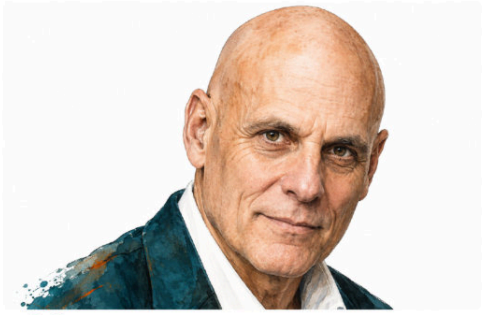
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ACT / PSYCHOLOGICAL FLEXIBILITY

Acceptance & Commitment Therapy

Help people act in valued directions while making room for difficult inner experiences.



Steven C. Hayes
Co-developer of acceptance and commitment therapy

THE CLINICAL IDEA

The question is not whether a thought disappears, but whether life can expand around it.

AI-ILLUSTRATED PORTRAIT

A10

Steven Hayes and colleagues • Functional contextualism • Behavioural tradition

SECTION 01 THE MODEL

1. Notice experience
2. Open up
3. Choose a value
4. Take action

Six processes: acceptance, cognitive defusion, present-moment awareness, self-as-context, values and committed action. Flexibility means choosing workable behaviour in context.

SECTION 02 THE TECHNIQUES

- **Analyse workability.** Explore what the person does to control inner experiences and what those strategies cost in daily life.
- **Practise defusion.** Notice a thought as a mental event: I am having the thought that... The aim is changed function, not proving it false.
- **Clarify values.** Identify chosen qualities of action in relationships, work or care; distinguish values from completed goals.
- **Build committed action.** Choose small observable steps, make room for discomfort, review barriers and adjust with self-compassion.

SECTION 03 IN THE ROOM

Fictional example: A person waits to feel confident before reconnecting with friends. They choose one brief call while allowing anxiety to be present.

SECTION 04 WHAT THE EVIDENCE SAYS

Trials support ACT for several psychological and health-related problems, with effects varying by condition and comparator. It is not consistently established as superior to other bona fide therapies. [1,2]

CLINICAL CAUTION

Acceptance concerns internal experience, not submission to abuse or avoidable harm. Avoid turning mindfulness or values into demands to tolerate an unsafe situation.

SELECTED REFERENCES
1. Hayes SC, Strosahl KD, Wilson KG. Acceptance and Commitment Therapy. 2nd ed. Guilford; 2012.
2. Ost LG. Efficacy of ACT: updated systematic review and meta-analysis. Behav Res Ther. 2014. PMID:25193001.

Acceptance commitment therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A11

WEAVE
100+ / PSYCHOTHERAPY
EXISTENTIAL / MEANING-ORIENTED

THE PSYCHOTHERAPY EDITION
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Logotherapy

Explore sources of meaning without prescribing what suffering should mean to another person.



Viktor E. Frankl
Founder of logotherapy

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

Meaning is discovered or chosen with the person; it is not assigned by the therapist.

AI-ILLUSTRATED PORTRAIT

Viktor Frankl • Freedom within limits • Responsibility and meaning

A11

SECTION 01 THE MODEL

Experience of loss → Meaning enquiry
Meaning enquiry → Available choice
Available choice → Purposeful action
Purposeful action → Experience of loss

Meaning may be approached through creating or contributing, loving or encountering, and one's attitude toward unavoidable suffering. Preventable suffering should still be relieved.

SECTION 02 THE TECHNIQUES

- **Explore meaning concretely.** Ask about relationships, work, contribution, memories and commitments; allow uncertainty and loss of meaning.
- **Use Socratic dialogue.** Help the person articulate their own values and possibilities rather than supplying a therapist's philosophy.
- **Understand classic techniques.** Dereflection redirects excessive self-monitoring; paradoxical intention approaches selected feared responses with deliberate distance and humour.
- **Translate into action.** Identify a small expression of meaning that is feasible despite limits; integrate practical, social and medical care.

SECTION 03 IN THE ROOM

Fictional example: During serious illness, a person chooses to record family stories. The choice expresses connection without requiring optimism about prognosis.

SECTION 04 WHAT THE EVIDENCE SAYS

Modern meaning-centred psychotherapy has trial evidence in advanced cancer. These adapted, manualised interventions do not establish efficacy of classical logotherapy for every psychiatric disorder. [1,2]

CLINICAL CAUTION

Never imply illness reflects insufficient meaning or attitude. Paradoxical intention is not an instruction to enact dangerous behaviour; use only with a suitable formulation.

SELECTED REFERENCES
1. Frankl VE. The Will to Meaning. 1969.
2. Breitbart W et al. Individual meaning-centered psychotherapy: randomized trial in advanced cancer. Cancer. 2018. PMCID:PMC6097940.

WEAVE 100+ / CLINICIAN APPENDIX
Narrative evidence appraisal • Fictional examples • White-background magazine edition

Logotherapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A12

WEAVE
100+ / PSYCHOTHERAPY
NLP / CRITICAL APPRAISAL

THE PSYCHOTHERAPY EDITION
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Neuro-Linguistic Programming

Distinguish appealing communication practices from claims that lack adequate clinical validation.



Richard Bandler and John Grinder
Co-founders of neuro-linguistic programming

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

A borrowed effective technique cannot validate an entire branded explanatory system.

AI-ILLUSTRATED PORTRAIT

Richard Bandler and John Grinder • 1970s • Not natural language processing

A12

SECTION 01 THE MODEL

Specific claim → Testable prediction
Clinical judgement ← Controlled evidence

NLP proposes that modelling language and subjective experience can change behaviour. Terms such as anchoring, representational systems and reframing do not themselves demonstrate a mechanism.

SECTION 02 THE TECHNIQUES

- **Describe claims accurately.** Examples include anchoring, modelling, reframing and the swish pattern. Description is not endorsement or a treatment recommendation.
- **Separate common elements.** Rapport, imagery and goal-setting occur in other therapies; their usefulness does not validate NLP's distinctive theory.
- **Examine evidence.** Look for preregistered trials, credible comparators, independent replication and patient-important outcomes, not testimonials.
- **Communicate honestly.** Do not infer truthfulness from eye movements or promise rapid cures. Offer treatments with evidence for the presenting problem.

SECTION 03 IN THE ROOM

Fictional appraisal: A course claims that eye direction reveals lying. Ask for replicated diagnostic-accuracy evidence before using this in any clinical judgement.

SECTION 04 WHAT THE EVIDENCE SAYS

A systematic review found little evidence that NLP improves health outcomes; included studies were few and methodologically weak. This supports an insufficient-evidence judgement, not proof that every component is ineffective. [1]

CLINICAL CAUTION

Do not market NLP as a validated treatment for serious mental illness or trauma. Consider opportunity cost, exaggerated neuroscience language and unsupported certification claims.

SELECTED REFERENCES
1. Sturt J et al. Neurolinguistic programming: systematic review of effects on health outcomes. Br J Gen Pract. 2012;62:e757-e764. doi:10.3399/bjgp12X658287

Neuro linguistic programming. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A13

WEAVE
100+ / PSYCHOTHERAPY
EXPERIENTIAL / HERE-AND-NOW

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Gestalt Therapy

Increase awareness of how a person experiences, interrupts and completes contact with self, others and environment.

MODEL PRACTICE EVIDENCE



Fritz Perls and Laura Perls
Co-founders of Gestalt therapy

THE CLINICAL IDEA

An experiment invites discovery; a performance demands a particular result.

AI-ILLUSTRATED PORTRAIT

A13

Fritz Perls • Laura Perls • Paul Goodman • Phenomenological and field perspectives

SECTION 01 THE MODEL

1. Present experience
2. Awareness
3. Experiment
4. New contact

Attend to bodily experience, emotion, language and relational context. Figure-ground shifts and interrupted contact are conceptual tools, not neurological diagnoses.

SECTION 02 THE TECHNIQUES

- **Use descriptive enquiry.** Ask what is noticed now: sensation, posture, feeling, impulse and contact with the therapist; avoid imposing explanations.
- **Design an experiment.** Invite a small change in posture, language or expression and observe its effect. The result informs understanding, not a predetermined theory.
- **Explore polarities.** Use two-chair or empty-chair work when appropriate to examine conflicting positions or unfinished relational experience.
- **Integrate the experience.** Reflect on what changed, its meaning and a feasible action outside therapy; catharsis alone is not the endpoint.

SECTION 03 IN THE ROOM

Fictional example: While saying everything is fine, a person clenches their hands. Curious attention helps them recognise anger and practise a direct request.

SECTION 04 WHAT THE EVIDENCE SAYS

Gestalt has an older, limited and heterogeneous outcome literature; the cited meta-analysis is from 1994. Evidence for chairwork or another experiential approach should not be attributed automatically to Gestalt therapy as a whole. [1,2]

CLINICAL CAUTION

Avoid coercive confrontation, theatrical pressure or interpreting body language as fact. Obtain consent for experiments and pace emotional intensity collaboratively.

SELECTED REFERENCES
1. Perls F, Hefferline R, Goodman P. Gestalt Therapy. 1951.
2. Bretz HJ et al. Meta-analysis of the effectiveness of Gestalt therapy. 1994. PMID:7941644.

WEAVE 100+ / CLINICIAN APPENDIX
Narrative evidence appraisal • Fictional examples • White-background magazine edition

Gestalt therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A14

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

EFT / INDIVIDUAL EXPERIENTIAL THERAPY

Emotion-Focused Therapy

Help people access, understand and transform emotion through a safe relationship and carefully chosen experiential tasks.



Leslie S. Greenberg
Co-developer of emotion-focused therapy

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

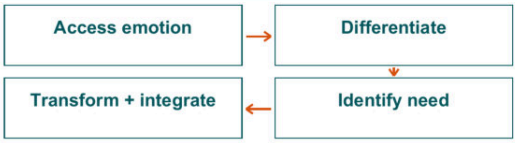
Accessing emotion is a beginning; transformation requires new emotional meaning and action.

AI-ILLUSTRATED PORTRAIT

A14

Leslie Greenberg • Process-experiential tradition • Not the couple model

SECTION 01 THE MODEL



Distinguish primary adaptive emotion, primary maladaptive emotion, secondary reactions and instrumental expression. Emotion schemes organise feeling, meaning, bodily experience and action tendencies.

SECTION 02 THE TECHNIQUES

- **Build emotional safety.** Offer empathic attunement, validation and consent; regulate intensity so the person can remain engaged and reflective.
- **Follow process markers.** Select tasks from the person's current difficulty, such as self-criticism, unresolved hurt or unclear felt experience.
- **Use experiential tasks.** Two-chair dialogue explores self-critical splits; empty-chair work addresses unfinished business; focusing develops felt meaning.
- **Transform and carry forward.** Access adaptive needs, protective anger, grief or compassion, then connect new meaning with interpersonal action.

SECTION 03 IN THE ROOM

Fictional example: A self-critical attack initially produces shame. Chairwork helps the person recognise a need for protection and develop a compassionate response.

SECTION 04 WHAT THE EVIDENCE SAYS

A 2025 pilot randomised 111 adults with major depression to EFT or CBT. Symptoms improved in both conditions; no significant between-group difference was detected. This small trial does not establish equivalence or superiority and does not concern couple EFT or tapping. [1,2]

CLINICAL CAUTION

Do not maximise emotional intensity as a goal. Support regulation and consent; distinguish this approach from emotionally focused couple therapy and tapping-based methods.

SELECTED REFERENCES
1. Greenberg LS. Emotion-Focused Therapy. APA; 2011.
2. Aardal H et al. CBT and EFT for depression: randomized pilot trial. Psychother Res. Online 2025. doi:10.1080/10503307.2025.2560935

WEAVE 100+ / CLINICIAN APPENDIX

Narrative evidence appraisal • Fictional examples • White-background magazine edition

Emotion focused therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A15

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

EFCT / ATTACHMENT + INTERACTION

Emotionally Focused Couple Therapy

Move from defensive cycles toward accessible, responsive and emotionally engaged connection.

MODEL PRACTICE EVIDENCE



Sue Johnson
Co-developer of emotionally focused couple therapy

THE CLINICAL IDEA

Emotion becomes useful when it creates a different, safer interaction.

AI-ILLUSTRATED PORTRAIT

A15

Sue Johnson • Attachment theory • Experiential and systemic practice

SECTION 01 THE MODEL

De-escalate cycle → Access vulnerability → New interaction → Consolidate security → De-escalate cycle

Secondary anger or withdrawal may obscure primary fear and attachment needs. The target is a new pattern of engagement, not simply better communication vocabulary.

SECTION 02 THE TECHNIQUES

- **Map and de-escalate.** Identify the negative cycle and each partner's position, meanings and attachment fears; externalise the cycle without excusing harm.
- **Deepen emotion.** Help partners name primary vulnerability and needs beneath protective protest or withdrawal.
- **Shape enactments.** Support partners in expressing those needs directly and responding differently to each other in the session.
- **Consolidate change.** Review new interactions, solve practical problems and develop a shared account of repair and reconnection.

SECTION 03 IN THE ROOM

Fictional example: Beneath angry protest, one partner describes fear of being unimportant. The other practises responding to that need rather than the attack.

SECTION 04 WHAT THE EVIDENCE SAYS

A small trial of 16 couples found greater relationship-satisfaction gains with EFCT than usual couple therapy; a depressive-symptom advantage was reported in men. This preliminary study does not justify guaranteed relationship repair or broad claims about treating depression. [1,2]

CLINICAL CAUTION

Assess coercive control and relationship safety before conjoint work. Vulnerability disclosure must be safe and voluntary; attachment language must never excuse abuse.

SELECTED REFERENCES
1. Johnson SM. The Practice of Emotionally Focused Couple Therapy. 3rd ed. Routledge; 2019.
2. Wittenborn AK et al. EFCT versus usual care for depression: RCT. J Marital Fam Ther. 2019;45:395-409. doi:10.1111/jmft.12350

Emotionally focused couple therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A16

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

EYE MOVEMENT DESENSITISATION & REPROCESSING

EMDR

A structured trauma-focused treatment: more than eye movements, and more than recalling a difficult event.



Francine Shapiro
Originator of EMDR

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

Evidence supports the clinical protocol; mechanistic explanations require separate evidence.

AI-ILLUSTRATED PORTRAIT

A16

Francine Shapiro • Eight-phase protocol • Adaptive information processing model

SECTION 01 THE MODEL

1. Assess + prepare
2. Target memory
3. Reprocess
4. Re-evaluate

The adaptive information processing model proposes that distressing memories can remain inadequately integrated. It is a treatment model; its full biological mechanism is not established.

SECTION 02 THE TECHNIQUES

- **Know all eight phases.** History/planning; preparation; assessment; desensitisation; installation; body scan; closure; re-evaluation.
- **Assess the target.** Specify image, negative and preferred positive cognition, emotion, distress and body sensation within the protocol.
- **Use dual attention.** Follow a trained protocol with bilateral stimulation and brief reporting, adjusting to the person's response and capacity.
- **Close and revisit.** Ensure a safe session ending, agree between-session support and reassess target distress, current triggers and future coping.

SECTION 03 IN THE ROOM

Fictional example: A road-collision memory remains linked to danger in safe settings. Treatment monitors distress and meaning while revisiting the target in a structured way.

SECTION 04 WHAT THE EVIDENCE SAYS

VA/DoD recommends EMDR among individual trauma-focused psychotherapies for PTSD. Clinical efficacy does not prove popular claims about hemisphere balancing, memory erasure or a fully established mechanism. [1,2]

CLINICAL CAUTION

Use trained clinicians, informed consent and assessment of safety and dissociation. Do not use suggestive questioning or treat emerging imagery as verified historical memory.

SELECTED REFERENCES
1. Shapiro F. Eye Movement Desensitization and Reprocessing Therapy. 3rd ed. Guilford; 2018.
2. VA/DoD. PTSD guideline; 2023. ptsd.va.gov/professional/treat/txessentials/overview_therapy.asp

EMDR. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A17

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

MBT / REFLECTING ON MINDS

Mentalization-Based Treatment

Restore curiosity about mental states when attachment stress makes certainty feel easier than reflection.



Peter Fonagy and Anthony Bateman
Co-developers of mentalization-based treatment

THE CLINICAL IDEA

First restore the capacity to wonder; then explore what the behaviour might mean.

MODEL PRACTICE EVIDENCE

AI-ILLUSTRATED PORTRAIT

A17

Anthony Bateman and Peter Fonagy • Attachment • Reflective functioning

SECTION 01 THE MODEL

Attachment arousal → Mentalizing narrows
↓
Explore perspectives ← Pause + regulate

Mentalizing links behaviour with possible thoughts, feelings, wishes and intentions. Minds are partly opaque; a plausible interpretation remains open to revision.

SECTION 02 THE TECHNIQUES

- **Notice loss of mentalizing.** Watch for psychic equivalence (feeling becomes fact), pretend mode (detached talk) and teleological thinking (only actions count).
- **Lower arousal first.** Validate the affect, slow the exchange and return to a concrete recent event before exploring complex meanings.
- **Use a not-knowing stance.** Clarify what happened, identify the person's perspective and gently consider alternatives without pretending to read minds.
- **Work with the relationship.** Stop, rewind and examine a misunderstanding in the session; acknowledge the therapist's contribution and repair ruptures.

SECTION 03 IN THE ROOM

Fictional example: An unanswered message means definite rejection. The therapist validates the hurt, then explores what is known and what remains uncertain.

SECTION 04 WHAT THE EVIDENCE SAYS

MBT has trial evidence in BPD and is delivered in structured programmes. Relative benefits across specialist treatments and outcomes remain uncertain; mentalizing language alone is not a complete programme. [1,2]

CLINICAL CAUTION

Detailed interpretations during high arousal can worsen confusion. Curiosity must be genuine, and risk management remains explicit rather than purely exploratory.

SELECTED REFERENCES
1. Bateman A, Fonagy P. Mentalization-Based Treatment for Personality Disorders. Oxford UP; 2016.
2. Oud M et al. Aust N Z J Psychiatry. 2018. doi:10.1177/0004867418791257

Mentalization based treatment. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A18

WEAVE
100+ / PSYCHOTHERAPY
REBT / BELIEFS + ACCEPTANCE

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE
SEPTEMBER 2026

Rational Emotive Behaviour Therapy

Challenge rigid demands and global self-rating while building flexible preferences and effective action.

MODEL PRACTICE EVIDENCE



Albert Ellis
Founder of rational emotive behaviour therapy

THE CLINICAL IDEA

Prefer strongly, tolerate discomfort, and evaluate behaviour without condemning the whole person.

AI-ILLUSTRATED PORTRAIT

A18

Albert Ellis • ABC model • Cognitive, emotive and behavioural methods

SECTION 01 THE MODEL

A: activating event → B: belief
↓
D/E: dispute + new belief ← C: consequence

Demandingness can lead to awfulizing, low frustration tolerance and global evaluations of self or others. REBT distinguishes healthy negative emotion from debilitating emotional responses.

SECTION 02 THE TECHNIQUES

- **Map ABC precisely.** Separate the event from beliefs and emotional/behavioural consequences; identify the specific rigid demand.
- **Dispute collaboratively.** Examine logic, evidence and practical usefulness. Replace musts with strong preferences, not indifference or denial.
- **Practise acceptance.** Develop unconditional self-, other- and life-acceptance without approving harmful behaviour or abandoning change.
- **Test new beliefs.** Use rational coping statements, imagery and graded behavioural tasks; review whether action changes alongside belief.

SECTION 03 IN THE ROOM

Fictional example: I must never fail becomes I strongly want to succeed, but a failure does not define my worth. The person then attempts an avoided task.

SECTION 04 WHAT THE EVIDENCE SAYS

Reviews report benefits across varied settings and outcomes, but methodological quality and populations differ. Do not interpret pooled effects as proof of superiority for every diagnosis. [1,2]

CLINICAL CAUTION

A forceful therapist style is not essential. Validate suffering; disputation should not become argument, humiliation or dismissal of actual injustice.

SELECTED REFERENCES
1. Ellis A, Dryden W. The Practice of Rational Emotive Behavior Therapy. 2nd ed. Springer; 1997.
2. David D et al. 50 years of REBT: systematic review and meta-analysis. J Clin Psychol. 2018. PMCID:PMC5836900.

Rational emotive behaviour therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A19

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

INTERPERSONAL & SOCIAL RHYTHM THERAPY

IPSRT

Protect daily rhythms while addressing interpersonal stress that can destabilise mood.



Ellen Frank
Developer of interpersonal and social rhythm therapy

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

Treat the relationship event and the rhythm disruption it creates.

AI-ILLUSTRATED PORTRAIT

A19

Ellen Frank • Bipolar disorder • Adjunct to ongoing clinical treatment

SECTION 01 THE MODEL

1. **Interpersonal event**
2. **Rhythm disruption**
3. **Mood vulnerability**
4. **Regularity + repair**

Social zeitgebers such as waking, meals and contact help organise daily rhythms. Interpersonal disruption can alter these routines and increase vulnerability to episodes.

SECTION 02 THE TECHNIQUES

- **Chart rhythms and mood.** Use a Social Rhythm Metric or diary to track waking, meals, activity, social contact and mood over time.
- **Build achievable regularity.** Agree consistent anchors, especially sleep-wake timing; plan for shifts, travel and weekends without rigid perfectionism.
- **Address interpersonal themes.** Work on grief, role disputes, role transitions and interpersonal difficulties, including loss associated with illness.
- **Plan early response.** Link rhythm changes to personal warning signs, medication adherence and a shared relapse plan with the treating team.

SECTION 03 IN THE ROOM

Fictional example: A job change disrupts waking and meals. Therapy combines role-transition work with realistic daily anchors and early-warning monitoring.

SECTION 04 WHAT THE EVIDENCE SAYS

Randomized research supports IPSRT as an adjunct in bipolar disorder, including maintenance-related outcomes. It is not a stand-alone treatment for acute mania or a replacement for indicated pharmacotherapy. [1,2]

CLINICAL CAUTION

Do not confuse rhythm regularisation with sleep restriction. Monitor reduced need for sleep, activation and safety; adjust plans promptly with the treating clinician.

SELECTED REFERENCES
1. Frank E. Treating Bipolar Disorder: A Clinician's Guide to IPSRT. Guilford; 2005.
2. Frank E et al. Two-year outcomes for IPSRT in bipolar I disorder. Arch Gen Psychiatry. 2005. PMID:16143731.

IPSRT. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A20

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

SYSTEMIC WORK / FAMILY INTERVENTIONS

Family Therapy

Understand symptoms in a relational context while protecting each person's voice, autonomy and safety.



Salvador Minuchin and Virginia Satir
Pioneers of family therapy

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

Think systemically, intervene specifically, and keep responsibility for harm clear.

AI-ILLUSTRATED PORTRAIT

A20

Structural • Strategic • Bowenian • Systemic • Psychoeducational approaches

SECTION 01 THE MODEL

Pattern + context → Shared formulation
↓
Review functioning ← New interactions

Circular causality asks how responses influence one another over time. It does not imply that families cause psychiatric disorders or share equal responsibility for harmful acts.

SECTION 02 THE TECHNIQUES

- **Map the system.** Use a genogram, family timeline and interaction map; include strengths, culture, roles, boundaries and external stressors.
- **Choose the approach.** Structural work examines boundaries and hierarchies; systemic enquiry explores meanings; psychoeducation supports coping and relapse prevention.
- **Practise concrete skills.** Use communication rehearsal, problem-solving, agreed responsibilities and an early-warning or crisis response plan.
- **Review multiple perspectives.** Measure patient outcomes, family burden and functioning; clarify consent, confidentiality and whose goals guide treatment.

SECTION 03 IN THE ROOM

Fictional example: Relatives respond to withdrawal with repeated criticism. Psychoeducation and a calmer problem-solving routine reduce conflict and support help-seeking.

SECTION 04 WHAT THE EVIDENCE SAYS

Family interventions have evidence for relapse prevention in schizophrenia. This applies to specified interventions, often with psychoeducation and skills, rather than every systemic theory or every family problem. [1,2]

CLINICAL CAUTION

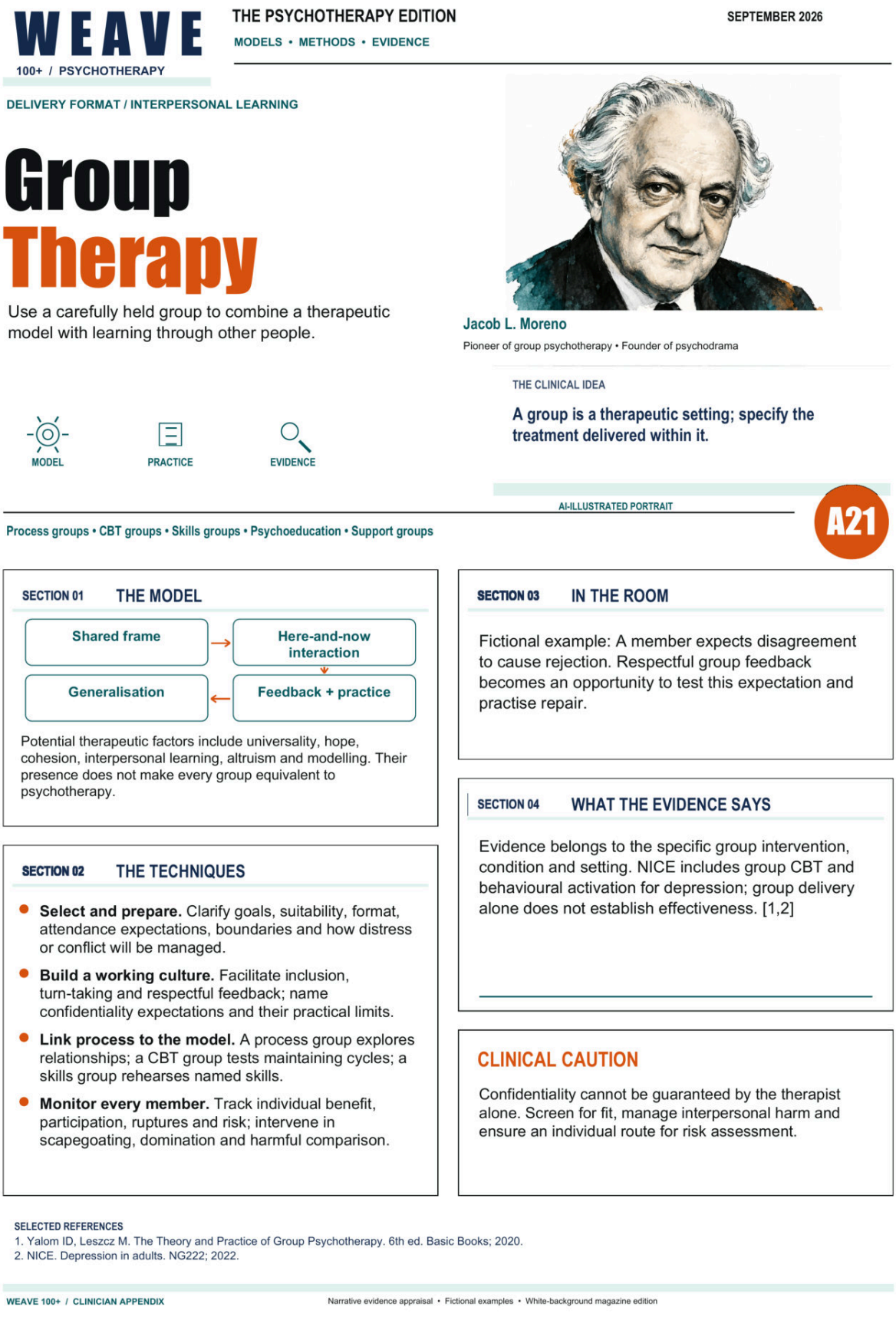
Never use expressed emotion to blame relatives. Assess violence, coercion and safeguarding; conjoint sessions may be unsuitable when disclosure creates danger.

SELECTED REFERENCES
1. Minuchin S. Families and Family Therapy. Harvard UP; 1974.
2. Rodolico A et al. Family interventions for relapse prevention in schizophrenia. Lancet Psychiatry. 2022. PMID:35093198.

Family therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A21



Group therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A22

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

JPMR / SOMATIC SKILL

Jacobson Progressive Muscle Relaxation

Learn to recognise muscular tension and release it deliberately, then apply that discrimination in daily life.



Edmund Jacobson
Developer of progressive muscle relaxation

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

The learning target is noticing and releasing tension, not achieving perfect relaxation.

AI-ILLUSTRATED PORTRAIT

A22

Edmund Jacobson • Progressive muscle relaxation • Adapted brief protocols

SECTION 01 THE MODEL

1. Notice tension
2. Gently tense
3. Release + compare
4. Generalise

The core skill is discrimination between tension and relaxation across muscle groups. Modern abbreviated protocols differ from Jacobson's original training programme.

SECTION 02 THE TECHNIQUES

- **Prepare comfortably.** Explain the method, choose supported sitting or lying, and check pain, injury and physical limitations.
- **Use gentle cycles.** For a suitable muscle group, tense gently for about 5 seconds, then release for 10-20 seconds; breathe comfortably and avoid strain.
- **Progress systematically.** Move through selected groups such as hands, arms, shoulders, face and legs; skip painful areas or use release-only attention.
- **Apply the learning.** Practise regularly, then notice and release unnecessary tension during ordinary tasks; record comfort and functional benefit.

SECTION 03 IN THE ROOM

Fictional example: A student notices clenched shoulders while studying. Brief release practice reduces tension and allows a return to the task.

SECTION 04 WHAT THE EVIDENCE SAYS

Reviews suggest benefit for stress and anxiety symptoms, but studies vary in population, protocol and quality. JPMR is a useful skill or adjunct, not a disorder-specific treatment for every presentation. [1,2]

CLINICAL CAUTION

Stop painful contractions or dizziness. Avoid breath-holding and maximal effort. Offer eyes-open or externally focused alternatives if body attention increases distress.

SELECTED REFERENCES
1. Jacobson E. Progressive Relaxation. University of Chicago Press; 1938.
2. Muhammad Khir S et al. Efficacy of PMR in adults: systematic review. Psychol Res Behav Manag. 2024;17:345-365. doi:10.2147/PRBM.S437277

Jacobson progressive muscle relaxation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A23

WEAVE
100+ / PSYCHOTHERAPY
SKILLS / TARGETED ADJUNCTS

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE
SEPTEMBER 2026

Relaxation Techniques

Choose the method for the maintaining process; feeling calm is useful, but it is not the aim of every psychotherapy.

MODEL PRACTICE EVIDENCE



Johannes Heinrich Schultz
Developer of autogenic training

THE CLINICAL IDEA

Use a skill to support valued functioning, not to make all discomfort forbidden.

AI-ILLUSTRATED PORTRAIT

A23

Breathing • Muscle relaxation • Imagery • Applied relaxation • Autogenic training

SECTION 01 THE MODEL

Identify target → Choose method
Practise gently → Evaluate function

Relaxation can reduce arousal or muscular tension. Grounding instead orients attention to present safety; mindfulness changes the relationship to experience and need not produce calm.

SECTION 02 THE TECHNIQUES

- **Paced breathing.** Encourage comfortable, slower breathing without large forced breaths. Stop if light-headed; avoid routine breath-holding prescriptions.
- **Muscle relaxation.** Use tension-release discrimination or release-only practice adapted to physical comfort.
- **Imagery and autogenic work.** Use a personally acceptable image or sensations of warmth/heaviness; offer alternatives when imagery is distressing.
- **Applied relaxation.** Learn early cue detection and progressively practise rapid relaxation in real situations as part of an appropriate protocol.

SECTION 03 IN THE ROOM

Fictional example: Before a difficult call, a person uses brief comfortable breathing, then makes the call even though some anxiety remains.

SECTION 04 WHAT THE EVIDENCE SAYS

Evidence varies by technique and condition. NICE includes applied relaxation as a high-intensity option for GAD; this does not validate every breathing exercise or wellness claim. [1]

CLINICAL CAUTION

In exposure therapy, mandatory calming rituals may become safety behaviours. Adapt inward attention for trauma or panic sensitivity; do not promise autonomic reset or cure.

SELECTED REFERENCES
1. NICE. Generalised anxiety disorder and panic disorder in adults. CG113; applied relaxation recommendations. nice.org.uk/guidance/cg113
2. Jacobson E. Progressive Relaxation. 1938.

WEAVE 100+ / CLINICIAN APPENDIX
Narrative evidence appraisal • Fictional examples • White-background magazine edition

Relaxation techniques. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A24

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

ACTIVE SUPPORT / ADAPTIVE FUNCTION

Supportive Psychotherapy

Strengthen coping, self-esteem, relationships and reality-based functioning through a purposeful therapeutic relationship.



John C. Markowitz
Developer of brief supportive psychotherapy

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

Supportive psychotherapy is an active method with goals, techniques and review.

AI-ILLUSTRATED PORTRAIT

A24

Psychodynamic and common-factor traditions • Flexible intensity and duration

SECTION 01 THE MODEL

Contain distress → Strengthen coping → Mobilise support → Restore function → Contain distress

Supportive work actively reinforces adaptive capacities and helps manage stress. It is more than friendly conversation, reassurance or an absence of interpretation.

SECTION 02 THE TECHNIQUES

- **Clarify and validate.** Listen accurately, name emotions and summarise problems in manageable terms; offer realistic hope without false reassurance.
- **Support practical agency.** Break tasks into steps, anticipate stressors, rehearse coping and help the person use social and service supports.
- **Offer judicious guidance.** Give advice with permission, explain the rationale and preserve choice; reinforce strengths and adaptive defences.
- **Maintain a frame.** Agree goals and review functioning, symptoms, risk and dependence; plan endings or continuity explicitly.

SECTION 03 IN THE ROOM

Fictional example: During a destabilising transition, sessions focus on manageable routines, a difficult conversation and reconnecting with dependable support.

SECTION 04 WHAT THE EVIDENCE SAYS

A narrative review identified nine trials of manualised brief supportive psychotherapy, mainly in mood disorders. This nondirective, affect-focused approach is narrower than all supportive practice. An active control can itself be therapeutic; similar outcomes do not establish equivalence. [1,2]

CLINICAL CAUTION

Avoid reflex reassurance, rescuing and indefinite dependence. Support must not replace necessary disorder-specific treatment, social intervention or risk management.

SELECTED REFERENCES
1. Pinsker H. A Primer of Supportive Psychotherapy. Analytic Press; 1997.
2. Markowitz JC. Supportive Evidence. Am J Psychother. 2022;75:122-128. doi:10.1176/appi.psychotherapy.2021.20210041

Supportive psychotherapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A26

WEAVE
100+ / PSYCHOTHERAPY
A FAMILY OF SPECIFIC TREATMENTS

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE
SEPTEMBER 2026

Trauma-Focused Therapy

Work directly with trauma-related memories, meanings or avoidance through a chosen, evidence-based protocol.



Edna B. Foa and Patricia A. Resick
Developers of PE and CPT, respectively

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

Trauma-informed describes how care is offered; trauma-focused describes what treatment targets.

AI-ILLUSTRATED PORTRAIT

A26

PE • CPT • EMDR • Trauma-focused CBT • Age and protocol matter

SECTION 01 THE MODEL

Assess + choose → Prepare proportionately
↓
Process trauma → Reclaim life

Avoidance and persistent threat meanings can maintain PTSD. Trauma-focused treatments approach these processes differently; trauma-informed care is a broader service stance, not the same intervention.

SECTION 02 THE TECHNIQUES

- **Assess and choose together.** Review trauma symptoms, current danger, dissociation, comorbidity, goals and preferences; agree a risk and support plan.
- **Prolonged exposure.** Use protocol-based imaginal and in-vivo exposure to reduce avoidance and build corrective learning.
- **Cognitive processing therapy.** Identify and examine stuck points involving safety, trust, power, esteem and intimacy; distinguish responsibility from hindsight blame.
- **Respect protocol differences.** EMDR follows eight phases. Child/adolescent TF-CBT typically includes caregiver work and developmentally adapted trauma processing.

SECTION 03 IN THE ROOM

Fictional example: A person avoids safe journeys after an assault. A shared formulation guides gradual return to activities while addressing persistent threat beliefs.

SECTION 04 WHAT THE EVIDENCE SAYS

VA/DoD strongly recommends PE, CPT and EMDR for adult PTSD. NICE also recommends age-appropriate trauma-focused treatments. Evidence for an established protocol cannot be assumed for every method labelled trauma therapy. [1,2]

CLINICAL CAUTION

Do not force detailed retelling or routine single-session debriefing. Address immediate danger; prepare enough for engagement without imposing an indefinite stabilisation requirement on everyone.

SELECTED REFERENCES
1. VA/DoD. Clinical Practice Guideline for PTSD and Acute Stress Disorder; 2023.
2. NICE. Post-traumatic stress disorder. NG116; 2018. nice.org.uk/guidance/ng116

WEAVE 100+ / CLINICIAN APPENDIX
Narrative evidence appraisal • Fictional examples • White-background magazine edition

Trauma focused therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A27

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

IPT / MOOD + CURRENT RELATIONSHIPS

Interpersonal Psychotherapy

Treat symptoms by working on a focused current interpersonal problem and strengthening social support.



Gerald L. Klerman and Myrna M. Weissman
Leading developers of interpersonal psychotherapy

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

Name the current interpersonal focus and show how work on it relates to symptoms.

AI-ILLUSTRATED PORTRAIT

A27

Gerald Klerman • Myrna Weissman • Time-limited and structured

SECTION 01 THE MODEL

Interpersonal inventory → Choose focus
Consolidate + end ← Practise change

Four classic problem areas: grief, role disputes, role transitions and interpersonal deficits/sensitivities. IPT links symptoms and relationship events without treating all problems as unconscious conflict.

SECTION 02 THE TECHNIQUES

- **Begin with a shared account.** Assess symptoms, make an interpersonal inventory and link the current episode to a chosen problem area.
- **Work with the focus.** Facilitate mourning, clarify a dispute, support a role transition or develop connections according to the formulation.
- **Analyse communication.** Reconstruct a recent exchange, clarify feelings and expectations, then rehearse more effective communication and problem-solving.
- **Plan termination.** Review gains, identify warning signs and support resources, and discuss the meaning of ending treatment.

SECTION 03 IN THE ROOM

Fictional example: Depression follows relocation and loss of familiar roles. Therapy focuses on grieving the old role and building relationships in the new one.

SECTION 04 WHAT THE EVIDENCE SAYS

IPT is an evidence-based option for depression. WHO has a structured eight-session group IPT manual; that delivery adaptation should be distinguished from standard individual IPT and from IPSRT. [1,2]

CLINICAL CAUTION

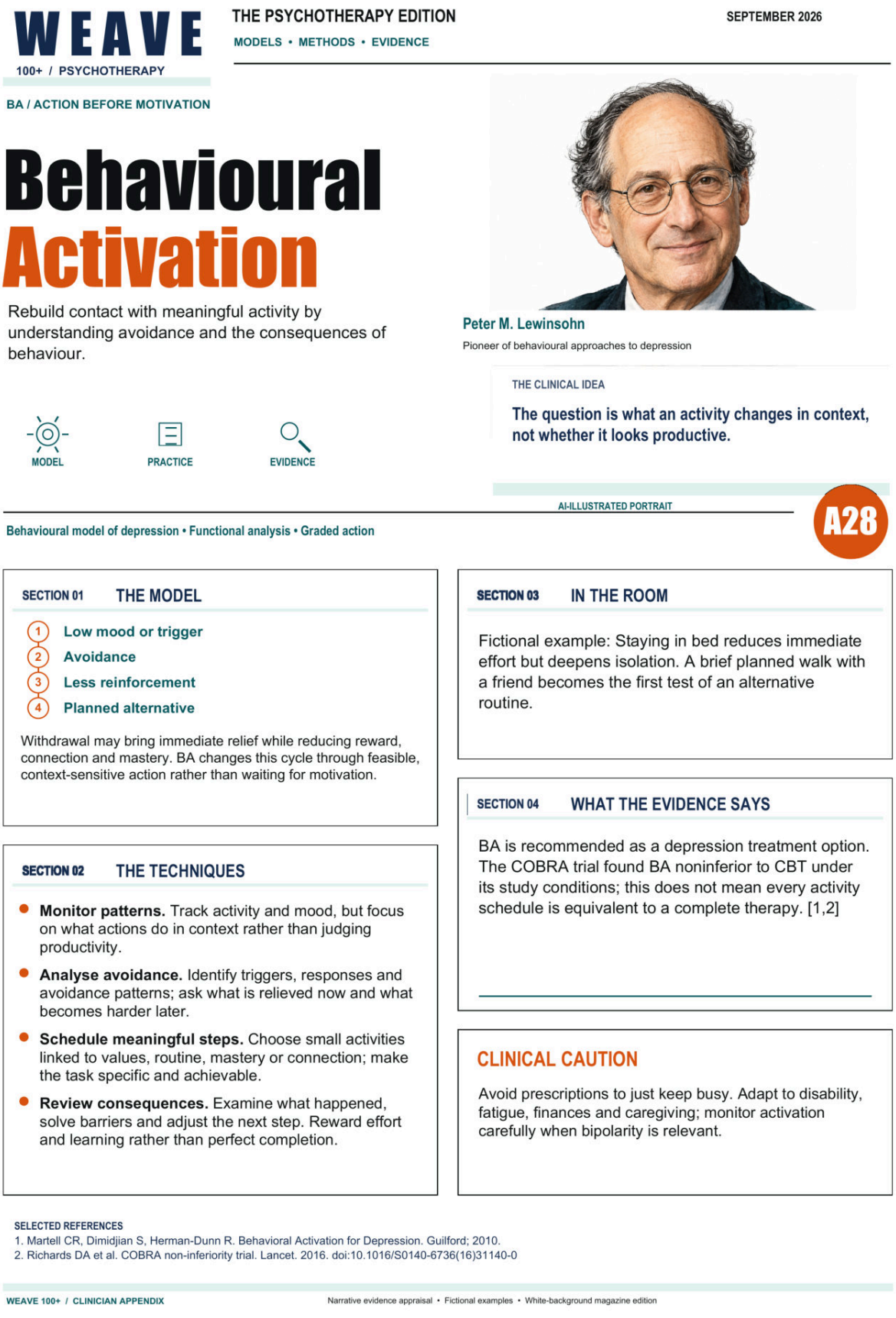
Do not reduce structural adversity to communication skill. Address practical supports and ongoing safety alongside the interpersonal focus.

SELECTED REFERENCES
1. Weissman MM, Markowitz JC, Klerman GL. The Guide to Interpersonal Psychotherapy. Oxford UP; 2018.
2. WHO. Group Interpersonal Therapy (IPT) for Depression. 2016. who.int/publications-detail-redirect/group-interpersonal-therapy-for-depression

Interpersonal psychotherapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A28



Behavioural activation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A29

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

EXPOSURE / ERP / INHIBITORY LEARNING

Exposure & Response Prevention

Approach feared but sufficiently safe situations while reducing rituals and other responses that prevent new learning.



Victor Meyer
Pioneer of exposure and response prevention for OCD

THE CLINICAL IDEA

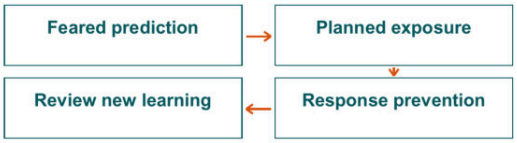
Success is new learning and less ritual control, not proving that risk never exists.

AI-ILLUSTRATED PORTRAIT

A29

Behavioural learning • Anxiety disorders • ERP is central to OCD treatment

SECTION 01 THE MODEL



Avoidance and rituals can produce short-term relief while maintaining threat beliefs. Exposure creates opportunities to learn that feared outcomes are less inevitable or discomfort more tolerable than expected.

SECTION 02 THE TECHNIQUES

- **Specify the feared outcome.** Identify triggers, predictions, compulsions and subtle neutralising, including reassurance seeking and mental rituals.
- **Plan collaboratively.** Choose a graded but meaningful task, agree boundaries and distinguish ordinary precautions from excessive safety behaviour.
- **Prevent the maintaining response.** Practise contact with the trigger without the target ritual; reduce therapist reassurance that functions as a compulsion.
- **Review and vary.** Compare prediction with outcome, repeat across contexts and plan for setbacks; success need not require anxiety to reach zero.

SECTION 03 IN THE ROOM

Fictional example: A person sends a routine email after one review and resists repeated checking, then observes what actually happens over the agreed period.

SECTION 04 WHAT THE EVIDENCE SAYS

CBT including ERP is guideline-supported for OCD. Exposure methods also have disorder-specific evidence for phobias and anxiety; the precise task and response-prevention component depend on formulation. [1,2]

CLINICAL CAUTION

Never use coercive exposure or create genuine danger. Obtain consent and distinguish therapeutic uncertainty from ignoring medical, occupational or safeguarding precautions.

SELECTED REFERENCES
1. NICE. Obsessive-compulsive disorder and body dysmorphic disorder. CG31. nice.org.uk/guidance/cg31
2. Craske MG et al. Maximizing exposure therapy: an inhibitory learning approach. Behav Res Ther. 2014. doi:10.1016/j.brat.2014.04.006

Exposure response prevention. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A30

WEAVE
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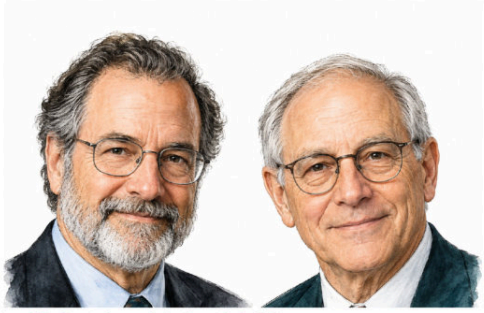
SEPTEMBER 2026

CBT-I / MULTICOMPONENT TREATMENT

CBT for Insomnia

Re-establish sleep regulation by changing habits, conditioned arousal and beliefs that perpetuate insomnia.

MODEL PRACTICE EVIDENCE



Richard R. Bootzin and Arthur J. Spielman
Developers of key behavioural components of CBT-I

THE CLINICAL IDEA

CBT-I is a structured treatment, not a list of sleep hygiene tips.

AI-ILLUSTRATED PORTRAIT

A30

Sleep diary • Stimulus control • Time-in-bed adjustment • Cognitive work

SECTION 01 THE MODEL

Sleep assessment → Behavioural plan
↓
Weekly adjustment → Relapse prevention

Time in bed, sleep drive, circadian timing and conditioned arousal interact. Trying harder to sleep and extending time in bed can unintentionally maintain insomnia.

SECTION 02 THE TECHNIQUES

- **Assess and monitor.** Use a sleep diary and clinical history; check other sleep disorders, substances, medication and relevant medical or psychiatric conditions.
- **Use stimulus control.** Strengthen the bed-sleep association with a consistent waking time and a plan for prolonged wakefulness adapted to the person.
- **Adjust time in bed.** Use clinician-guided sleep restriction or compression based on diary data, safety and response; review sleepiness and functioning.
- **Address sleep beliefs.** Reduce catastrophic predictions and sleep effort; add appropriate relaxation, education and a plan for future disruption.

SECTION 03 IN THE ROOM

Fictional example: A person spends increasing hours in bed trying to recover lost sleep. A monitored plan restores a more consistent schedule and reduces sleep effort.

SECTION 04 WHAT THE EVIDENCE SAYS

AASM strongly recommends multicomponent CBT-I for chronic insomnia in adults. Sleep hygiene alone is not an equivalent treatment; component choice and delivery should follow assessment. [1]

CLINICAL CAUTION

Time-in-bed restriction needs special caution with bipolar disorder, seizure vulnerability, excessive sleepiness and safety-critical work. Do not give a universal unsupervised sleep window.

SELECTED REFERENCES
1. Edinger JD et al. Behavioral and psychological treatments for chronic insomnia disorder in adults: AASM guideline. J Clin Sleep Med. 2021. doi:10.5664/jcsm.8986

WEAVE 100+ / CLINICIAN APPENDIX

Narrative evidence appraisal • Fictional examples • White-background magazine edition

CBT for insomnia. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A31

WEAVE
100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION
MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

MBCT / RELAPSE PREVENTION

Mindfulness-Based Cognitive Therapy

Recognise early patterns of depressive thinking and relate to them with awareness rather than automatic rumination.



Zindel Segal, Mark Williams and John Teasdale
Co-developers of mindfulness-based cognitive therapy

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

The skill is noticing a familiar mental process before it dictates the next action.

AI-ILLUSTRATED PORTRAIT

A31

Zindel Segal • Mark Williams • John Teasdale • Structured group programme

SECTION 01 THE MODEL

1. Early mood shift
2. Notice thinking
3. Decentre
4. Respond deliberately

A small mood shift can reactivate familiar negative thinking. Decentring means experiencing thoughts as mental events rather than facts or commands.

SECTION 02 THE TECHNIQUES

- **Teach through practice.** Use body scan, mindful movement and sitting practice within a structured course; explore experience through enquiry.
- **Recognise automatic pilot.** Notice rumination, avoidance and habitual reactions to unpleasant feelings without adding self-criticism.
- **Use the breathing space.** Practise a brief pause to notice experience, gather attention and widen awareness; adapt it when inward attention is unhelpful.
- **Build a relapse plan.** Identify personal warning signs, helpful actions and when to seek care; connect practice with daily functioning.

SECTION 03 IN THE ROOM

Fictional example: A mild mood dip triggers the thought that another episode is inevitable. The person recognises the pattern and follows an agreed early-response plan.

SECTION 04 WHAT THE EVIDENCE SAYS

Individual-patient meta-analysis supports MBCT for prevention of depressive relapse in recurrent depression. It is not evidence that all mindfulness apps or meditation courses are equivalent to MBCT. [1,2]

CLINICAL CAUTION

Adapt practice for trauma sensitivity, dissociation or destabilisation. Discuss medication changes with the prescriber; completing MBCT does not automatically justify stopping treatment.

SELECTED REFERENCES
1. Segal ZV, Williams JMG, Teasdale JD. Mindfulness-Based Cognitive Therapy for Depression. 2nd ed. Guilford; 2013.
2. Kuyken W et al. Individual patient data meta-analysis of MBCT for relapse prevention. JAMA Psychiatry. 2016. PMCID:PMC6640038.

WEAVE 100+ / CLINICIAN APPENDIX

Narrative evidence appraisal • Fictional examples • White-background magazine edition

Mindfulness based cognitive therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · A32

WEAVE

100+ / PSYCHOTHERAPY

THE PSYCHOTHERAPY EDITION

MODELS • METHODS • EVIDENCE

SEPTEMBER 2026

PST / STRUCTURED COPING

Problem-Solving Therapy

Turn an overwhelming situation into a defined, workable problem and a tested next step.



Thomas J. D'Zurilla and Marvin R. Goldfried

Originators of the problem-solving therapy model

MODEL PRACTICE EVIDENCE

THE CLINICAL IDEA

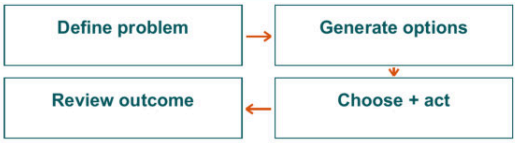
Choose a problem small enough to act on and important enough to matter.

AI-ILLUSTRATED PORTRAIT

A32

Problem orientation • Practical skills • Brief structured intervention

SECTION 01 THE MODEL



Distress can narrow perceived choices and undermine action. PST combines a constructive problem orientation with a repeatable method for addressing solvable difficulties.

SECTION 02 THE TECHNIQUES

- **Define one problem.** Describe a specific situation under partial control, separate facts from assumptions and choose a realistic goal.
- **Generate alternatives.** Brainstorm before evaluating; include seeking support, changing the environment or accepting a genuine limit.
- **Choose and plan.** Weigh feasibility, benefits and costs, then specify who will do what, when and with which resources.
- **Review without blame.** Check the result, reinforce effort, learn from obstacles and revise the plan rather than treating a setback as personal failure.

SECTION 03 IN THE ROOM

Fictional example: Missed appointments are reframed as a transport-and-timing problem. The person compares two feasible options and tests one for the next visit.

SECTION 04 WHAT THE EVIDENCE SAYS

Meta-analytic evidence supports PST for depression, although effects depend on study quality and comparator. A concrete method is especially useful when practical problems contribute to distress. [1,2]

CLINICAL CAUTION

Some problems require protection, resources or collective action rather than individual coping. Do not frame abuse, poverty or discrimination as deficient problem-solving.

SELECTED REFERENCES
1. Nezu AM, Nezu CM, D'Zurilla TJ. Problem-Solving Therapy: A Treatment Manual. Springer; 2013.
2. Cuijpers P et al. Problem-solving therapy for adult depression: updated meta-analysis. Eur Psychiatry. 2018;48:27-37. doi:10.1016/j.eurpsy.2017.11.006

Problem solving therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychotherapies.

Credits and references: retained within the illustration.

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

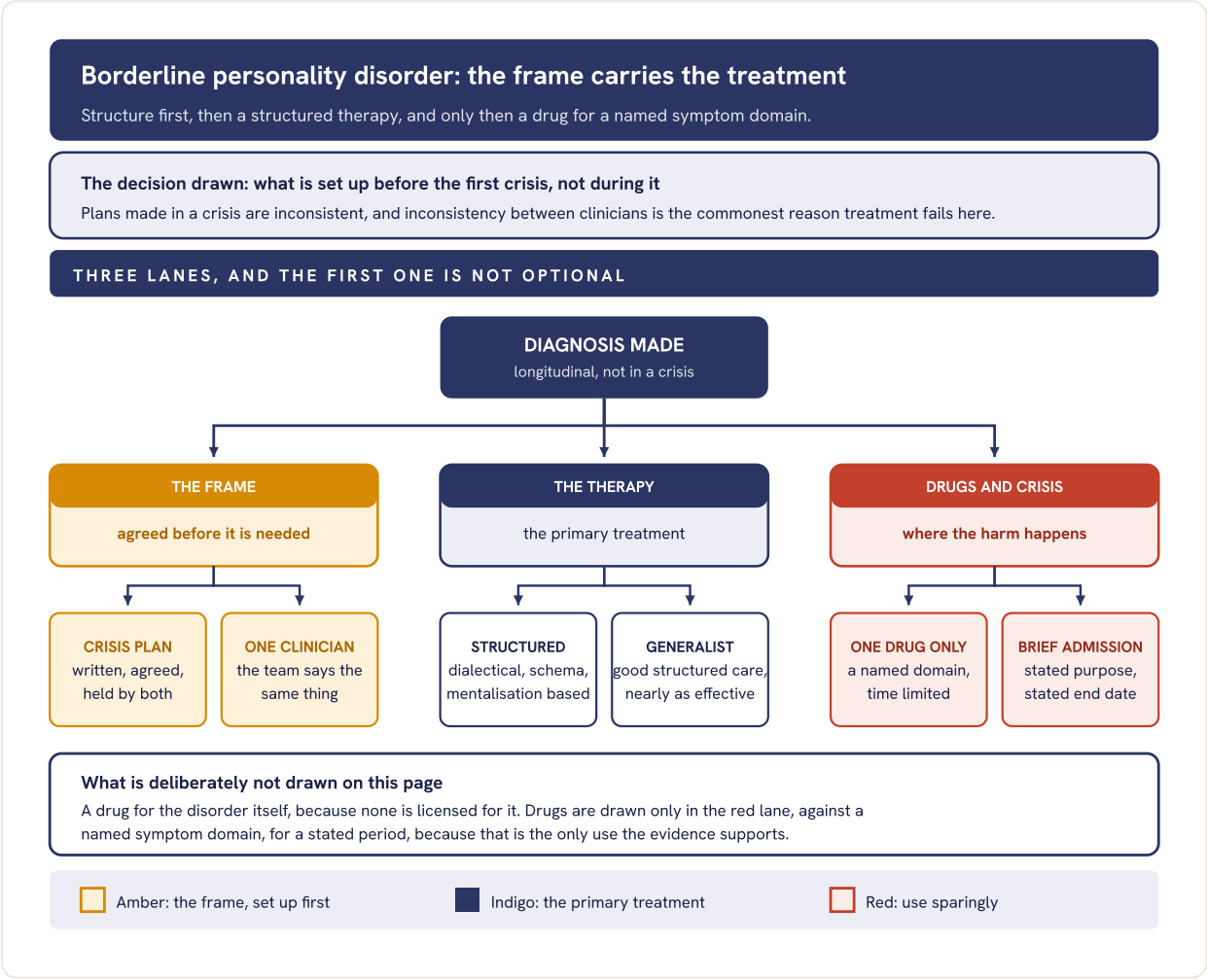


Fig 073.1 The frame drawn as a lane of its own and placed first, because it is what makes the other two safe rather than a preliminary to them.

- **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

WEAVE 100 · PAPER II · 74.1

Personality disorders: diagnosis

ICD-10 → ICD-11 · DSM-5 categorical model retained in DSM-5-TR (2022)

First establish a disorder, not merely a personality style

- Enduring, pervasive maladaptation with distress/impairment; assess culture and development.
- Assess longitudinally; exclude episodes, psychosis, developmental, substance and medical causes.

ICD-10 · CDDG 1992

F60: specific personality disorders.
- Longstanding disharmony in several personality domains; pervasive maladaptation.
- Childhood/adolescent onset continuing into adulthood; personal distress may emerge late.
- Not due to brain disease or another psychiatric disorder.
- Categories: paranoid, schizoid, dissocial, emotionally unstable (impulsive/borderline), histrionic, anankastic, anxious-avoidant, dependent; other/unspecified.
- CDDG usually asks ≥3 listed traits for most subtypes; research criteria use their own thresholds. Do not mix manuals.

ICD-11 · CDDR 2024

6D10: personality disorder.
- Self-functioning and/or interpersonal dysfunction.
- Maladaptive cognition, emotion and behaviour across situations.
- Extended duration (e.g. ≥2 years), distress/impairment and exclusions.
- Rate mild / moderate / severe (or severity unspecified).
- Add any relevant trait domains; optional borderline pattern.
- Personality difficulty (QE50.7) is below disorder threshold and is NOT a mental disorder.
- No DSM clusters; no one-to-one conversion from old categories.

DSM-5-TR · 2022

Section II: 10 categorical disorders in clusters A, B and C.
- Cultural deviation in ≥2: cognition, affectivity, interpersonal function, impulse control.
- Inflexible/pervasive; distress or impairment; stable, long duration, onset by adolescence/early adulthood.
- Exclude other disorders, substances and medical causes.

Section III: alternative model combines impaired personality functioning with pathological traits; six named types plus trait-specified disorder.
Its trait model is NOT identical to ICD-11.

ICD-11 severity: judge breadth and degree of dysfunction

Mild · 6D10.0	Some self-functioning problems; relationships/roles partly preserved. Limited or milder impairment; substantial harm is not typical.
Moderate · 6D10.1	Multiple self-functioning areas affected; marked problems in most relationships/roles, some functioning preserved. Harm may occur.
Severe · 6D10.2	Severe self dysfunction; virtually all relationships and most life areas affected. Harm is often present, but is not a mandatory gate.

Five ICD-11 trait domains · any combination, not new "types"

Negative affectivity	Frequent/intense negative emotions, vulnerability and poor self-regard.
Detachment	Social/interpersonal distance and restricted emotional experience/expression.
Dissociality	Self-centredness and disregard for others' feelings/rights; low empathy.
Disinhibition	Rash, distractible, irresponsible acts without weighing consequences.
Anankastia	Rigid perfectionism and control; not obsessive–compulsive disorder.

What changed—and what did not

ICD-11 replaces named PD types with severity + traits; retains a borderline-pattern qualifier (not a sixth trait). Schizotypal disorder remains in the psychosis grouping. ICD-11 adopted 2019; health-reporting basis from Jan 2022; CDDR published 2024. National implementation varies. DSM-5-TR retains DSM-5's categorical architecture.

Sources: WHO ICD-10 CDDG (1992), F60 pp157–162; WHO ICD-11 CDDR (2024), pp553–569; DSM-5-TR (2022), pp733–777 and Section III alternative model. WHO implementation history. Original condensed comparison; subtype thresholds on cluster sheets are DSM-5-TR, not ICD-10.

Q74 · Personality-disorders classification supplement · 17 Sep 2026 A4 · 300 dpi

Personality disorders ICD10 ICD11 DSM5TR. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-74.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 74.2

Cluster A personality disorders

DSM-5-TR · paranoid, schizoid and schizotypal · individual criteria, course and care

Apply the general personality-disorder criteria first

Longstanding, pervasive impairment across contexts; interpret culture and developmental history. Exclude psychotic/mood episodes, substances and acquired causes; distinguish autism where relevant. Counts below are DSM-5-TR. ICD-10/11 place schizotypal disorder in the psychosis grouping, not among PD types.

Paranoid · ≥4 of 7 · persistent unjustified distrust

1 Unfounded fear of exploitation, harm or deceit.
2 Unjustified doubts about others' loyalty.
3 Reluctance to confide for fear of misuse.
4 Threatening meanings read into neutral events.
5 Persistent grudges.
6 Perceived attacks on reputation → anger/counterattack.
7 Unjustified suspicion of partner's infidelity.
Assess actual threats/discrimination; mistrust alone is not illness.

Care / prognosis
Transparent, consistent alliance; collaborative cognitive work on interpretations and interpersonal patterns. Avoid confrontational "proof".
Schema therapy: mixed-PD trial evidence; paranoid subgroup small.
Longitudinal treatment data are limited; do not assume inevitable psychosis.

Schizoid · ≥4 of 7 · detachment + restricted emotional expression

1 Little wish for/enjoyment of close relationships.
2 Usually chooses solitary activities.
3 Little interest in sex with another person.
4 Few pleasurable activities.
5 No close friends/confidants except first-degree relatives.
6 Appears indifferent to praise/criticism.
7 Emotional coldness, detachment or flattened affect.
Solitude or asexuality alone does not establish this disorder.

Care / prognosis
Respect preferred interpersonal distance. Agree functional goals; supportive work and social skills if wanted. Treat comorbidity.
No established disorder-specific treatment from robust trials.
Detachment can persist; outcome evidence is sparse, so avoid categorical predictions.

Schizotypal · ≥5 of 9 · eccentricity + cognitive/perceptual distortion

1 Ideas of reference, not fixed delusions of reference.
2 Culturally incongruent magical/odd beliefs.
3 Unusual perceptions, including bodily illusions.
4 Odd thinking/speech. 5 Suspiciousness/paranoia.
6 Inappropriate/constricted affect.
7 Eccentric behaviour/appearance.
8 No close friends except first-degree relatives.
9 Persistent social anxiety despite familiarity, driven by paranoid fears rather than negative self-judgment.

Care / prognosis
Supportive/cognitive work, social and vocational support; monitor emerging psychosis.
Small risperidone trials suggest symptom benefit; not routine prophylaxis or a cure.
Use a clear target and monitor harms.
Some develop a psychotic disorder; transition is not inevitable. Function may remain impaired.

Evidence and prescribing limits

No dedicated IPS cluster-A CPG found in the checked corpus; BPD guidance is not cluster-A guidance. Treatment combines limited trials and supportive practice. An antipsychotic is not indicated just by a "cluster A" label; treat established psychosis/comorbidity under its own guideline.

Sources: DSM-5-TR (2022), pp737–748; WHO ICD-11 CDDR (2024), personality/psychosis boundaries. Bamelis et al. (2014), PMID24322378 (mixed-PD schema trial); Koenigsberg et al. (2003), PMID12823075 (small schizotypal risperidone trial). DSM course descriptions; supportive-care suggestions have limited disorder-specific trial evidence.

Q74 supplement · Cluster A is DSM terminology · 17 Sep 2026 A4 · 300 dpi

Cluster A personality disorders. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-74.

Credits and references: retained within the illustration.

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.

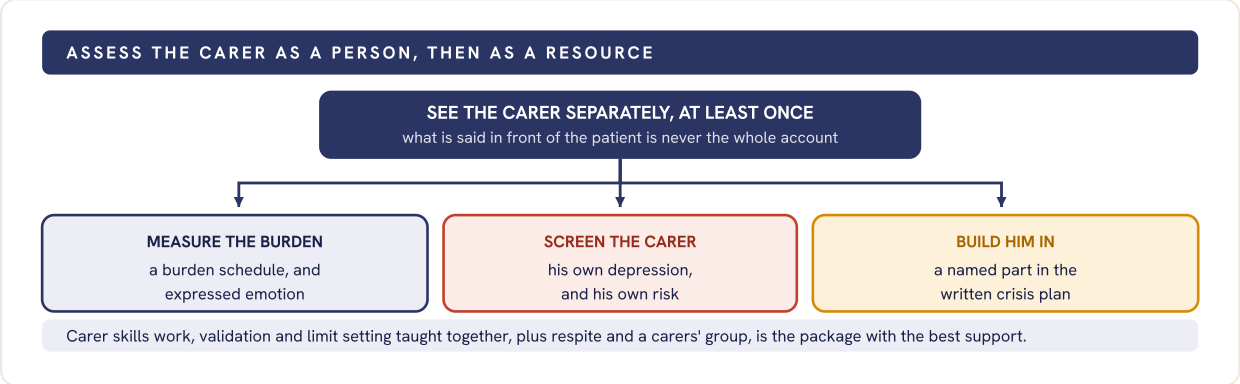


Fig 075.1 Three destinations from one interview, with the carer's own morbidity in red because it is the part most often left unasked.

- **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

WEAVE 100 · PAPER II · 76.1

Cluster B personality disorders

DSM-5-TR · four distinct diagnoses · thresholds plus general PD criteria

Diagnosis is longitudinal, not a reading of someone's "motive"

Patterns begin by early adulthood and occur across contexts; assess function, culture, comorbidity and risk. A crisis alone does not establish PD. All criteria are paraphrased; count each numbered feature once.

Antisocial · ≥3 of 7

Rights violations since age 15:
1 Repeated unlawful acts.
2 Deceit for gain/pleasure.
3 Impulsivity/poor planning.
4 Repeated fights/aggression.
5 Reckless disregard for safety.
6 Persistent irresponsibility.
7 Lack of remorse.
PLUS age ≥18 and evidence of conduct disorder before 15; not exclusively schizophrenia/bipolar disorder.
Care: NICE CG77—consider group cognitive/behavioural interventions; treat substance use/comorbidity. No routine drugs for ASPD, aggression or impulsivity.
Course: overt antisocial acts may lessen with age; functional and interpersonal problems can persist.

Borderline · ≥5 of 9

1 Frantic avoidance of abandonment.
2 Unstable ties: idealisation ↔ devaluation.
3 Unstable identity/self-image.
4 Impulsivity in ≥2 harmful domains.
5 Recurrent suicidal behaviour, gestures/threats or self-injury.
6 Reactive mood shifts, usually hours.
7 Chronic emptiness.
8 Intense anger/poor anger control.
9 Stress-related paranoia/severe dissociation.
Do not count self-harm again under 1/4.
Care: structured psychotherapy (see IPS box); assess suicide risk and use a shared crisis plan.
Course: diagnostic remission is common; social/vocational recovery often lags. Not synonymous with bipolar disorder.

Histrionic · ≥5 of 8

1 Uncomfortable outside attention.
2 Inappropriately seductive/provocative interaction.
3 Rapidly shifting, shallow emotion.
4 Appearance used to attract attention.
5 Impressionistic, detail-poor speech.
6 Theatrical/exaggerated expression.
7 Suggestibility.
8 Overestimates intimacy.
Care: consistent psychotherapy addressing relationships/emotion; schema evidence from a mixed-PD trial, small subgroup.
Course: limited outcome data; traits/function can change. No established core-drug treatment.

Narcissistic · ≥5 of 9

1 Grandiose self-importance.
2 Preoccupation with fantasies of unlimited success/power, brilliance, beauty or ideal love.
3 Belief in specialness/high-status ties.
4 Excessive need for admiration.
5 Entitlement. 6 Exploitation.
7 Lack of empathy.
8 Envy or belief others envy them.
9 Arrogance.
Care: psychotherapy targeting self-esteem regulation, empathy and relationships; evidence limited, including a small mixed-trial subgroup.
Course: variable; shame/depression after setbacks needs assessment. No proven core-drug treatment.

BPD care · Indian Psychiatric Society CPG 2023

- Psychotherapy first: dialectical behaviour, mentalisation-based, schema and transference-focused therapies; structured general psychiatric management is another option.
- Shared crisis plan; adjunctive drugs for selected targets; avoid polypharmacy/benzodiazepines.

Named supplements · medicine limits

APA 2024: medicines time-limited, target-based, adjunctive. NICE CG78: no drugs for core BPD/symptoms or medium/long-term antipsychotics. Maudsley15e (2025): weak drug evidence. No universally best psychotherapy.

Sources: DSM-5-TR (2022), pp748–764; IPS BPD CPG (2023), IJP65:221–237 (BPD scope only). NICE CG77/CG78 (2009, amended); APA BPD guideline released2024; Maudsley15e (2025), pp787–790. Bamelis2014 PMID24322378; Zanarini2010 PMID20395399. Other-PD evidence must not be inferred from BPD trials.

Q76 · Cross-links Q73/Q75 · Personality-disorders area · 17 Sep 2026 A4 · 300 dpi

Cluster B personality disorders. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-76.

Credits and references: retained within the illustration.

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.

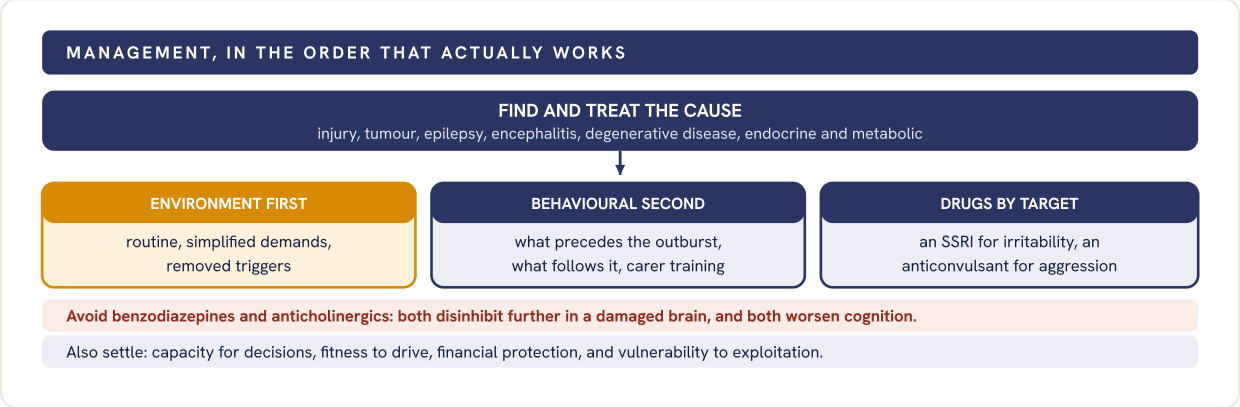


Fig 077.1 Cause, then environment, then behaviour, then drugs, with the two classes that make it worse carried as a red strip.

- **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

WEAVE 100 · PAPER II · 77.1



Organic personality change diagnosis and management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-77.

Credits and references: retained within the illustration.

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

WEAVE 100 · PAPER II · 79.1

Cluster C personality disorders

DSM-5-TR · avoidant, dependent and obsessive–compulsive personality disorder

Anxious/fearful cluster: apply general PD criteria before counting

Enduring, pervasive maladaptation and impairment; assess development and culture. Dependency imposed by illness, realistic danger, religious practice or economic necessity is not sufficient. ICD-11 uses severity and traits; it does not retain cluster C as a diagnostic category.

Avoidant · ≥4 of 7 · inadequacy and rejection sensitivity

1 Avoids interpersonal work through fear of rejection/criticism.
2 Engages only when confident of being liked.
3 Restrained intimacy through fear of shame/ridicule.
4 Preoccupied with criticism/rejection.
5 Inhibited with new people through inadequacy.
6 Sees self as inept, unappealing or inferior.
7 Avoids new activities/risks through embarrassment.
Usually wants closeness. Social anxiety can also be early, generalised and pervasive; assess both rather than rely on a simplistic boundary.

Care / prognosis
Cognitive behavioural therapy (CBT): graded exposure, behavioural experiments and social skills. Schema therapy is another supported option.
Treat comorbid social anxiety/depression when present.
Can improve with treatment; avoidance and impaired relationships may persist.
Build function, not just symptom reduction.

Dependent · ≥5 of 8 · excessive need for care and separation fears

1 Everyday decisions need excessive reassurance.
2 Others assume major responsibilities.
3 Cannot disagree for fear of losing support (not realistic threat).
4 Cannot initiate independently through low confidence.
5 Goes excessively far to obtain care/support.
6 Feels helpless alone through fear of self-care failure.
7 Urgently seeks replacement when a close relationship ends.
8 Unrealistic fear of being left to care for self.

Care / prognosis
Collaborative therapy: graded independent decisions, assertiveness and separation tolerance. Schema/CBT approaches; dedicated trials limited.
Avoid making the therapist a permanent substitute decision-maker.
Course is variable; relationship losses may precipitate depression/anxiety.
Autonomy and function are treatment outcomes.

Obsessive–compulsive PD · ≥4 of 8 · rigidity at the cost of efficiency

1 Details/rules/lists obscure the purpose.
2 Perfectionism prevents completion.
3 Work excludes leisure/friends (not economic necessity).
4 Inflexible ethics/values beyond cultural/religious norms.
5 Cannot discard worthless objects without sentimental value.
6 Delegates only if others follow the exact method.
7 Miserly spending for feared future catastrophes.
8 Rigidity/stubbornness.
Not OCD: obsessions/compulsions are not required. Both disorders can coexist.

Care / prognosis
CBT/schema work on rigid standards, uncertainty, delegation, flexibility and relationships; behavioural experiments.
Evidence is smaller than for BPD.
No established core-drug treatment; treat genuine comorbidity.
Traits can be longstanding; symptoms and function may improve.
Conscientiousness alone is not disorder.

Evidence scope

No dedicated IPS cluster-C CPG found. Avoidant CBT and mixed-PD schema trials support therapy; efficacy is not established equally for each subtype.

Sources: DSM-5-TR (2022), pp764–775; WHO ICD-11 CDDR (2024), pp553–569. Emmelkamp et al. (2006), PMID16816307 (avoidant CBT trial); Bamelis et al. (2014), PMID24322378 (schema therapy, mixed-PD sample). Weave Q79 p108: social-anxiety differential refined; individualised therapy goals include evidence-informed extrapolation.

Q79 · Cluster-C supplement · Personality-disorders area · 17 Sep 2026 A4 · 300 dpi

Cluster C personality disorders. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-79.

Credits and references: retained within the illustration.

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

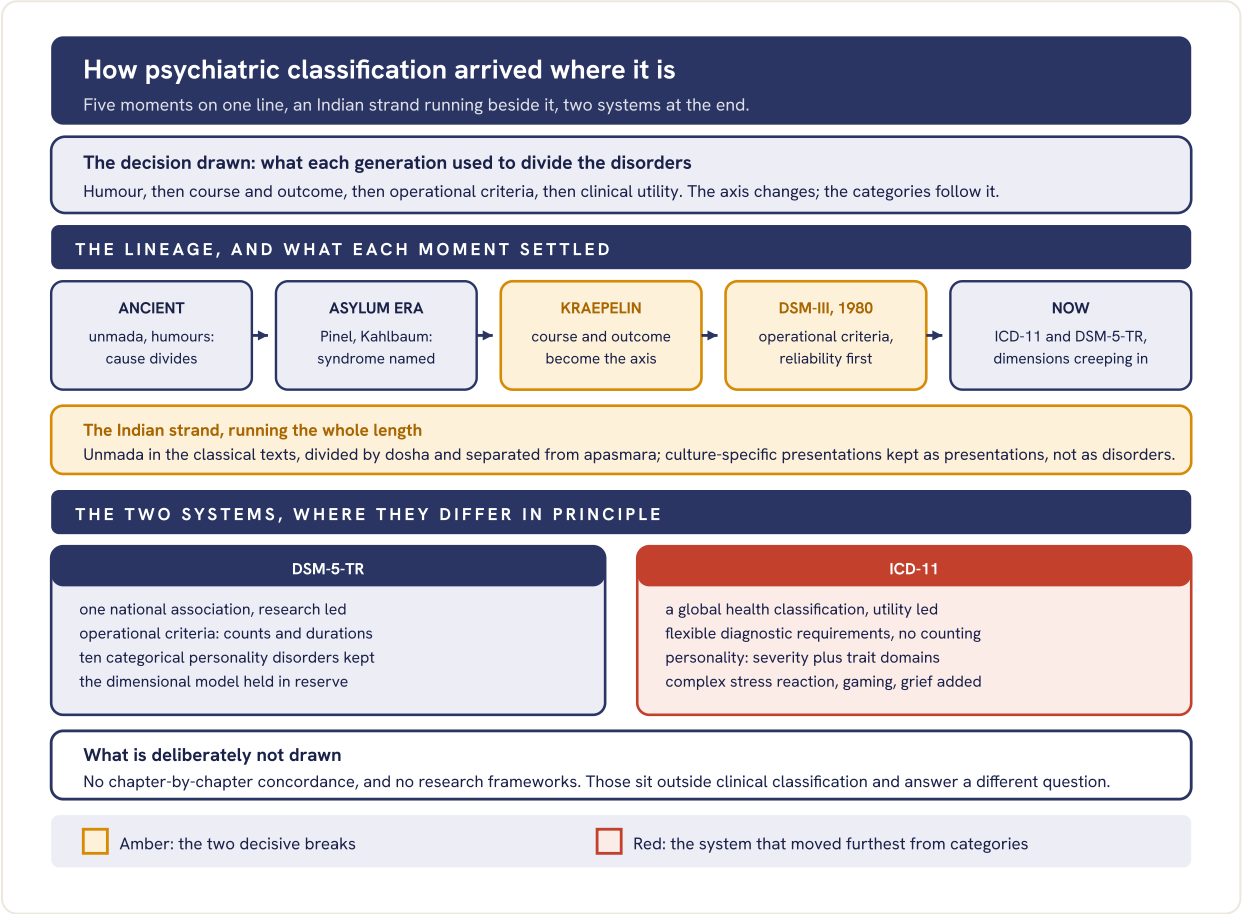


Fig 082.1 One axis, what each generation used to divide the disorders, carrying the field from cause to course to criteria to clinical utility, with the Indian strand shown as continuous rather than as an appendix.

- **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

WEAVE 100 · PAPER II · 85.1

Treatment adherence

Understand the barrier → agree a practical plan → review what changed

TERMS: RELATED, NOT INTERCHANGEABLE

Compliance	Extent to which behaviour follows prescribed instructions; often implies a clinician-directed relationship.
Adherence	Extent to which behaviour corresponds with an agreed treatment plan.
Concordance	The collaborative consultation and agreement process; not a numerical measure of doses taken.
Persistence	How long treatment is continued from initiation until discontinuation. Initiation and day-to-day implementation are separate issues.

ASK WITHOUT BLAME

"Many people miss doses. What has taking this treatment been like over the last week?" Explore benefit, concerns, side effects, cost, access and daily routine.
Intentional barriers: beliefs, stigma, adverse effects or disagreement with the plan.
Unintentional barriers: forgetting, cognitive difficulties, complex schedules or supply problems. Both can occur together.

ASSESS: TRIANGULATE, DO NOT ACCUSE

Conversation / records	Use a defined time window; review prescribed and actually taken treatment, including non-drug interventions.
Refills / pill counts	Useful clues to access and implementation, but possession or missing tablets does not prove ingestion.
Levels / other evidence	Drug concentrations help for selected medicines; consider sampling time, dose, interactions and metabolism. No single measure is a universal gold standard.

MATCH THE INTERVENTION TO THE BARRIER

Concerns / adverse effects	Shared decisions, clear benefit–risk discussion, motivational work and active side-effect management; agree a tolerable regimen.
Practical obstacles	Simplify when appropriate; link doses to routines; reminders, accessible refills and affordability support; involve carers with consent.
Repeated missed treatment	Review formulation and follow-up intensity. Offer a long-acting injectable when clinically appropriate and acceptable; it does not solve every barrier.

IPS CPG 2026: SCHIZOPHRENIA-SPECIFIC APPLICATION

Check poor adherence as a cause of apparent treatment resistance. Offer long-acting antipsychotics proactively when non-adherence is detected / after relapse, using shared decisions and considering access and cost.
Review benefit and adverse effects; respect informed choices.

Sources: IPS schizophrenia CPG (2026), adherence / LAI sections; Weave Q85, p115.
Supplement: NICE CG76 (2009), CG178 (2014). Checked 17 Sep 2026.

Weave 100+ · Paper II · Q85.1 A4 · 300 dpi

Treatment compliance and adherence. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-85.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 80.1

CT: computed tomography

Structural imaging · rapid assessment of acute intracranial pathology

PRINCIPLE → IMAGE

Rotating X-ray source → differential tissue attenuation → reconstructed slices.
Attenuation is expressed in Hounsfield units (HU): water 0; air about −1000. Bone and calcium are bright; cerebrospinal fluid (CSF) is dark. Acute blood is often hyperdense.

WHAT TO LOOK FOR

Blood / mass effect	Haemorrhage, extra-axial collections, midline shift, compressed cisterns or ventricles.
Brain / CSF spaces	Atrophy, ventricular enlargement, hydrocephalus and established infarcts; correlate with age and clinical course.
Bone / calcium	Fractures and calcification are well shown. Use appropriate brain and bone windows.

WHEN IT HELPS IN PSYCHIATRY

- Acute altered behaviour with head injury, focal neurological signs, reduced consciousness or suspected haemorrhage: urgent medical assessment and targeted imaging.
- New atypical neuropsychiatric syndrome: assess for a structural cause; MRI may be preferable when clinically feasible.
- Cognitive decline: identify structural contributors. An atrophy pattern alone does not establish the dementia subtype.

STRENGTHS, RISKS AND CHOICE

Strengths	Fast, widely available and less sensitive to motion than MRI; useful when MRI cannot be performed.
Limitations	Ionising radiation; poorer soft-tissue detail than MRI. A normal CT does not exclude early ischaemia, encephalitis or epilepsy.
Contrast	Non-contrast CT is typical for acute haemorrhage. Iodinated contrast is indication-specific; assess previous reactions and renal risk.

EXAM / CLINICAL PEARL

Choose the test to answer a clinical question. CT does not diagnose schizophrenia, depression or bipolar disorder from a characteristic scan.
IPS schizophrenia CPG (2026): reserve neuroimaging for suspected neurological pathology / specific clinical indications. NICE TA136 (2008) also advises against routine imaging in uncomplicated first-episode psychosis.
CT = rapid structural screen; MRI = greater tissue detail.

Sources: IPS schizophrenia CPG (2026); ACR/RSNA Head CT; NICE TA136 (2008), NG97 (2018).
Weave Q80 neuroimaging supplement; educational synthesis, checked 17 Sep 2026.

Weave 100+ · Paper II · Q80.1 A4 · 300 dpi

CT. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical assessment and cross-cutting nosology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 80.2

MRI: magnetic resonance imaging

Structural detail · sequences answer different clinical questions

PRINCIPLE → IMAGE

Hydrogen nuclei in a strong magnetic field → radiofrequency excitation → relaxation signals, spatially encoded by gradients. Tissue contrast depends on the sequence.
MRI uses no ionising radiation. A structural MRI is different from functional MRI (fMRI).

CORE SEQUENCES

T1-weighted	Anatomy and tissue volumes; CSF dark, white matter relatively bright. Useful for atrophy and structural assessment.
T2-weighted	Water / oedema usually bright; useful for many pathological lesions.
FLAIR	Fluid-attenuated inversion recovery: CSF suppression highlights periventricular and other parenchymal lesions.
DWI + ADC	Diffusion-weighted imaging + apparent diffusion coefficient. Bright DWI with low ADC suggests restricted diffusion; not stroke-specific.
SWI / T2*	Susceptibility-weighted imaging / T2*: blood products and microbleeds; interpret the complete study.

CLINICAL INDICATIONS

- Focal signs, seizures, atypical or progressive cognitive / personality change, or suspected tumour, inflammatory disease or vascular pathology.
- Dementia assessment: structural causes and patterns of atrophy / vascular injury; normal imaging does not exclude neurodegeneration.
- Suspected encephalitis requires urgent clinical investigation; a normal MRI does not exclude autoimmune encephalitis.

SAFETY AND PRACTICAL LIMITS

Screen every implant and possible metallic foreign body: MR-conditional devices require their specified conditions, not a blanket prohibition.
Claustrophobia, noise, motion and examination length affect feasibility.
Gadolinium is not needed for every scan; assess indication, renal status and pregnancy. Contrast safety differs from CT iodinated contrast.

INTERPRETATION PEARL

Read the sequence and clinical context before calling an abnormality. Incidental findings are common and may not explain symptoms.
IPS schizophrenia CPG (2026): imaging is selective for suspected neurological pathology / specific indications.
Group volume differences are not individual diagnostic tests.

Sources: IPS schizophrenia CPG (2026); ACR/RSNA Brain MRI; NICE NG97 (2018), TA136 (2008).
Weave Q80 structural-imaging supplement; checked 17 Sep 2026.

Weave 100+ · Paper II · Q80.2 A4 · 300 dpi

MRI. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical assessment and cross-cutting nosology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 80.3

fMRI: functional MRI

Blood-oxygen-level-dependent signal · an indirect index of neural activity

NEUROVASCULAR COUPLING → BOLD

Neural activity → altered local blood flow and oxygenation → change in deoxyhaemoglobin → T2*-weighted signal change.
The haemodynamic response is delayed and unfolds over seconds. BOLD reflects vascular physiology as well as neural activity; it does not directly record neuronal firing.

TWO COMMON APPROACHES

Task-based	Compare defined tasks / conditions, e.g. language, motor or emotional processing. Results depend on task performance and the contrast chosen.
Resting-state	Correlations in spontaneous BOLD fluctuations estimate functional connectivity. Correlation does not demonstrate a direct anatomical connection or causation.
Networks	Default mode, salience and executive-control networks are common research targets; network boundaries depend on methods.

APPLICATIONS

- Selected presurgical mapping of language / motor functions alongside other clinical information; specialist interpretation is essential.
- Research examples: schizophrenia—prefrontal / salience dysfunction; depression—altered emotion regulation; OCD—cortico-striatal circuits; PTSD—threat / extinction; addiction—reward / control. Findings vary across studies.
- Connectivity-informed stimulation targeting and real-time neurofeedback are developing uses; evidence and availability vary by protocol.
- Longitudinal studies can examine treatment-associated change; a changed scan is not by itself proof of clinical benefit.

WHY RESULTS NEED CAUTION

Confounding	Head motion, respiration, vascular disease, medication, arousal and task performance can change BOLD.
Analysis choices	Preprocessing, multiple comparisons, sample size and reproducibility affect conclusions.
Reverse inference	Activation in a region does not uniquely identify a mental process: most regions contribute to multiple functions.

EXAM PEARL: GROUP EFFECT ≠ INDIVIDUAL TEST

A statistically different average activation pattern in depression, schizophrenia or OCD does not establish a clinically validated diagnostic test for one person.
Spatial detail is generally better than scalp EEG; timing is slower because of the haemodynamic response.
Apply standard MRI safety screening. Most BOLD studies do not require an injected contrast agent.

Sources: Weave Q80, p110; ACR/RSNA RadiologyInfo, Functional MRI;
Companion to Psychiatric Studies, neuroimaging chapter / local map. Checked 17 Sep 2026.

Weave 100+ · Paper II · Q80.3 A4 · 300 dpi

fMRI. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical assessment and cross-cutting nosology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 80.4

PET: positron emission tomography

Radiotracer imaging · metabolism or molecular targets, depending on tracer

PRINCIPLE → IMAGE

A positron-emitting radionuclide decays; positron–electron annihilation usually produces two 511-keV photons travelling in approximately opposite directions. Coincidence detection reconstructs tracer distribution.
PET/CT or PET/MRI combines molecular information with anatomy.

THE TRACER DETERMINES THE QUESTION

18F-FDG	Fluorodeoxyglucose: glucose analogue indexing regional glucose utilisation. Supports selected dementia differential diagnoses and epilepsy presurgical evaluation.
Amyloid tracers	Estimate cerebral amyloid plaque burden in selected specialist pathways. Positivity alone does not establish the clinical syndrome or prove its sole cause.
Tau tracers	Assess aspects of tau pathology; indications, validation and availability depend on tracer and jurisdiction.
Receptor ligands	e.g. dopamine receptor / transporter studies. Mainly specialist research in psychiatry; binding depends on multiple biological and methodological factors.

DEMENTIA: PATTERNS SUPPORT THE CLINICAL ASSESSMENT

FDG patterns can assist when subtype remains uncertain and the result would change management. Typical associations include temporoparietal / posterior cingulate involvement in Alzheimer disease and frontal / anterior temporal involvement in frontotemporal dementia.
Patterns overlap. Interpret with history, examination, cognitive testing and structural imaging; do not diagnose from a coloured map alone.

PREPARATION AND LIMITATIONS

- Ionising radiation from the tracer, with additional exposure if CT is used. Pregnancy / breastfeeding require tracer-specific specialist advice.
- For brain FDG studies, glucose level, fasting, uptake conditions, medication and movement can influence results; follow the department protocol.
- Cost, tracer supply, access and limited specificity constrain use. PET is not a routine diagnostic test for depression, ADHD or schizophrenia.

PET VERSUS SPECT

PET detects paired annihilation photons; SPECT detects individual emitted gamma photons through collimation. PET generally offers better sensitivity and spatial resolution, but performance depends on equipment and protocol. FDG-PET measures a metabolic correlate; BOLD fMRI measures a haemodynamic correlate. Neither directly reads thoughts.

Sources: ACR/RSNA RadiologyInfo, PET/CT and Seizures & Epilepsy; NICE NG97.
Weave Q80 molecular-imaging supplement; checked 17 Sep 2026.

Weave 100+ · Paper II · Q80.4 A4 · 300 dpi

PET. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical assessment and cross-cutting nosology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 80.5

SPECT: single-photon emission CT

Gamma-emitting tracers · perfusion and selected molecular targets

PRINCIPLE → IMAGE

An administered radiotracer emits gamma photons. Rotating gamma cameras with collimators acquire projections; reconstruction gives a three-dimensional distribution.
The signal reflects the chosen tracer's distribution. SPECT/CT can add anatomical localisation and attenuation correction.

DISTINGUISH THE MAIN BRAIN STUDIES

Perfusion SPECT	99mTc-HMPAO or 99mTc-ECD: regional perfusion around the uptake period. An indirect physiological measure, not a structural brain scan.
Dopamine transporter	123I-FP-CIT (DaTscan): presynaptic striatal dopaminergic terminals. Helps selected differential diagnoses; it is not a general psychiatric scan.
Epilepsy study	Ictal perfusion may localise hyperperfusion; injection timing is crucial. Interictal / ictal comparison can aid specialist presurgical assessment.
Dementia work-up	Perfusion SPECT can support selected uncertain dementia subtypes. DAT-SPECT can support suspected dementia with Lewy bodies when diagnosis is uncertain.

WHAT A DAT SCAN CANNOT TELL YOU

Reduced striatal uptake supports presynaptic dopaminergic deficit in an appropriate clinical setting. It does not reliably distinguish Parkinson disease from all other degenerative parkinsonian syndromes.
A normal scan alone must not rule out dementia with Lewy bodies. Medication interference and technical factors require review.

ADVANTAGES AND LIMITS

- Often more accessible than PET, although access varies. Spatial resolution and sensitivity are generally lower than PET.
- Ionising radiation; use pregnancy / breastfeeding precautions specific to the radiopharmaceutical. Motion, attenuation and reconstruction can create artefacts.
- Perfusion changes are not disease-specific. Routine SPECT "brain typing" for ADHD, mood disorders or personality diagnosis is not an established clinical use.

EXAM PEARL

SPECT ≠ PET: single gamma photon detection versus paired annihilation-photon detection.
Perfusion SPECT ≠ DAT-SPECT: different tracers, different biological questions.
A useful scan resolves a defined differential diagnosis and can change care; an abnormal image alone is not a diagnosis.

Sources: NICE NG97; ACR/RSNA RadiologyInfo, Seizures & Epilepsy.
Weave Q80 functional-imaging supplement; checked 17 Sep 2026.

Weave 100+ · Paper II · Q80.5 A4 · 300 dpi

SPECT. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical assessment and cross-cutting nosology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 81.1

EEG: electroencephalography

Electrical activity · excellent timing, limited anatomical localisation

WHAT THE SCALP ELECTRODES RECORD

Voltage differences produced mainly by synchronised postsynaptic activity of cortical neuronal populations.
Standard placement uses the international 10–20 system.
Assess background, symmetry, reactivity, state, focal abnormalities, epileptiform discharges and artefacts.

RHYTHMS: APPROXIMATE CONVENTIONAL BANDS

Delta / theta	Delta <4 Hz; theta approximately 4–<8 Hz. Normal in appropriate sleep / age contexts; excess awake slowing needs interpretation.
Alpha / beta	Alpha approximately 8–13 Hz, usually posterior with eyes closed and attenuating on eye opening. Beta >13–30 Hz; may increase with benzodiazepines.
Gamma	>30 Hz. High-frequency scalp signals are readily contaminated by muscle artefact; not a routine psychiatric diagnostic marker.

HIGH-YIELD CLINICAL ASSOCIATIONS

Diffuse slowing	Supports diffuse cerebral dysfunction, including delirium / encephalopathy; does not identify a single cause.
Focal slowing / spikes	Focal dysfunction / epileptiform activity respectively. Interpret alongside the history and neurological assessment.
Urgent EEG	Consider non-convulsive seizures / status with unexplained altered awareness; continuous EEG may be required.
Special patterns	Periodic sharp-wave complexes can support CJD but are not diagnostic. Triphasic morphology is not specific to hepatic encephalopathy.

USES AND IMPORTANT LIMITS

- Routine EEG can support epilepsy diagnosis; a normal interictal EEG does NOT exclude epilepsy.
- Functional seizures: capture a typical event on video-EEG with expert clinical interpretation. Some epileptic seizures have no clear scalp correlate; the conditions can coexist.
- Electroconvulsive therapy (ECT) seizure monitoring and sleep studies are other uses. P300 / mismatch negativity and many quantitative EEG findings remain mainly research tools.

DO NOT TURN AN ASSOCIATION INTO A DIAGNOSIS

Extreme delta brush can support anti-NMDA receptor encephalitis, but is neither necessary nor sufficient for diagnosis.
Medication, drowsiness, eye movements and muscle activity alter recordings. EEG is not a stand-alone test for schizophrenia, depression or ADHD.
Temporal resolution: milliseconds. Localisation is constrained by volume conduction and the inverse problem.

Sources: Weave Q81, p111; NICE NG217 (EEG must not exclude epilepsy);
Stern, Atlas of EEG Patterns, 2e / local EEG clinical map. Checked 17 Sep 2026.

Weave 100+ · Paper II · Q81.1 A4 · 300 dpi

EEG. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical assessment and cross-cutting nosology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 82.1

Categorical versus dimensional

Two ways to represent psychopathology · often combined in clinical practice

THE CORE DISTINCTION

Categorical: does this presentation meet the definition of a disorder?
Dimensional: how much of a symptom, trait or functional impairment is present?
“Classification” is the broader activity: either approach can be classificatory.

COMPARE THE APPROACHES

Representation	CATEGORY: present / absent label. DIMENSION: score or profile across a continuum.
Boundary	CATEGORY: a diagnostic threshold. DIMENSION: graded variation; clinical action may still require a threshold.
Strength	CATEGORY: shared language, coding, service decisions and pragmatic treatment pathways. DIMENSION: preserves severity and subthreshold variation; tracks change.
Main difficulty	CATEGORY: within-label heterogeneity, overlapping labels and threshold effects. DIMENSION: measurement burden and actionable cut-offs.
Example	CATEGORY: a depressive episode meeting diagnostic requirements. DIMENSION: depressive symptom severity over time, with functioning assessed separately.

WHY “COMORBIDITY” LOOKS DIFFERENT

Several categorical diagnoses may describe one person. Dimensional models can represent shared vulnerability and overlapping symptom domains without requiring every pattern to be a separate disease.
However, correlation between symptoms does not prove a single cause; clinically distinct conditions can also coexist.

ACTUAL SYSTEMS ARE HYBRIDS

ICD-11 retains many disorder categories while using severity, course and symptom qualifiers. Personality disorder is organised around severity and optional trait-domain qualifiers.
DSM-5-TR retains categories alongside specifiers and dimensional measures; its alternative personality-disorder model combines impaired functioning with pathological traits.

EXAM CONCLUSION

Use categories for clear communication and decisions; use dimensions to describe severity, heterogeneity and change. Reliability, validity and clinical utility must each be tested—none follows automatically from choosing a model.
HiTOP and RDoC extend dimensional thinking in different ways (see Q82.3).

Sources: Weave Q82, p112; WHO ICD-11 CDDR (2024); APA DSM-5-TR (2022).
User-confirmed topic: categorical versus dimensional. Checked 17 Sep 2026.

Weave 100+ · Paper II · Q82.1 A4 · 300 dpi

Categorical versus dimensional. Read across the labelled contrasts to distinguish the related concepts. Section companion: Clinical assessment and cross-cutting nosology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 82.2



Changes in ICD11. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical assessment and cross-cutting nosology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 82.3

HiTOP and RDoC

Dimensional approaches · different starting points and different purposes

HiTOP

Hierarchical Taxonomy of Psychopathology
Organises patterns of co-occurring symptoms and traits into dimensions at different levels of breadth.
Starting point: observed psychopathology.

RDoC

Research Domain Criteria
NIMH research framework linking functions and mechanisms across multiple units of analysis.
Starting point: basic biobehavioural functions.

HiTOP: A HIERARCHY OF PHENOTYPES

Broad dimensions / superspectra → spectra → narrower subfactors → syndromes → components / traits → symptoms.
Representative spectra include internalizing, thought disorder, detachment, disinhibited externalizing and antagonistic externalizing. The model is evolving; this is a simplified teaching hierarchy.

HiTOP: WHAT IT ADDS

A graded profile can represent both shared liability and specific problems, addressing overlap and heterogeneity in categorical diagnoses.
Example: assess broad internalizing distress, then distinguish narrower fear and distress features.
Association-based structure does not, by itself, identify a biological cause.

RDoC: SIX DOMAINS AND EXAMPLE CONSTRUCTS

Negative valence	Threat and loss.
Positive valence	Reward responsiveness, learning and valuation.
Cognitive systems	Attention, perception, memory, language and cognitive control.
Social processes	Affiliation, social communication and perception of self / others.
Arousal / regulatory	Arousal, circadian rhythms and sleep–wakefulness.
Sensorimotor	Motor actions, agency and ownership; habitual sensorimotor patterns.

RDoC: THE MATRIX

Constructs × units of analysis: genes, molecules, cells, circuits, physiology, behaviour and self-report; experimental paradigms probe these functions.
Study variation from typical to impaired functioning, within developmental and environmental contexts.

HOW TO COMPARE THEM IN AN EXAM

HiTOP = empirical organisation of psychopathology; RDoC = integrative research into functions and mechanisms. They can inform each other.
RDoC is not a clinical diagnostic manual. Neither currently replaces ICD / DSM coding in routine care.

Sources: HiTOP Consortium / Society framework; Kotov et al. (2017), HiTOP;
NIMH About RDoC, domain definitions and FAQ. Weave Q82 supplement; checked 17 Sep 2026.

Weave 100+ · Paper II · Q82.3 A4 · 300 dpi

HiTOP and RDoC. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical assessment and cross-cutting nosology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 85.2

LAI antipsychotics: formulation matters

LAI = long-acting injectable · Aporia 02 · adult schizophrenia · checked 17 Sep 2026

Establish oral tolerability first. Dose examples / labelled options below are not full conversion protocols.
IM = intramuscular; SC = subcutaneous; q = every; wk = week. Availability and licences vary by country.

MOLECULE / FORMULATION	MAINTENANCE DOSE / INTERVAL	START / ORAL COVER / DISTINCTIVE POINT
Fluphenazine decanoate	Example: 25 mg IM q2–3 wk	Individualise test / starting dose and conversion. Oral cover is not a universal fixed-duration rule.
Haloperidol decanoate	Typical: 50–200 mg IM q4 wk	Convert from stable oral dose; initial injection ≤100 mg. Additional oral treatment depends on the transition plan.
Flupentixol decanoate	Example: 20–40 mg IM q2–4 wk	Test 20 mg where indicated; taper oral treatment over several days. Higher-dose regimens exist.
Zuclopenthixol decanoate	Usual: 200–500 mg IM q1–4 wk	Test / titrate per label; individualise oral taper. Decanoate ≠ acetate (Acuphase).
Risperidone microspheres Consta	25 / 37.5 / 50 mg IM q2 wk	Continue oral antipsychotic for 3 weeks. Delayed main release; refrigerated storage.
Risperidone Rykindo	25 / 37.5 / 50 mg IM q2 wk	7 days of oral risperidone with first dose. Distinct from Consta and monthly ISM.
Risperidone ISM Okedi / Risvan	75 / 100 mg IM q28 days	No loading or oral supplementation after injection; prior oral response / tolerability required.
Risperidone SC Perseris — historical*	90 / 120 mg SC monthly	No loading / oral overlap. US supply ended 31 May 2026; residual stock may remain.
Risperidone SC Uzedy	50–125 mg SC monthly or 100–250 mg q2 months	No loading or oral supplementation. These are schizophrenia options; bipolar-I dosing differs.
Paliperidone palmitate PP1M — Xeplion / Sustenna	25–150 mg equivalent IM monthly (EU label units)	Start 150 mg eq day 1 + 100 mg eq day 8, both deltoid. No routine oral overlap; renal adjustment matters.
Paliperidone palmitate PP3M — Trevicta / Trinza	175 / 263 / 350 / 525 mg eq IM q3 months	Use after ≥4 months of adequate PP1M treatment; last two doses the same. Follow the named product label.
Paliperidone palmitate PP6M — Byannli / Hafyera	700 / 1000 mg eq IM q6 months; gluteal only	After qualifying PP1M (≥4 months) or PP3M (≥1 cycle); only specified preceding doses qualify.
Aripiprazole monohydrate Maintena	400 mg IM monthly 300 mg if indicated	14 days oral cover OR label-specific 1-day start (two injections + oral dose); check interactions.
Aripiprazole 2-monthly Asimtufii	960 mg IM q2 months 720 mg if indicated; gluteal	14-day or label-specific 1-day initiation; can replace scheduled Maintena. Protocol differs from Maintena.
Aripiprazole lauroxil Aristada	441 / 662 / 882 mg monthly; 882 mg q6 wk; 1064 mg q2 mo	21 days oral aripiprazole OR Initio 675 mg + oral 30 mg with initiation. Initio is NOT maintenance.
Olanzapine pamoate Zypadhera / Relprevv	150 / 210 / 300 mg IM q2 wk or 300 / 405 mg q4 wk	Gluteal only; no routine oral overlap. Observe ≥3 hours after EVERY dose for delirium / sedation.

UNITS Paliperidone mg eq ≠ palmitate salt mg: 150 eq = 234 salt; 100 eq = 156 salt. Use the named product's label.

MONITOR Weight / metabolic parameters; movement symptoms; prolactin symptoms; ECG when indicated. Aripiprazole: akathisia / impulse-control problems. Olanzapine: metabolic burden + post-injection syndrome.

PRACTICE IPS 2026: offer LAIs proactively for schizophrenia after relapse / detected non-adherence, using shared decisions. Missed doses, renal impairment and interactions require product-specific instructions.

SCOPE All Aporia 02 formulation families are represented. Other regional / older depots include pipotiazine, fluspirilene, perphenazine and bromperidol; not an Indian availability or exhaustive historical catalogue.

Sources: Ra, Aporia 02; Maudsley 15e (2025); IPS schizophrenia CPG (2026); current FDA / EMA / SmPC labels.
Perseris status: Indivior 2026 notice. Exact links, corrections and dose qualifications: accompanying source ledger.

Weave 100+ · Paper II · Q85.2 · A4 / 300 dpi

LAI antipsychotics. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical assessment and cross-cutting nosology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 85.3

Equivalent doses

ANTIPSYCHOTICS + BENZODIAZEPINES

Oral doses only • Approximate comparisons, not starting doses, target doses or switching instructions.

ANTIPSYCHOTICS

Olanzapine 20 mg/day

Gardner et al. (2010) • Delphi consensus

DRUG	mg/day
Olanzapine — reference	**20**
Amisulpride	700
Aripiprazole	30
Chlorpromazine	600
Clozapine*	400
Flupentixol	10
Fluphenazine	12
Haloperidol	10
Paliperidone	9
Quetiapine	750
Risperidone	6
Sulpiride	800
Trifluoperazine	20
Ziprasidone	160

BENZODIAZEPINES

Diazepam 10 mg

NHS SPS • updated 2 July 2025

DRUG	mg	ACTION
Diazepam — reference	**10**	**L**
Alprazolam	0.5	S
Chlordiazepoxide	25	L
Clobazam	20	L
Clonazepam	0.5	L
Flurazepam	15–30	L
Loprazolam	1–2	S
Lorazepam	1	S
Lormetazepam	1–2	S
Nitrazepam	10	L
Oxazepam	20	S
Temazepam	20	S

S = shorter acting; L = longer acting (SPS grouping; individual variation).

Source doses for diazepam 5 mg are doubled here to a 10 mg reference.

WHAT THE AP COLUMN MEANS

Median estimates for established schizophrenia in a non-refractory adult case. One consensus method throughout; minimum-effective-dose, mean-dose and defined daily dose (DDD) methods give different values.

WHAT THE BENZO COLUMN MEANS

Compare total oral amounts over the same period. Duration, dosing frequency and indication still differ. Other tables may give different estimates; ranges here are retained from SPS.

READ BEFORE USING AN EQUIVALENT

- Potency equivalence does not imply equal efficacy, sedation or adverse effects.
- Individualise for indication, age, organ function, interactions and prior response.
- Benzodiazepine cross-tolerance may be incomplete; abrupt changes risk withdrawal.
- Long-acting benzodiazepines may accumulate, especially with liver impairment.
- *Clozapine requires its own initiation / re-titration rules; do not start at 400 mg.
- These oral figures cannot be used to convert LAI injection amounts.

COVERAGE Selected established antipsychotics plus all 12 benzodiazepines in the SPS table (including diazepam). Newer APs absent from Gardner 2010 are intentionally not assigned mixed-method values. Z-drugs and injectables are excluded.

SOURCES Gardner et al., Am J Psychiatry 2010;167:686–693, Table 1 (expert consensus).
NHS Specialist Pharmacy Service, Oral benzodiazepines and choosing equivalent doses (2 Jul 2025).
Cross-check: Maudsley Prescribing Guidelines, 15e (2025), pp 14–16 and 466–467, Tables 1.4–1.5 / 3.24.
Transition caution: ASAM-led joint benzodiazepine tapering guideline (2025). Exact links: evidence ledger.

Weave 100+ · Paper II · Q85.3 supplement / cross-reference Q90 · A4 / 300 dpi

Antipsychotic and benzodiazepine equivalent doses. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical assessment and cross-cutting nosology.

Credits and references: retained within the illustration.

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

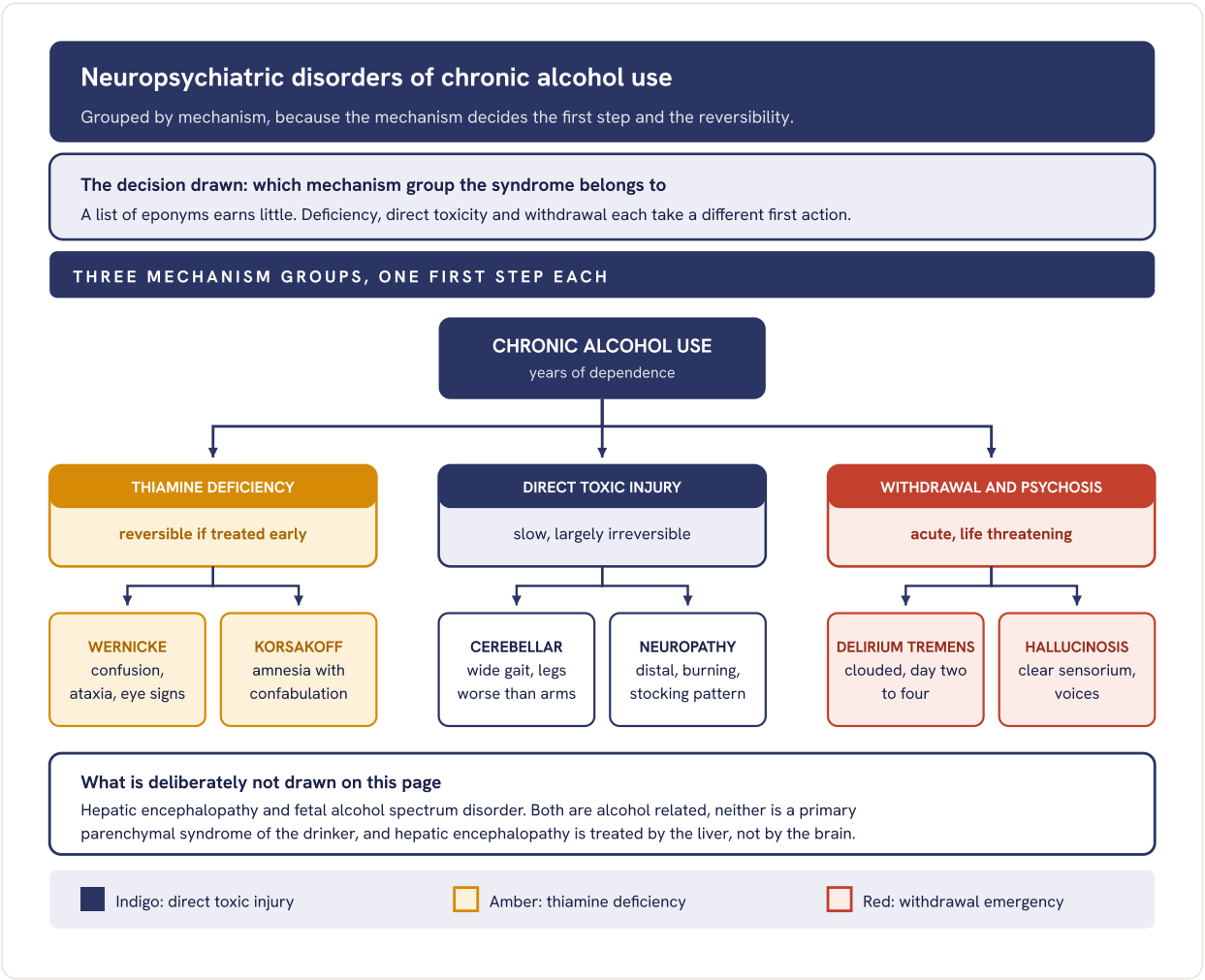


Fig 086.1 The syndromes sorted by mechanism, with the two reversible amber leaves kept together because they share one treatment.

- **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

WEAVE 100 · PAPER II · 86.1

86.1 ALCOHOL: EFFECTS → TREATMENT

Area 12 • Question 86 | Companion to 87.1 | Major complications + five typologies

ALCOHOL USE — WITH OR WITHOUT DEPENDENCE

1 ACUTE USE / INTOXICATION

- Disinhibition, impaired judgement, slurred speech, ataxia → falls / injury.
- Blackout = failed memory formation, not necessarily unconsciousness.
- Severe poisoning → coma, aspiration, respiratory depression, hypoglycaemia.
- Hangover ≠ dependence withdrawal.

2 REDUCTION / CESSATION

- After sustained heavy use: tremor, sweating, anxiety, insomnia → withdrawal.
- Seizures: usually 8–48 hours.
- Delirium tremens: often 48–96 hours; fluctuating inattention + autonomic arousal.
- Hallucinosis: relatively clear awareness; may also occur during use.

3 REPEATED / PRENATAL EXPOSURE

- Thiamine deficiency → Wernicke / Korsakoff. Toxic–nutritional injury → neuropathy, cerebellar / cognitive impairment.
- Liver / pancreatic / cardiac disease, cancer, mood / sleep / sexual problems.
- Pregnancy → fetal alcohol spectrum harm; injury and social harm at any stage.

FIRST ASSESS: airway, breathing, circulation, glucose, injury, co-use, vital signs and mental state.

Indian Psychiatric Society intoxication guideline • 2023: stabilise first; assess nutrition and give thiamine when indicated.

UNSTABLE / POISONED? → EMERGENCY CARE

- YES → protect airway; support breathing; monitor consciousness, glucose, electrolytes and heart rhythm. Exclude head injury and other causes of coma.
- Hypoglycaemia → glucose immediately; give thiamine promptly alongside / before if available. Never delay emergency glucose.
- NO → assess withdrawal risk, nutrition, psychiatric risk and dependence; choose supervised care setting.

WITHDRAWAL? → SEVERITY-BASED CARE

- Seizure / delirium / major illness / unsafe home → admission; severe cases need high-acuity monitoring.
- Benzodiazepines first-line: diazepam or chlordiazepoxide; lorazepam often preferred with significant liver disease. Titrate by monitored protocol.
- Seizure → parenteral benzodiazepine. Delirium → frequent clinical monitoring; symptom scales requiring self-report are unreliable.

WERNICKE SUSPECTED? → TREAT NOW

- Confusion, gait or eye signs + nutritional risk: do not wait for the full triad or imaging.
- UK guidance: thiamine 300–500 mg intravenously three times daily for 3–5 days; review daily. If still symptomatic, 300–500 mg daily for a further 3–5 days and while improving. Then oral replacement.
- Correct magnesium deficiency, nutrition and other electrolyte deficits; monitor refeeding risk.

KORSAKOFF / OTHER COMPLICATIONS

- Persistent amnesia → treat any active deficiency, support abstinence, cognitive rehabilitation, memory aids and supported living. No assured reversal.
- Neuropathy / ataxia → nutrition + rehabilitation; exclude other causes. Cognition may recover.
- Psychosis → exclude delirium; withdrawal care ± antipsychotic.
- Hepatic encephalopathy → correct triggers; lactulose ± rifaximin.

AFTER STABILISATION → CONTINUING DEPENDENCE TREATMENT → REVIEW

- Agree goals + motivational work → cognitive behavioural relapse prevention, family support, mutual-help and social recovery. Detoxification alone is insufficient. Assess suicide risk and coexisting illness.
- Choose medication with psychosocial care (not an automatic combination):
- Naltrexone: commonly 25 mg initially → 50 mg/day. No current opioids; check liver status; avoid acute hepatitis.
- Acamprosate: commonly 666 mg three times/day. Renal / weight-based dosing; avoid severe renal impairment.
- Disulfiram: selected, informed, supervised use after withdrawal. Check contraindications; explain alcohol reaction.
- Review drinking, adherence, adverse effects and functioning → adjust treatment / intensify support after relapse.

HISTORICAL TYPOLOGIES — EXAM COMPARISON, NOT DIAGNOSTIC CRITERIA

JELLINEK	Alpha: psychological reliance; beta: physical harm without dependence; gamma: loss of control; delta: inability to abstain; epsilon: episodic bouts.
CLONINGER	Type I: typically later onset, both sexes, anxiety / guilt, environmental influence. Type II: typically early onset, historically "male-limited", antisocial traits, stronger familial loading.
BABOR	Type A: lower vulnerability, later onset, fewer problems. Type B: higher vulnerability, earlier onset, greater severity, polysubstance use and psychiatric comorbidity.
SCHUCKIT	Primary: alcohol disorder precedes independent psychiatric illness. Secondary: follows an established psychiatric disorder. Chronology alone does not prove causation.
LESCH	I: withdrawal-driven drinking. II: anxiety / conflict relief. III: depressive-affective pathway. IV: premorbid cerebral impairment, behavioural and social difficulties.

OTHER CARE → liver / pancreatic / cardiac disease: condition-specific referral; pregnancy: abstinence + obstetric care.

LOCAL PLAN → screen + brief intervention → treatment access → family / peer support → measure harm and uptake.

Sources: IPS intoxication guideline (2023); ASAM alcohol withdrawal (2020); UK alcohol guidance (2025); NICE CG115; Jellinek / Cloninger / Babor historical review; Schuckit (1969); Lesch typology. Full evidence links in companion source ledger.

Gold = verified IPS guidance. Purple treatment boxes use named supplementary guidance. Timelines vary; individualise care.

Alcohol - Effects management and typologies. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-86.

Credits and references: retained within the illustration.

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

WEAVE 100 · PAPER II · 87.2

87.2 COMPLICATED ALCOHOL WITHDRAWAL

Seizures • delirium tremens • quiet delirium • mimics • tiered hospital management

ENTRY: RECENT REDUCTION / CESSATION + SEIZURE OR DELIRIUM → HOSPITAL

Timing supports diagnosis; it does not prove it. Withdrawal can begin despite a positive blood alcohol level. First seizure, focal signs or atypical course → investigate alternative / additional causes.

FEATURE	WITHDRAWAL SEIZURE	DELIRIUM TREMENS	HYPOACTIVE DELIRIUM
Timing / nature	Usually 8–48 h after reduction; often generalised tonic–clonic.	Often 48–96 h; may occur later. Withdrawal-related delirium.	No fixed alcohol timeline. Reduced activity / arousal; may have several causes.
Attention / awareness	Brief post-ictal impairment; persistent confusion needs reassessment.	Fluctuating inattention, disorientation and disturbed awareness.	Same core delirium features, but withdrawn, slowed, drowsy; easily missed.
Motor / autonomic	Convulsions; other withdrawal signs may be mild or absent.	Usually agitation, tremor, sweating, tachycardia; hallucinations possible.	Little agitation; autonomic signs may be absent or masked by medication.
Key distinction	Recurrent / focal / prolonged fits → do not assume simple withdrawal.	Not every delirium in a drinker is delirium tremens.	Motor subtype ≠ cause. Check sepsis, metabolic illness, liver failure and sedation.

Hallucinosis: hallucinations with relatively preserved attention / awareness. Mixed motor delirium can fluctuate.

TIER 1 STABILISE + SEARCH FOR THE CAUSE • ALL PATIENTS

- Airway, breathing, circulation; bedside glucose; intravenous access; oxygen when needed; seizure precautions.
- Check blood count, glucose, sodium, potassium, magnesium, renal / liver function; electrocardiogram. Add infection, injury and toxicology evaluation as indicated.
- Thiamine promptly; never delay glucose for hypoglycaemia. Correct deficits with monitored fluids / nutrition.

TIER 2A SEIZURE → STOP + PREVENT RECURRENCE

- Active fit: lorazepam 4 mg intravenously; repeat once after 10 min if needed. Adult UK / BNF example; resuscitation-ready care.
- ≥5 min / recurrent fits without recovery → status pathway now.
- After fit: benzodiazepine + monitor recurrence / delirium.
- No phenytoin for pure withdrawal; treat coexisting epilepsy.

TIER 2B WITHDRAWAL DELIRIUM → TITRATE

- Benzodiazepines first-line; monitored intravenous loading / titration by hospital protocol. Target calm, lightly somnolent but arousable.
- Liver dysfunction → often lorazepam. Monitor breathing, oxygenation, blood pressure and sedation.
- Use objective sedation / delirium assessments. Do not rely on the Clinical Institute Withdrawal Assessment self-report scale in delirium.

TIER 3 PERSISTENT SEVERE WITHDRAWAL / RESPIRATORY RISK → INTENSIVE CARE

- Recheck diagnosis, medication delivery, cumulative doses and coexisting disease → senior / critical-care review.
- Phenobarbital: specialist adjunct with close respiratory monitoring; not an unsupervised escalation.
- Dexmedetomidine: monitored adjunct for autonomic arousal; does not replace seizure-preventing treatment.
- Propofol: resistant withdrawal in patients who already require mechanical ventilation.
- Antipsychotic only as an adjunct for persistent severe psychotic / agitated symptoms; never sole withdrawal therapy.

COMPLICATIONS / COEXISTING ILLNESS → ACT

- Aspiration / hypoxia → airway support; assess pneumonia.
- Fever / rhabdomyolysis → cooling, renal / muscle tests, guided fluids.
- Electrolyte loss / arrhythmia → replace deficits, monitor heart; avoid rapid sodium correction.
- Suspected Wernicke → high-dose parenteral thiamine (86.1).
- Infection, pancreatitis, gut bleeding, hepatic encephalopathy / head injury → specific treatment.

ATYPICAL SEIZURE / CONFUSION → BRAIN WORK-UP

- New severe headache, papilloedema, focal signs / fits or persistent impaired awareness → urgent imaging.
- Cerebral venous thrombosis (CVT): brain imaging + computed-tomography / magnetic-resonance venography; plain scan cannot exclude it.
- Confirmed CVT → stroke-team anticoagulation unless contraindicated; treat seizures / raised pressure.
- Exclude subdural bleed, stroke, infection and non-convulsive status; electroencephalogram if indicated.

QUIET / DROWSY PATIENT? → REASSESS BEFORE MORE SEDATION

- Hypoactive delirium is not proof of recovery. Review sedatives, oxygenation, glucose, infection, liver / renal failure and electrolytes.
- Treat proven withdrawal, but do not escalate benzodiazepines solely because confusion persists.
- Orientation, sleep–wake cues, glasses / hearing aids, hydration, mobility and family support accompany causal treatment.

IPS GUIDELINE ANCHOR • DELIRIUM IN ELDERLY, 2018

- Correct causes; review offending medicines. Alcohol-withdrawal delirium → benzodiazepines + thiamine / nutrition.
- Gold = IPS principles. Adult escalation above: ASAM / UK supplementary guidance.

HISTORICAL ALCOHOL TYPOLOGIES • descriptive frameworks, not diagnostic criteria

Jellinek	Alpha: psychological reliance; beta: harm without dependence; gamma: loss of control; delta: cannot abstain; epsilon: episodic bouts.
Cloninger	I: later onset, both sexes, anxiety / guilt. II: early onset, historically male-limited, antisocial traits, stronger familial loading.
Babor	A: lower vulnerability / severity, later onset. B: higher vulnerability / severity, earlier onset, more comorbidity.
Schuckit	Primary: alcohol disorder first. Secondary: independent psychiatric disorder first. Chronology does not establish causation.
Lesch	I: withdrawal-driven; II: anxiety / conflict; III: depressive-affective; IV: premorbid cerebral / behavioural difficulties.

RECOVERY → cognition / nutrition review → safe step-down → relapse prevention + psychosocial care.

Sources: local IPS delirium CPG (2018, elderly scope); ASAM withdrawal (2020); UK hospital alcohol guidance / BNF seizure example; Canadian Stroke Best Practices (2024); typology evidence reused from 86.1. Full ledger retained.

CVT is a differential / coexisting condition, not a routine withdrawal stage. Adult doses: monitored hospital care only.

Complicated alcohol withdrawal - Seizures delirium and tiered management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-87.

Credits and references: retained within the illustration.

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

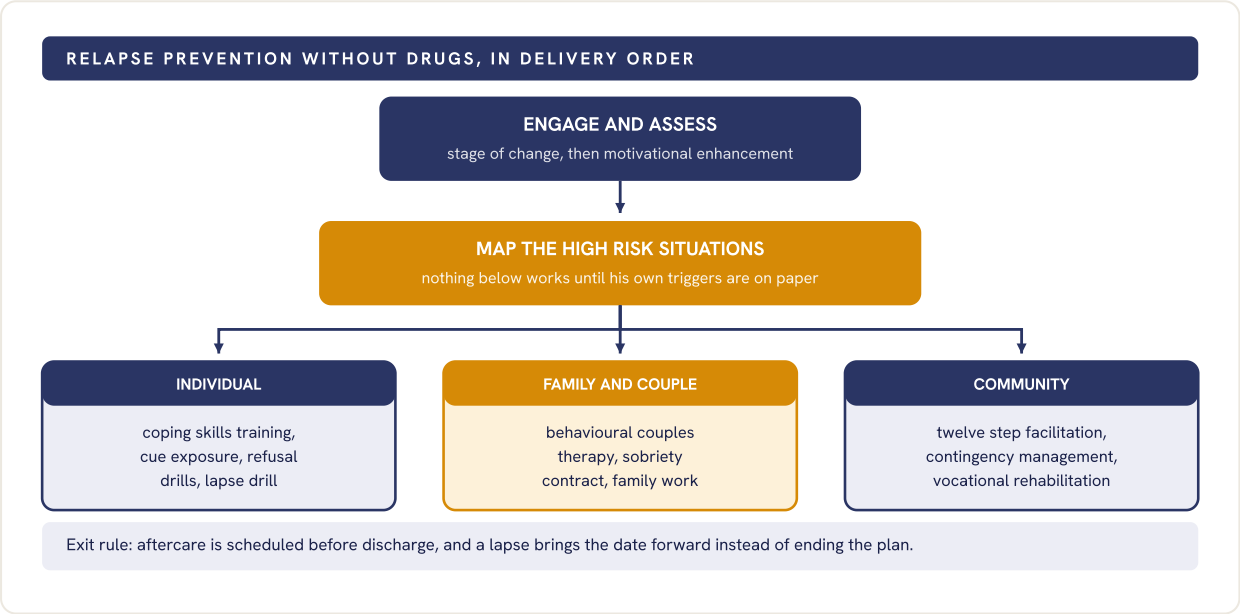


Fig 089.1 Engagement first, the trigger map as the gate, then three lanes that run together rather than in sequence.

- **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

WEAVE 100 · PAPER II · 86.2

Substance-use rating scales

Screening risk · dependence severity · current withdrawal are different questions

Choose before scoring

Alcohol screen → AUDIT. Alcohol dependence severity → SADQ. Alcohol withdrawal now → CIWA-Ar. Opioid withdrawal → COWS. Tobacco dependence → Fagerström test. Do not exchange thresholds between instruments or use a low score to dismiss a concerning clinical presentation.

Alcohol Use Disorders Identification Test · AUDIT · WHO

10 items, self-report or interview, total 0–40; mainly past-year consumption, dependence features and harm. Higher scores suggest greater risk / need for assessment.
Conventional zones: 0–7 lower risk; 8–15 hazardous use; 16–19 harmful use; ≥20 possible dependence requiring diagnostic assessment. These are screening / intervention zones, not diagnoses.
Clarify the standard drink used and population-specific thresholds. AUDIT-C is only the 3 consumption items (0–12); it is not the full AUDIT and has different cut-offs.

Severity of Alcohol Dependence Questionnaire · SADQ

Self-report dependence severity: 20 items, each 0–3, total 0–60. Covers withdrawal, affective discomfort, relief drinking, consumption and reinstatement after abstinence. The standard form asks about a recent typical period of heavy drinking; use that form's instructions.
SADQ ≥31 indicates severe dependence in NICE CG115 / Stockwell convention. Lower boundaries differ by protocol. A high score contributes to withdrawal planning alongside prior seizures / delirium, medical illness and support; it is not a current-withdrawal score.

Clinical Institute Withdrawal Assessment for Alcohol–Revised · CIWA-Ar

Clinician interview + observation of current withdrawal: 10 items; nine 0–7 and orientation 0–4, total 0–67. Repeat according to the clinical protocol.
ASAM 2020 illustrative ranges: <10 mild, 10–18 moderate, ≥19 severe. Thresholds vary; seizures, delirium or physiological instability demand assessment regardless of score.
It is not diagnostic of alcohol withdrawal and does not predict risk on its own. Symptoms can reflect pain, anxiety, infection or other illness. Do not use CIWA-Ar for delirious / non-communicative patients as if self-report were reliable; use an appropriate monitored-care assessment.

Other substances: use the matching instrument

Clinical Opiate Withdrawal Scale (COWS): clinician-rated, 11 items, total 0–48; current opioid withdrawal. Published bands: 5–12 mild, 13–24 moderate, 25–36 moderately severe, >36 severe. Interpret with timing and objective signs; not an opioid-use-disorder diagnosis.
Fagerström Test for Nicotine Dependence: 6 items, total 0–10; cigarette-related dependence. It does not measure acute withdrawal or all forms of tobacco use equally.

Bedside sequence

History and examination → assess risk of complicated withdrawal → choose the appropriate scale if valid → repeat and interpret the trajectory → treat and monitor using the clinical protocol.
Record time since last use, other substances, medications, cognition, vital signs and relevant medical conditions.
Withdrawal care should connect to ongoing treatment for the use disorder.

Do not turn a score into an automatic prescription

A screening score is not a detoxification regimen. A dependence score is not a withdrawal observation. A withdrawal score is not the whole risk assessment.
Name the edition, rater, time window, units and limitations in an exam answer. Scores supplement clinical judgement and a defined treatment setting.

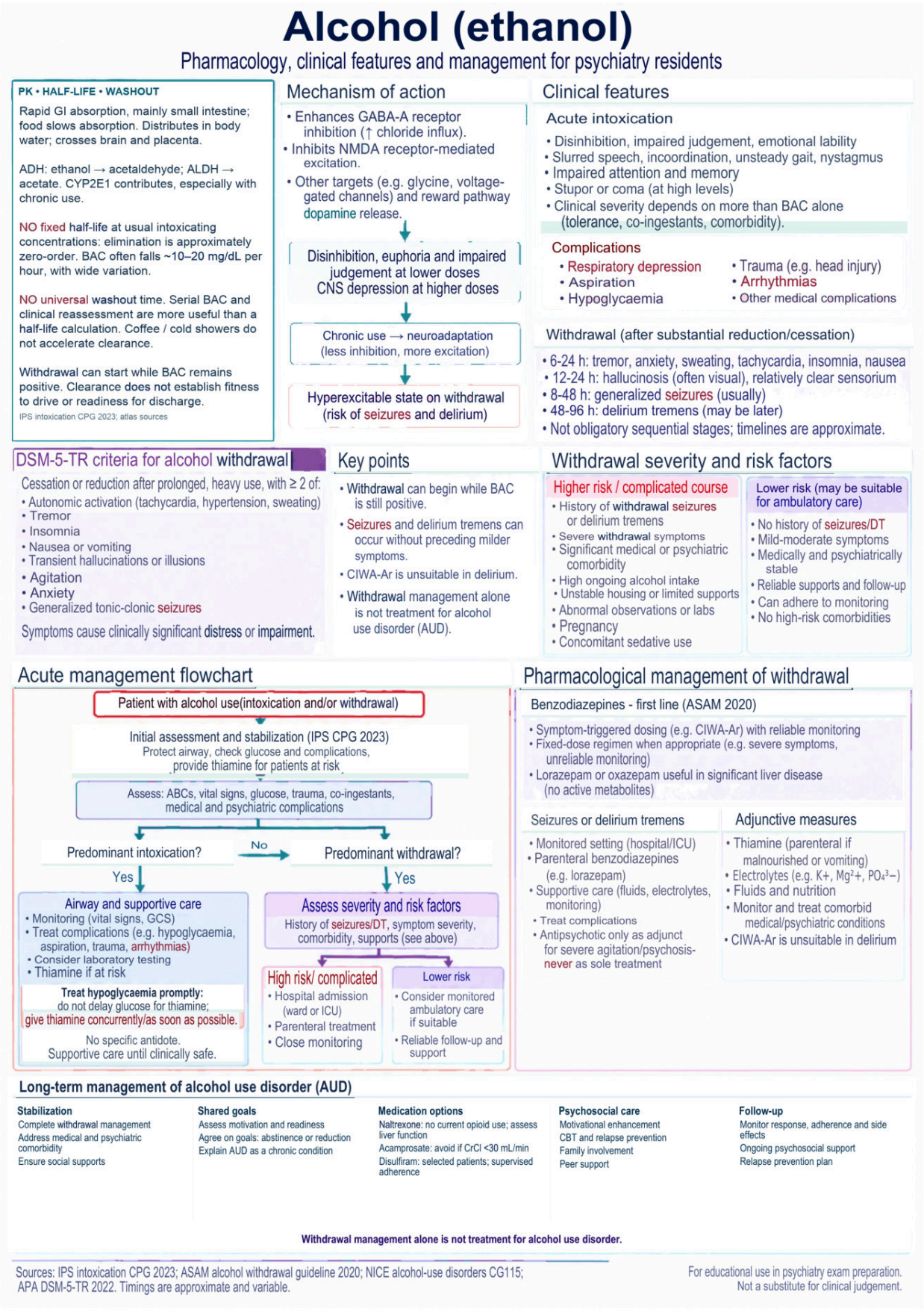
Sources: substance-area supplement Q11–18/Q86–89. WHO AUDIT manual 2nd ed. (2001); NICE PH24/CG115 (2010/2011). Stockwell SADQ (1979/1983). Sullivan et al. CIWA-Ar (1989), PMID2597811; ASAM alcohol withdrawal CPG (2020). Wesson & Ling COWS (2003), NIDA form. Heatherton et al. FTND (1991); Baer & Blais handbook (2010), Ch5.

Q86 supplement · ASAM CIWA-Ar bands explicitly labelled · 17 Sep 2026 A4 · 300 dpi

Substance use and withdrawal rating scales. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: general concepts and alcohol.

Credits and references: retained within the illustration.

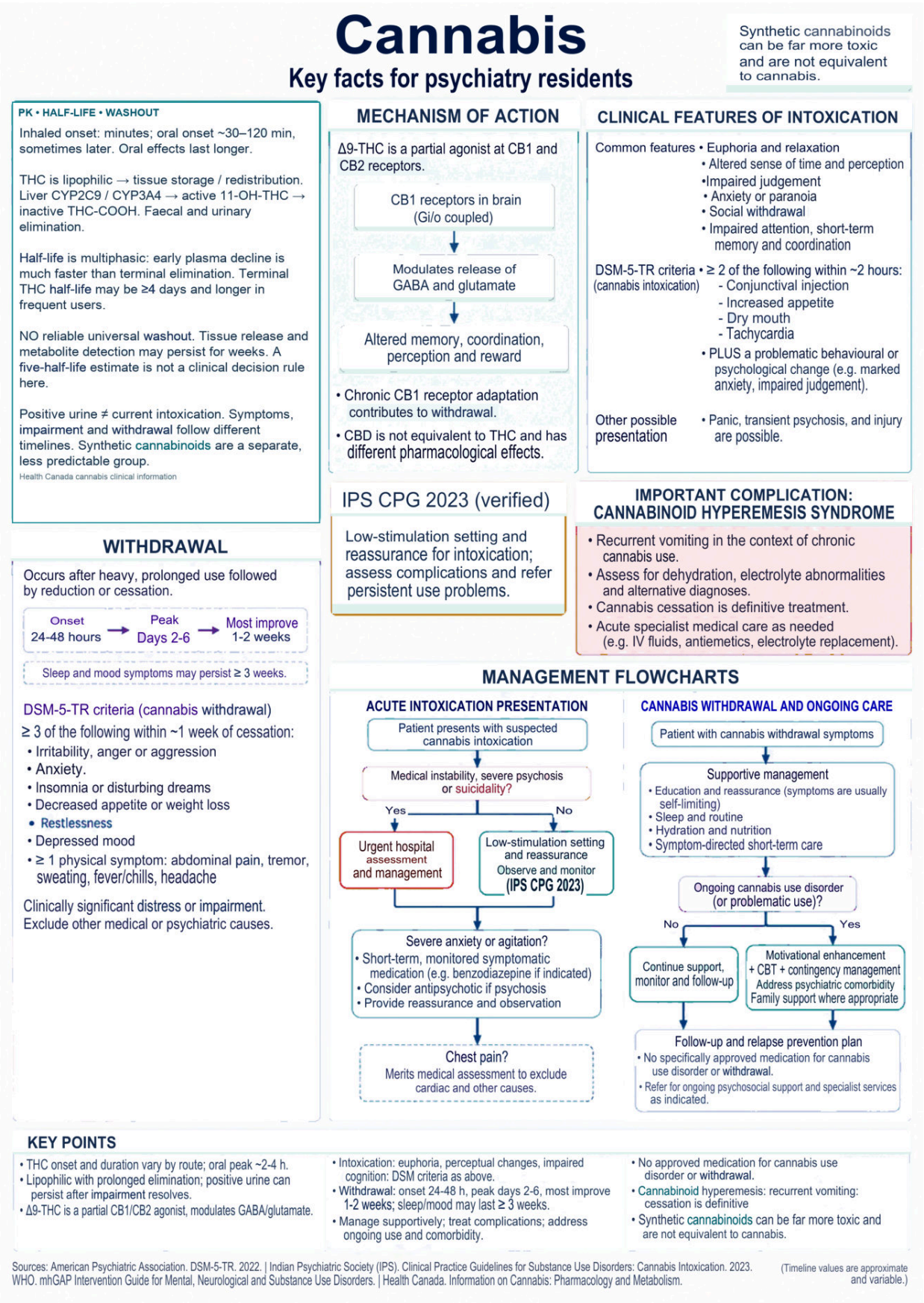
WEAVE 100 · PAPER II · 87.1



Alcohol - Pharmacology intoxication withdrawal and management. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: general concepts and alcohol.

Credits and references: retained within the illustration.

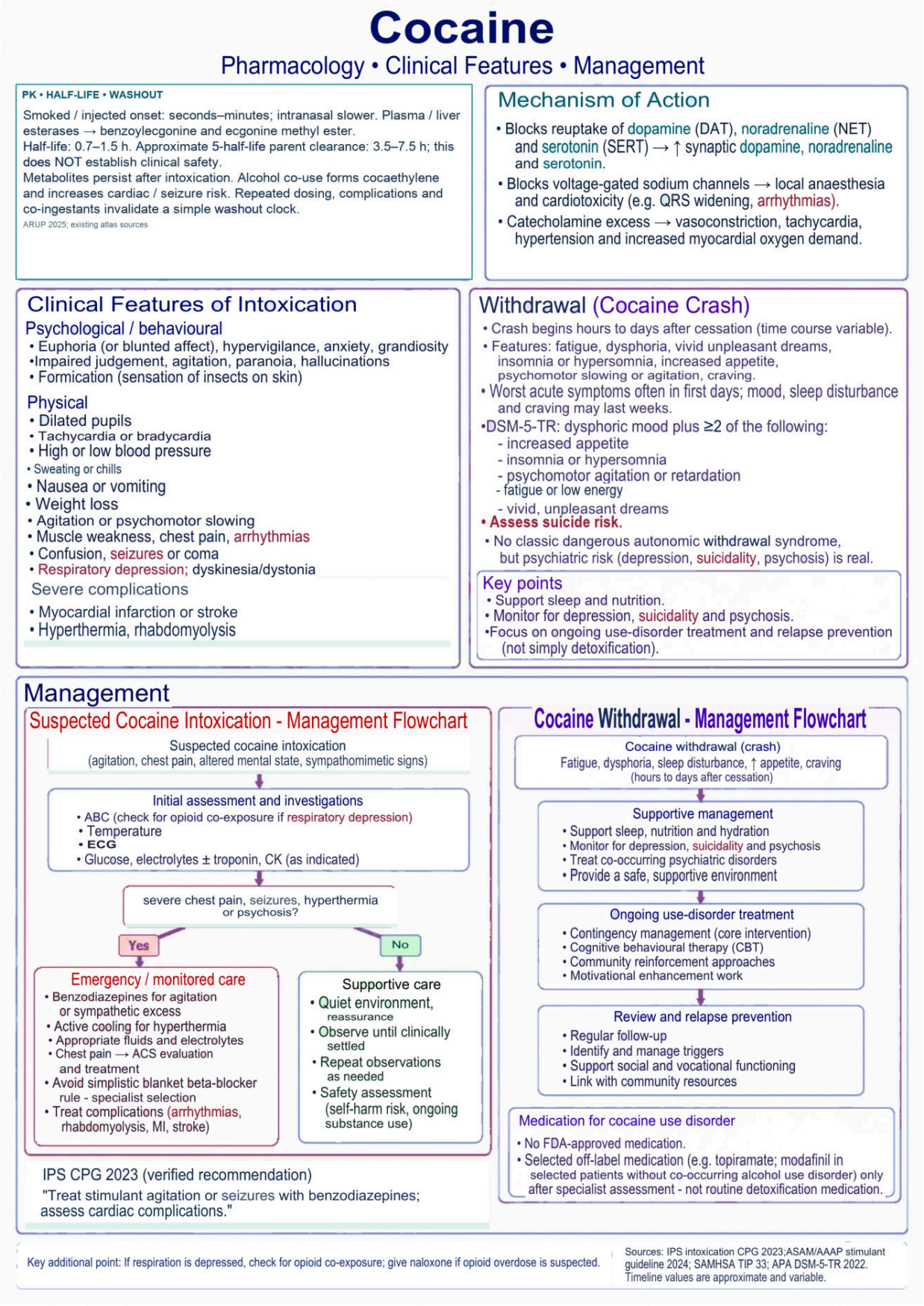
WEAVE 100 · PAPER II · 90.1



Cannabis - Pharmacology intoxication withdrawal and management. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: general concepts and alcohol.

Credits and references: retained within the illustration.

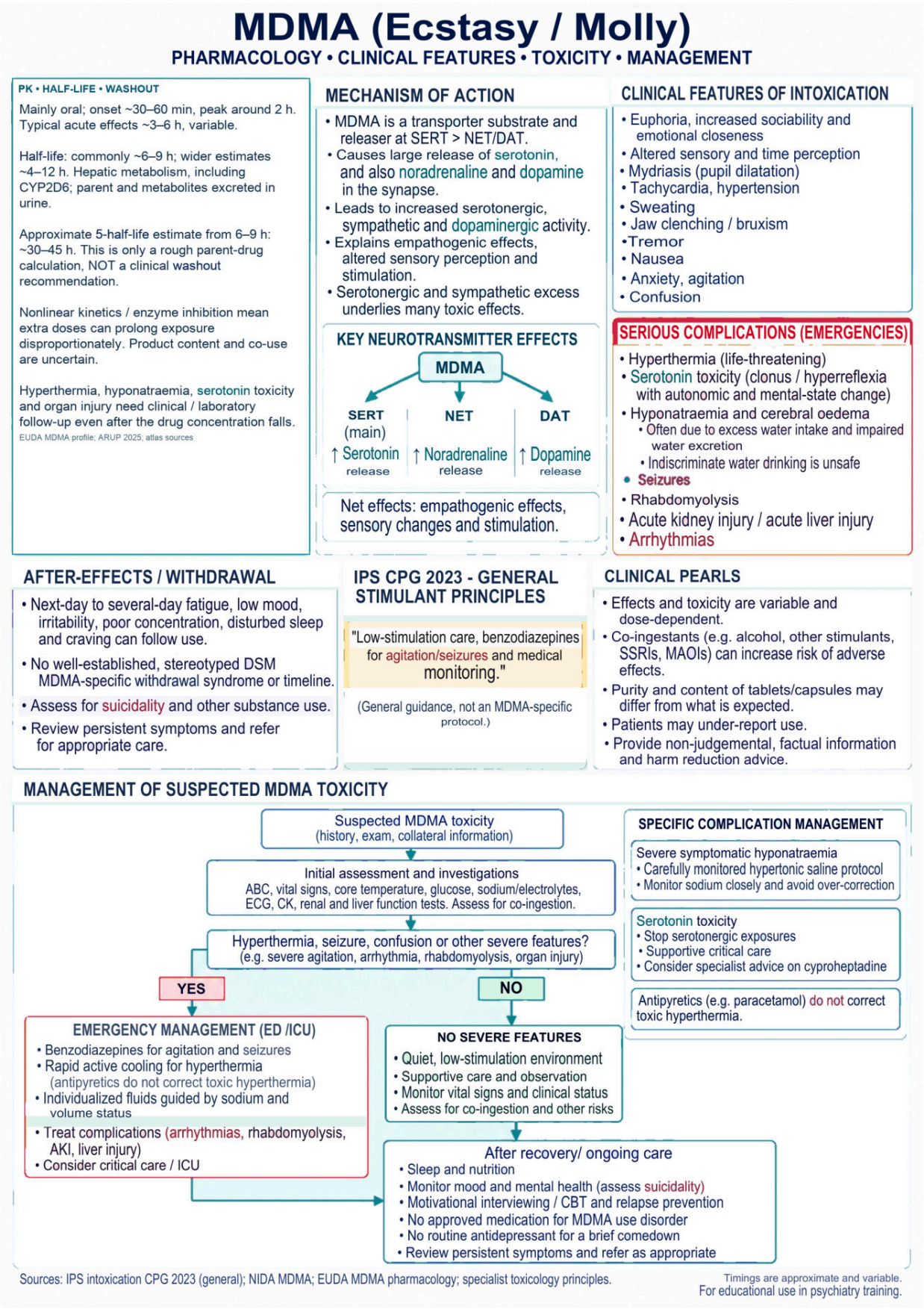
WEAVE 100 · PAPER II · 90.2



Cocaine - Pharmacology intoxication withdrawal and management. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: general concepts and alcohol.

Credits and references: retained within the illustration.

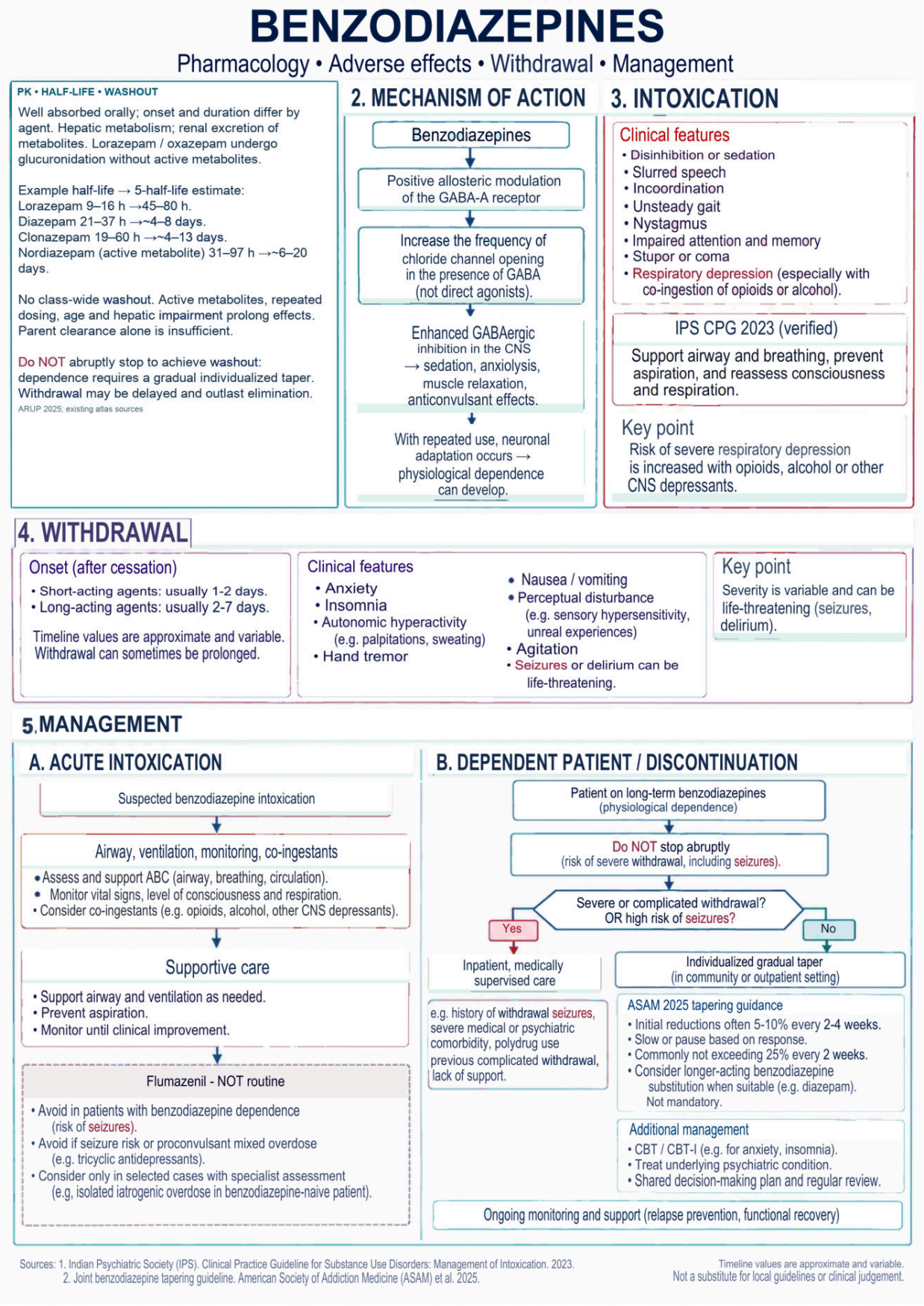
WEAVE 100 · PAPER II · 90.3



MDMA - Pharmacology toxicity and management. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: general concepts and alcohol.

Credits and references: retained within the illustration.

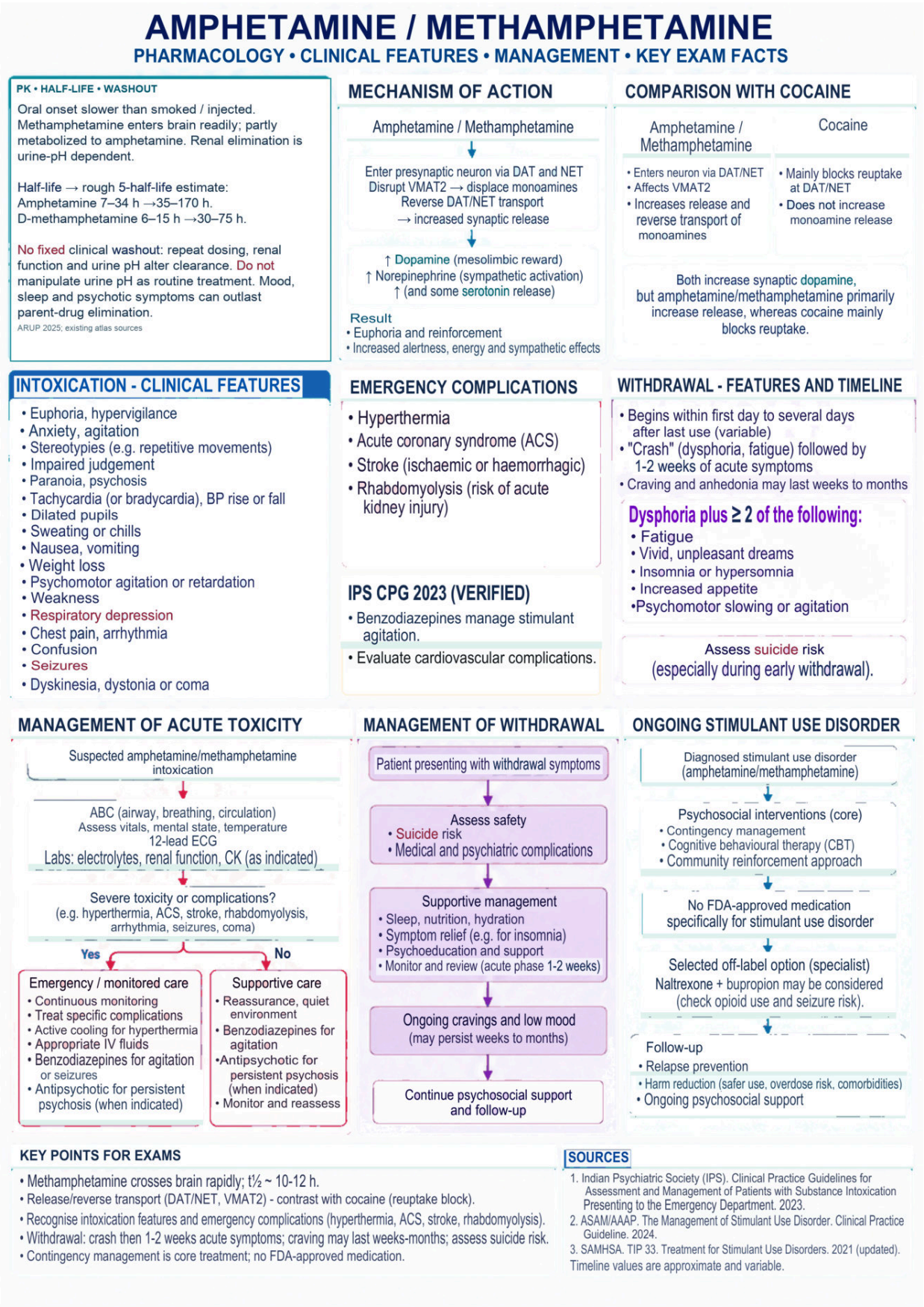
WEAVE 100 · PAPER II · 90.4



Benzodiazepines - Pharmacology intoxication withdrawal and management. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: general concepts and alcohol.

Credits and references: retained within the illustration.

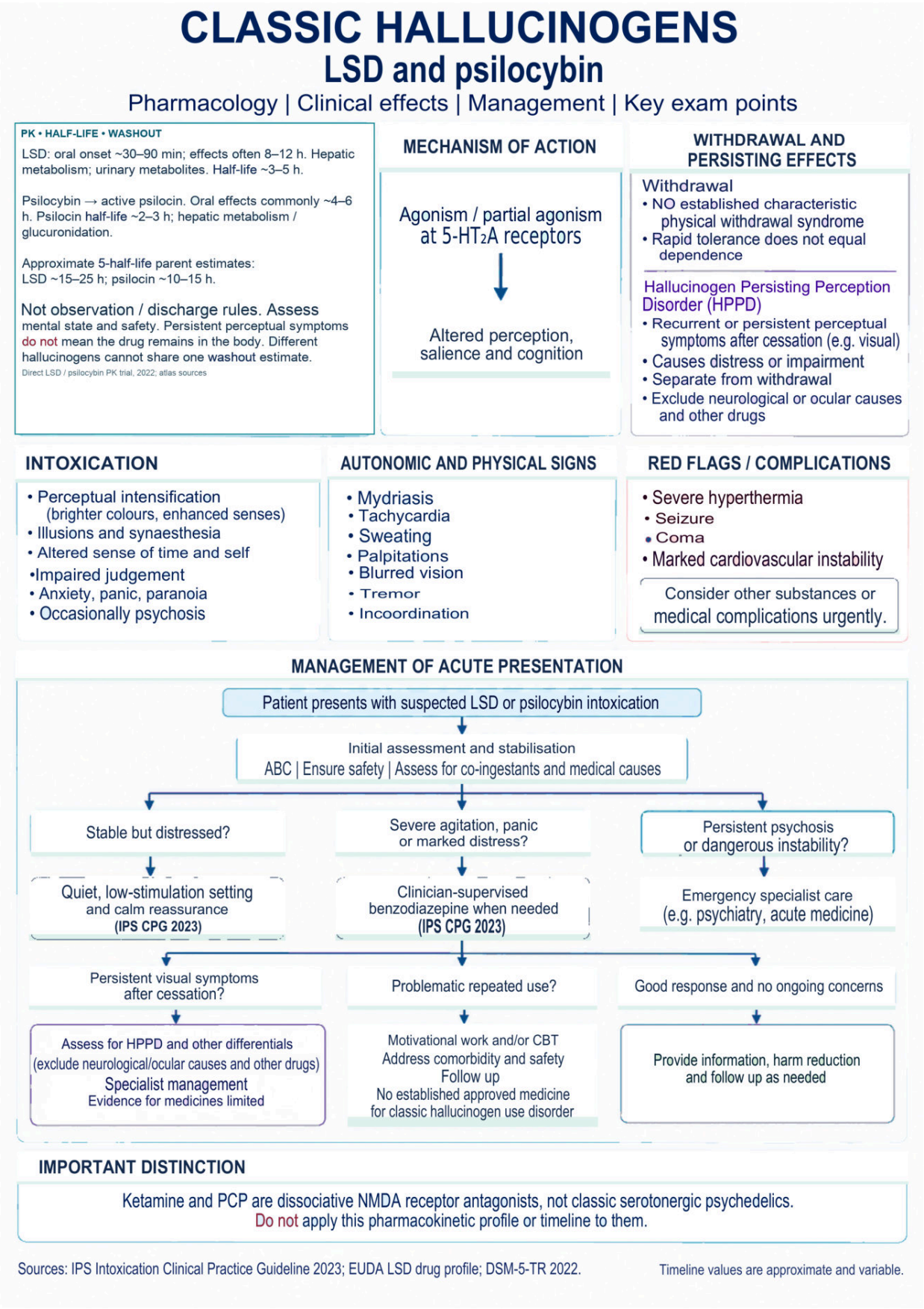
WEAVE 100 · PAPER II · 90.5



Amphetamine and methamphetamine - Pharmacology intoxication withdrawal and management. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: general concepts and alcohol.

Credits and references: retained within the illustration.

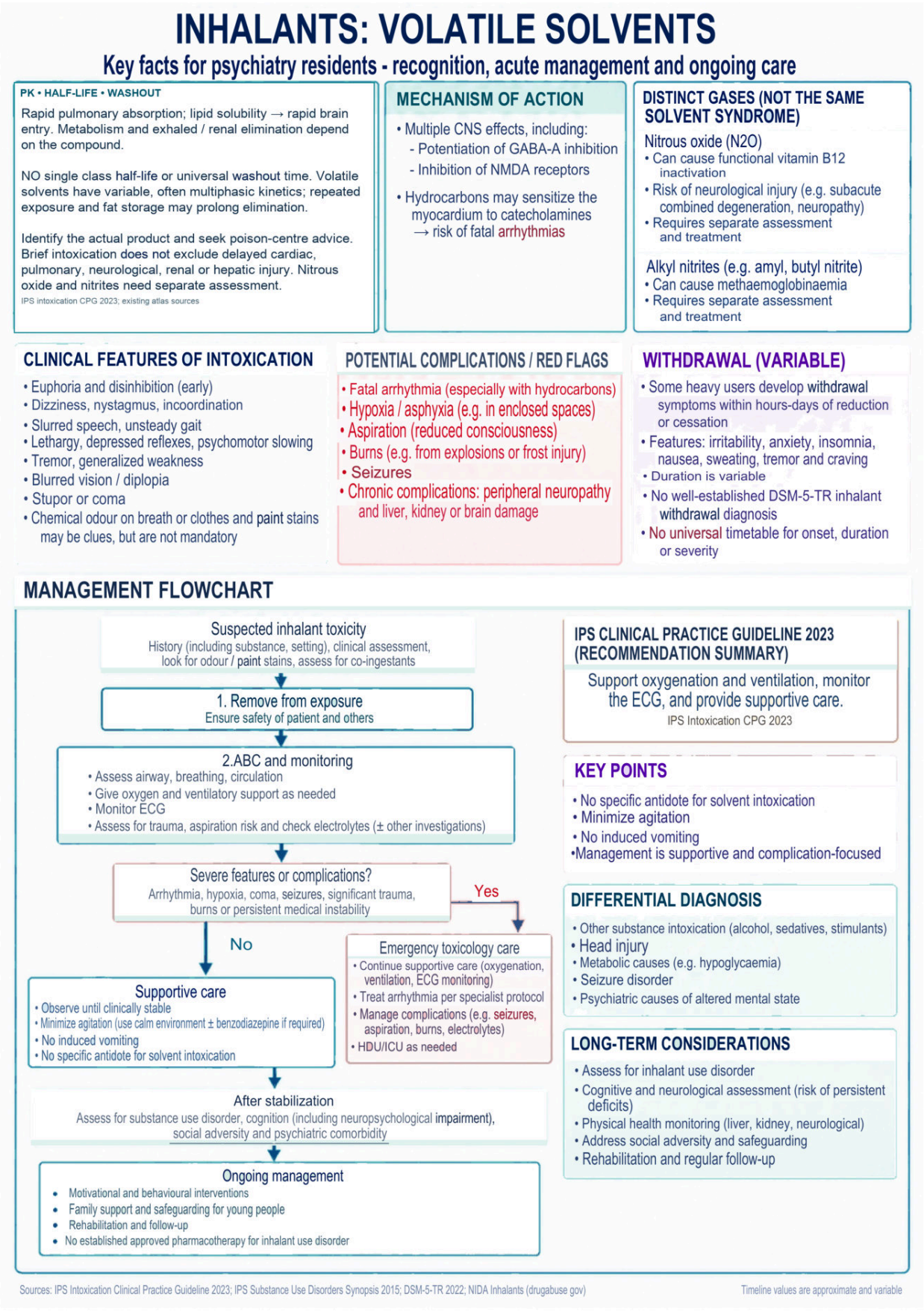
WEAVE 100 · PAPER II · 90.6



Classic hallucinogens - Pharmacology clinical effects and management. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: general concepts and alcohol.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER II · 90.7



Inhalants - Pharmacology intoxication withdrawal and management. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: general concepts and alcohol.

Credits and references: retained within the illustration.

90.8 WHY DOES THE BEHAVIOUR KEEP REPEATING?

Alcohol • anxiety • obsessive-compulsive disorder (OCD): reward or relief can reinforce repetition

ALCOHOL • SEEK → NEED

REWARD
Ventral tegmental area → nucleus accumbens

↓

CUES + REPETITION
Glutamate learning → stronger habits

↓

HABIT + WEAKER FLEXIBLE CONTROL
Dorsal striatum ↔ prefrontal cortex

↓

STRESS / WITHDRAWAL → RELIEF SEEKING
Extended amygdala + stress systems

Dopamine: "want / repeat"
Endogenous opioids: reward
Glutamate: learned cues
Noradrenaline + dynorphin: stress

Reward and relief can coexist.
Habit is not automatically compulsion.

ANXIETY • DETECT → AVOID

POSSIBLE THREAT

LOW ROAD
Thalamus → amygdala
Rapid, coarse input

HIGH ROAD
Thalamus → cortex → amygdala
Richer appraisal

DEFENSIVE RESPONSE / AROUSAL

↓

AVOIDANCE → RELIEF → REPEAT

Glutamate: threat learning
Gamma-aminobutyric acid: inhibition
Noradrenaline: arousal
Serotonin: circuit modulation

Classical model, not the whole fear system.
Cortical appraisal can also amplify threat.

OCD • DOUBT → REPEAT

INTRUSION / DOUBT / "NOT JUST RIGHT"

↓

CORTEX
Orbitofrontal + anterior cingulate

↓

STRIATUM → PALLIDUM → THALAMUS → back to cortex

↓

RITUAL → BRIEF RELIEF → REPETITION
Relief reinforces the ritual

Glutamate + gamma-aminobutyric acid:
excitation / inhibition in action loops
Serotonin + dopamine: modulation

Error monitoring + action control
+ habit / goal-directed balance

Not just anxiety. Some rituals address incompleteness rather than feared harm.

DO NOT EQUATE: ventral → dorsal habit recruitment ≠ low-road / high-road threat pathways

BREAK THE ALCOHOL CYCLE

Safe withdrawal care → relapse prevention
+ coping skills + social recovery

BREAK AVOIDANCE LEARNING

Exposure / cognitive treatment
→ learn new responses to threat

BREAK THE RITUAL CYCLE

Exposure + response prevention
± selective serotonin reuptake inhibitor

RESEARCH LAYER: immune signalling / cytokines + neuroplasticity may contribute.
Not routine diagnostic tests. Full molecules and evidence are in the companion notes.

Stahl 5th ed., indexed chapter summaries 8 & 13; Koob & Volkow 2016; LeDoux & Pine 2016; Gillan 2011. Simplified learning models, not identical diseases.
OCD treatment anchor: IPS 2025 update, published 2026. Full source ledger retained. The detailed original sheet is preserved in backups.

Impulsivity compulsivity - Alcohol anxiety and OCD neurobiology. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Substance use: general concepts and alcohol.

Credits and references: retained within the illustration.

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

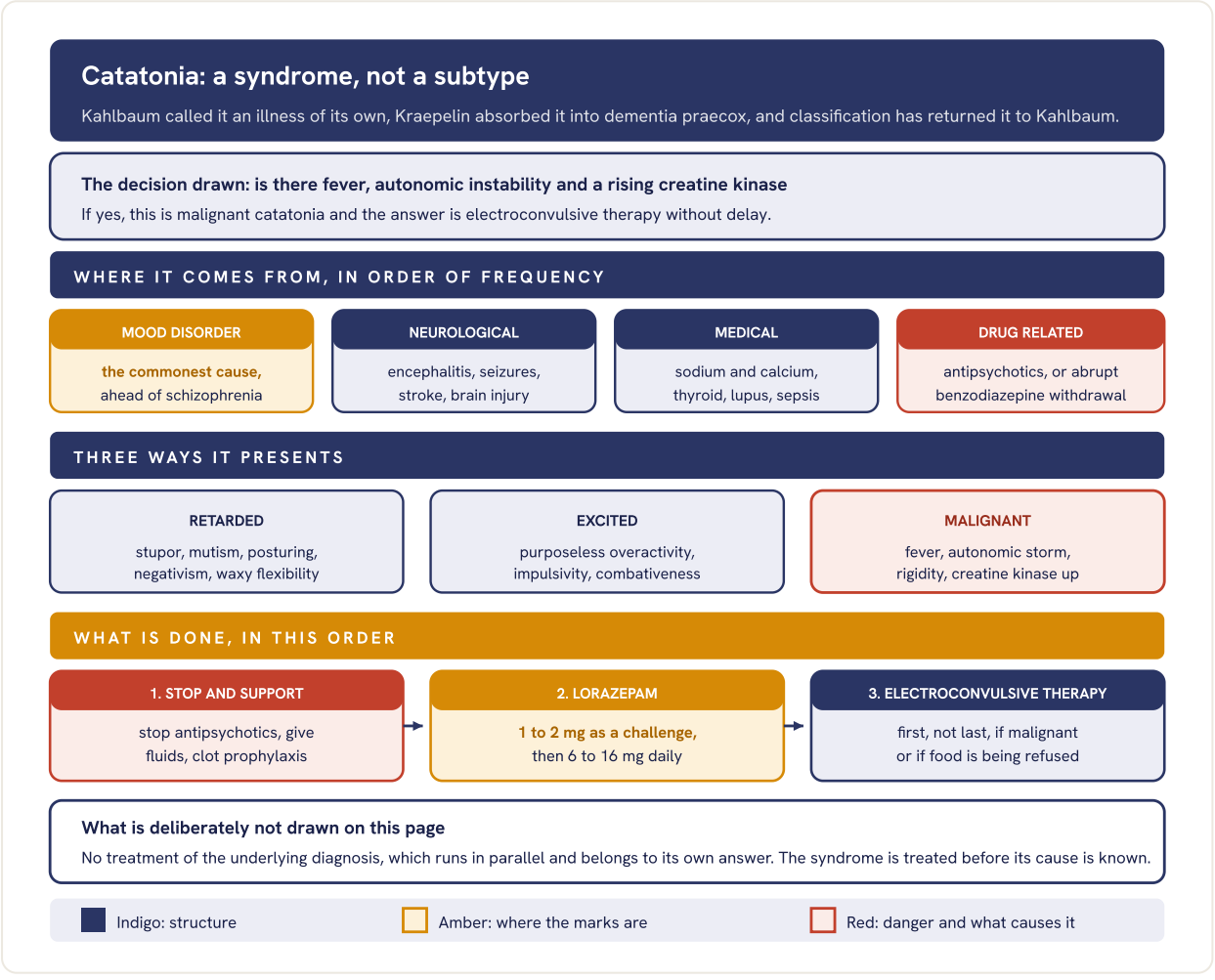


Fig 091.1 Catatonia as cause, presentation and treatment, with the malignant form separated because it changes the order.

- **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

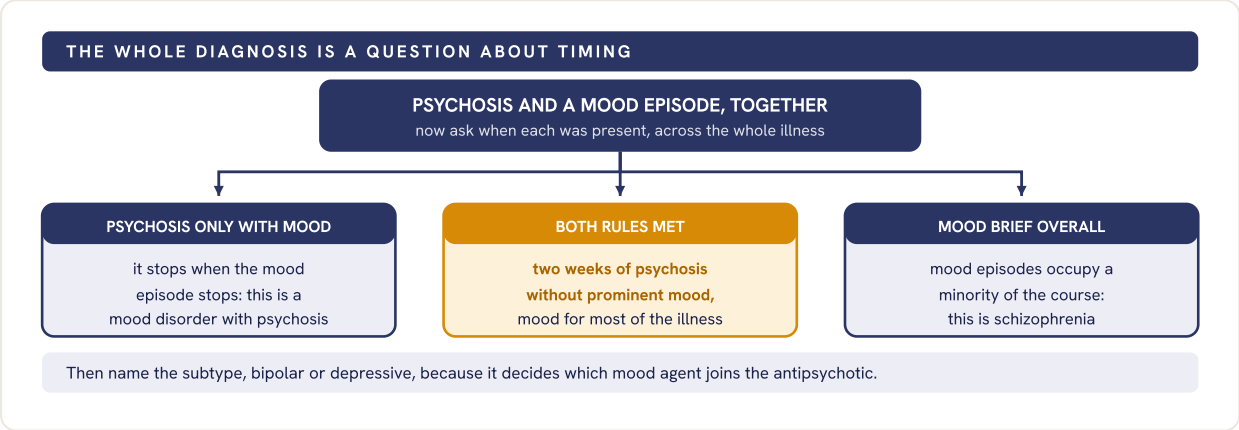


Fig 092.1 The differential drawn as a timing question, because both separating rules are rules about when.

- **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

WEAVE 100 · PAPER II · 92.1

Schizoaffective disorder

Nosology and timing · WHO ICD-11 criteria · phase-specific management

ICD-11 6A21 · CDDR 2024 · both syndromes in the same episode

1 Full schizophrenia requirements + a manic, mixed, or moderate / severe depressive episode. Depression must include depressed mood, not anhedonia alone.

2 Psychotic and mood symptoms begin together or within a few days. Both symptomatic syndromes last ≥1 month. Exclude substances / medicines and medical causes.

Schizophrenia core: ≥2 qualifying symptoms, ≥1 being delusions, hallucinations, disorganised thinking or influence / passivity / control; other domains include negative, disorganised behavioural and psychomotor symptoms.

Nosology: within schizophrenia / other primary psychotic disorders; an episode-based diagnosis. Specify first / multiple episodes or continuous course, and current symptom / remission status. Revisit the longitudinal formulation.

DSM-5-TR 2022 · different longitudinal rules

Major mood episode + schizophrenia Criterion A; ≥2 weeks of delusions / hallucinations without a major mood episode; mood episodes occupy most of the active + residual illness. Bipolar type includes mania; depressive type only depression.

DSM: psychosis only with mood episodes → mood disorder with psychosis; mood episodes a minority of the course → consider schizophrenia. These are not ICD-11 timing rules.

IPS first · schizophrenia CPG 2026 · relevant guidance, not a separate algorithm

IPS discusses schizoaffective presentations in its schizophrenia CPG. Below integrates IPS bipolar / depression guidance (2026); some decisions are extrapolated from the dominant syndrome.

Assess safety, catatonia, substances, adherence, past response and physical / reproductive health.

Treatment by phase / polarity · IPS 2026 synthesis

ACUTE PSYCHOSIS	Choose an antipsychotic by prior benefit, tolerability and patient preference. Use one adequate trial; assess dose, adherence, duration and substances before declaring failure.
MANIA / MIXED	Antipsychotic ± lithium or valproate according to severity / response. Avoid or withdraw antidepressants under supervision. Valproate requires reproductive-risk assessment; lithium needs serum / renal / thyroid monitoring.
DEPRESSIVE	Exclude negative symptoms / adverse effects. Bipolar history → bipolar-depression pathway; no antidepressant monotherapy. Otherwise consider adjunct antidepressant for persistent depression; monitor switching / psychotic worsening.

Persistence, urgency and maintenance

Severe / refractory mood illness: consider electroconvulsive therapy (ECT). Catatonia needs urgent care.

Persistent psychosis after ≥2 adequate trials → assess for clozapine. Maintain effective treatment; consider a long-acting injection for preference / adherence. Review benefit and burden.

Recovery and physical monitoring

Psychoeducation, cognitive-behavioural therapy for psychosis, family work, relapse planning and education / employment support.

Track weight / waist, BP, glucose / HbA1c, lipids, movement effects, prolactin symptoms and ECG if indicated. Add drug-specific monitoring; review sexual health and substances.

Supplement · US paliperidone label · current DailyMed

Paliperidone ER has a US schizoaffective indication alone or with mood stabilisers / antidepressants. This does not establish superiority; verify local approval and renal dosing.

Sources: Weave Q92 p126 (DSM-centred timing figure corrected by explicit system labels). WHO ICD-11 CDDR (2024), pp162–163,170–171; DSM-5-TR (2022), pp121–123. IPS schizophrenia CPG (2026), mood-symptom table; IPS bipolar / depression CPGs (2026). DailyMed paliperidone ER label (accessed 17 Sep 2026).

Q92 · full criteria require the clinical manuals · checked 17 Sep 2026 A4 · 300 dpi

Schizoaffective criteria and management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-92.

Credits and references: retained within the illustration.

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.

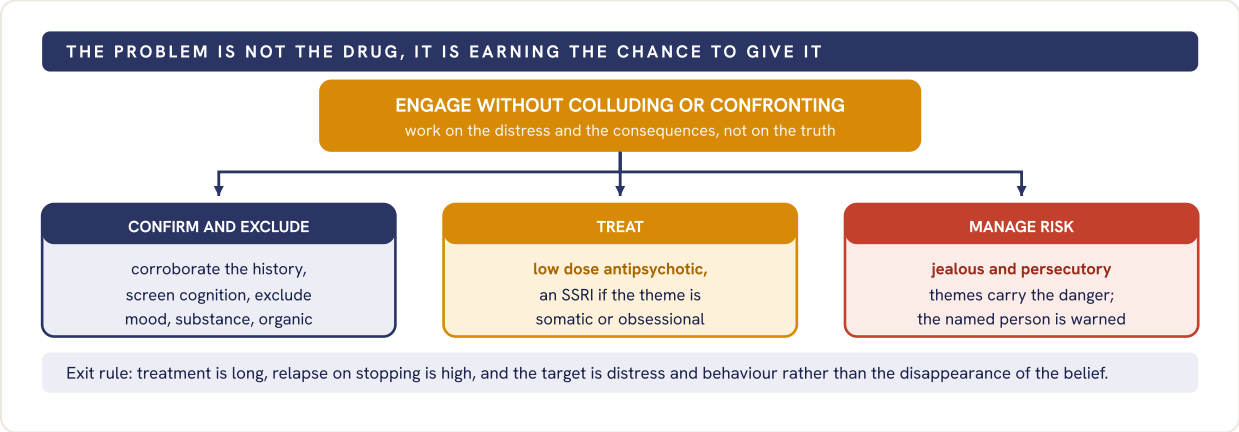


Fig 093.1 Management arranged around the real obstacle, engagement, with the three arms that follow it.

- **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.

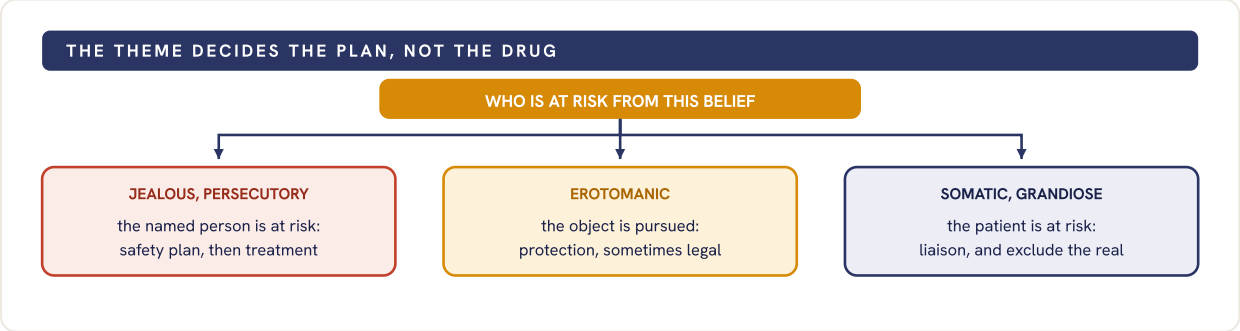


Fig 094.1 Management sorted by who the belief endangers, which is the axis that changes the plan.

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.

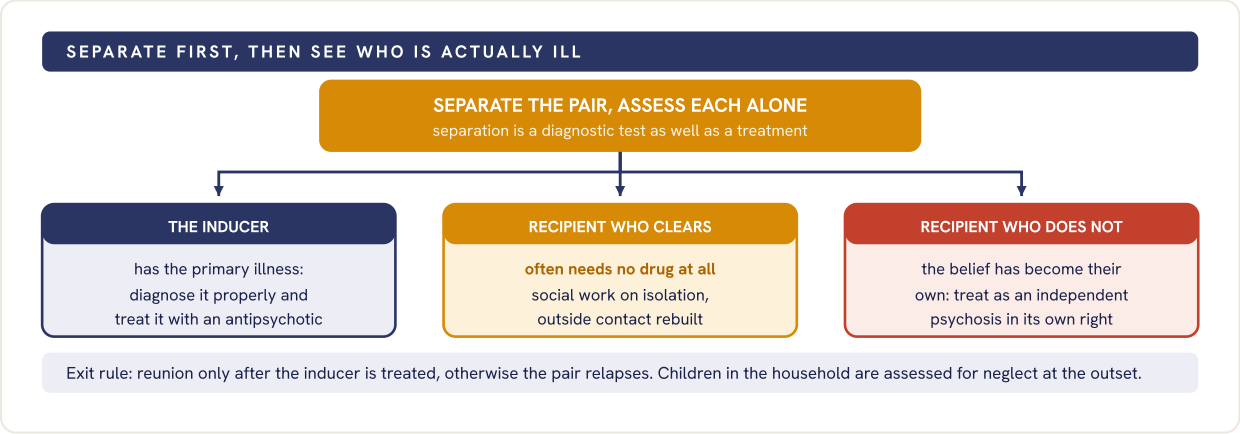


Fig 095.1 Separation used as both test and treatment, with the three outcomes it produces.

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

WEAVE 100 · PAPER II · 96.1



Suicide assessment and management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-96.

Credits and references: retained within the illustration.

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.

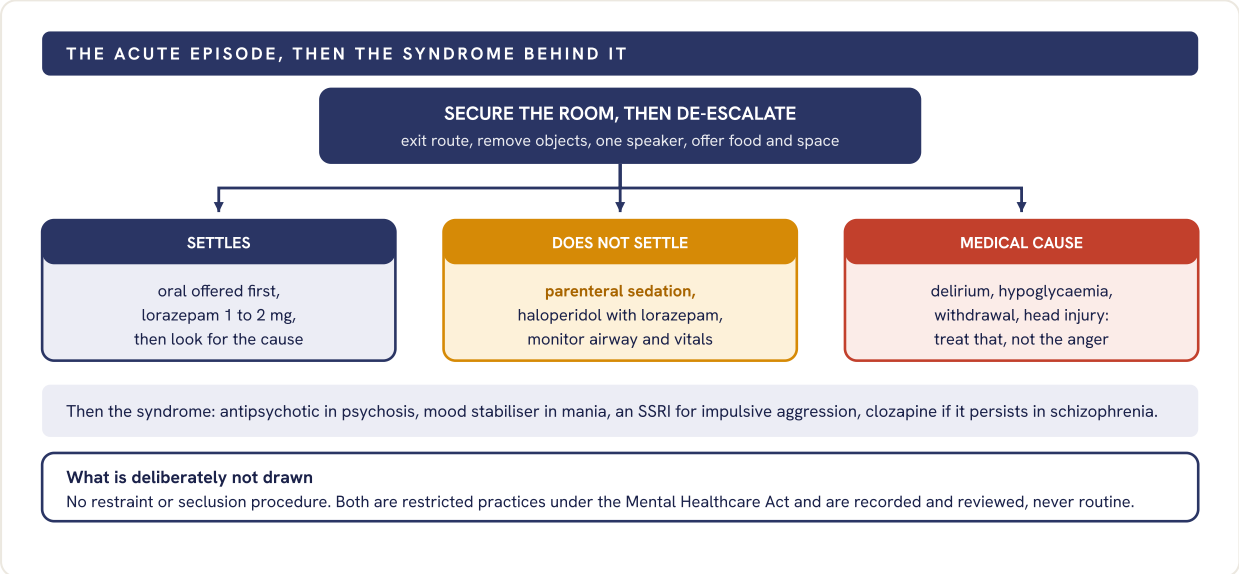


Fig 097.1 The room is made safe before anything is given, and the three lanes are separated by whether the episode settles and whether a medical cause is driving it.

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

WEAVE 100 · PAPER II · 97.1

Aggression: assess and manage

Understand the behaviour · treat its cause · calm without excessive sedation

Nature and types · a behaviour, not a diagnosis

Aggression: intended harm to someone who wishes to avoid it; verbal, physical or relational. Violence involves physical force / threat. Agitation need not involve aggression.

Reactive / impulsive: triggered, high arousal, poorly controlled. Proactive / instrumental: planned, goal-directed, often lower arousal. Types may coexist; neither alone selects a drug or proves mental illness.

IPS aggression CPG · 2023 · assess while securing safety

Call trained help; protect others and keep an exit route. Check airway, vital signs, oxygenation, glucose, attention / orientation and injury. Consider delirium, hypoxia, pain, head injury, seizures, intoxication or withdrawal before assuming psychiatric illness.

Assess threats / weapon access, past violence, triggers, psychosis / mania and supports; obtain collateral. Record antecedent → behaviour → consequence. Brøset supports repeated assessment, not a substitute for judgement.

IPS 2023 · de-escalate first, continue throughout

One calm speaker; respectful distance and short sentences. Listen, offer choices, meet basic needs, reduce noise / audience and set nonthreatening limits. Offer appropriate oral medication collaboratively.

If imminent danger persists, use the least restrictive effective intervention with trained staff and monitoring.

Cause-directed care · IPS 2023 framework

Delirium / medical	Treat the cause urgently; sedatives can obscure deterioration. Medication only when necessary for distress / danger.
Withdrawal / stimulants	Benzodiazepines often indicated; assess respiratory status. Alcohol / sedative intoxication: avoid adding respiratory depressants.
Psychosis / mania	Antipsychotic plus syndrome-specific care. Address trauma, communication and unmet needs; avoid indefinite PRN sedation.

Adult rapid tranquillisation · NICE NG10 (2015) + Maudsley 15e (2025)

Urgent calming when oral treatment is unsuitable / insufficient. Initial adult examples: IM lorazepam 2 mg OR IM haloperidol 5 mg + IM promethazine 25–50 mg. Individualise to cause, response and physical status.

No ECG, prolonged QT / cardiac disease → avoid haloperidol–promethazine [NICE]. Lorazepam only if otherwise safe: respiratory depression / intoxication caution. Lower doses in frailty; separate child / pregnancy protocols.

Reassess before any repeat; check cumulative doses and local limits. Do not stack injections. IM olanzapine must not be combined with IM benzodiazepines [Maudsley]. Resuscitation capability must be available.

After injection · Maudsley 2025 monitoring example

Observe continuously in sight. Pulse, BP, temperature and respiratory rate every 15 min for 1 h, then hourly until ambulatory; monitor consciousness and oxygen saturation (continuous oximetry if asleep / unconscious). Respiratory depression, hypotension or arrhythmia → urgent medical response.

India · MHCA 2017 §97 · restriction, review and prevention

Seclusion / solitary confinement is prohibited. Restraint: only means to prevent imminent harm; psychiatrist authorisation, shortest duration, supervision and immediate records. Notify nominated representative within 24 h of EVERY instance; monthly Board report.

Never punishment or a response to staff shortage. Debrief and revise prevention / ongoing treatment.

Sources: Weave Q97 p132 (figure reviewed; seclusion and medication cautions corrected). IPS aggression CPG (2023), pp131–139. NICE NG10 (2015), §§1.3–1.4. Maudsley Prescribing Guidelines 15e (2025), pp68–69. Mental Healthcare Act 2017, §97, India Code. Doses are monitored adult initial examples; local protocol / individual assessment governs.

Q97 · emergency clinician revision · checked 17 Sep 2026 A4 · 300 dpi

Aggression assessment and management. Use the visual organisation to recall the key distinctions and structure your answer. Read with II-97.

Credits and references: retained within the illustration.

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.

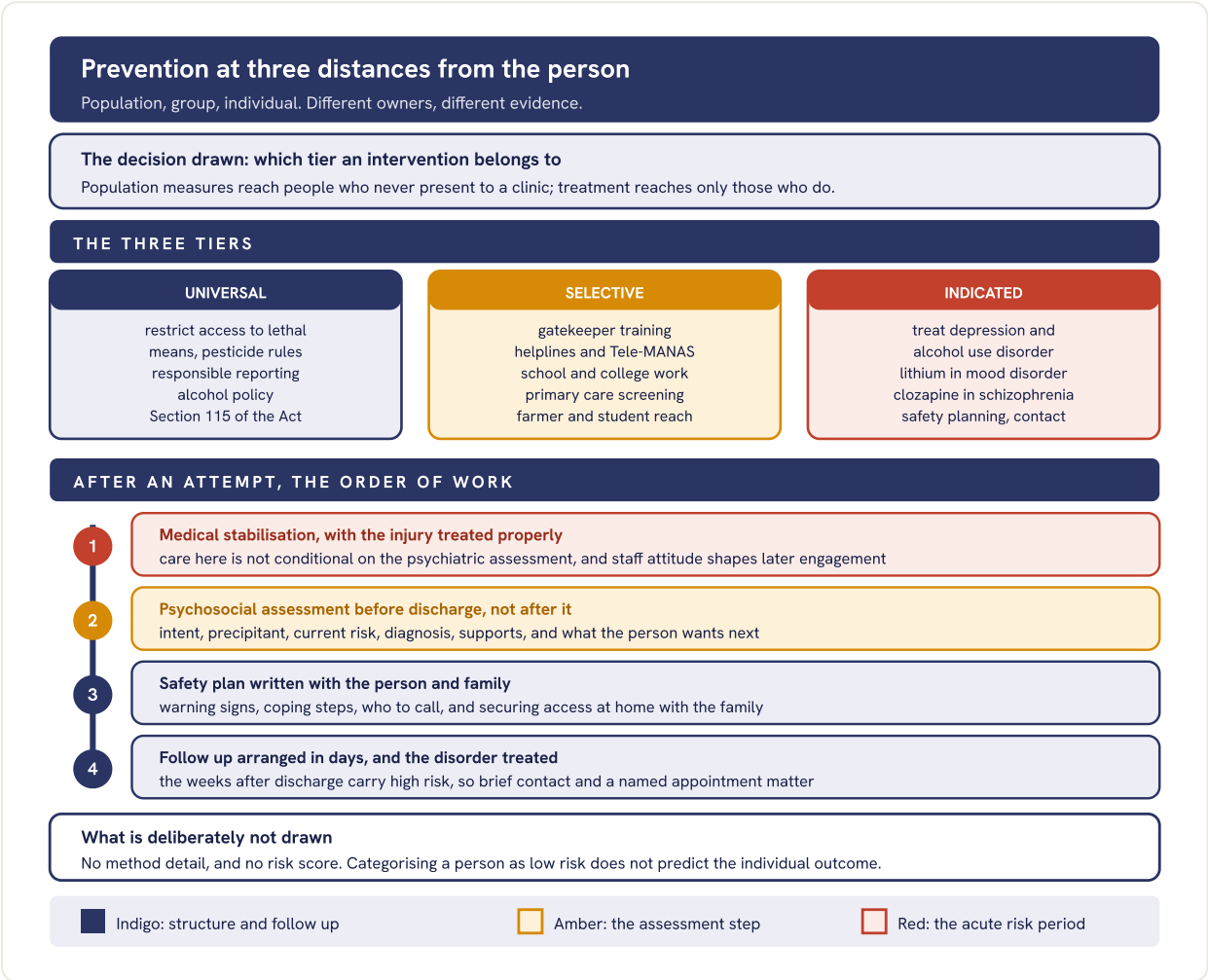


Fig 098.1 Prevention across three tiers, then the post-attempt sequence down a spine, with assessment before discharge, where it is most often lost.

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.

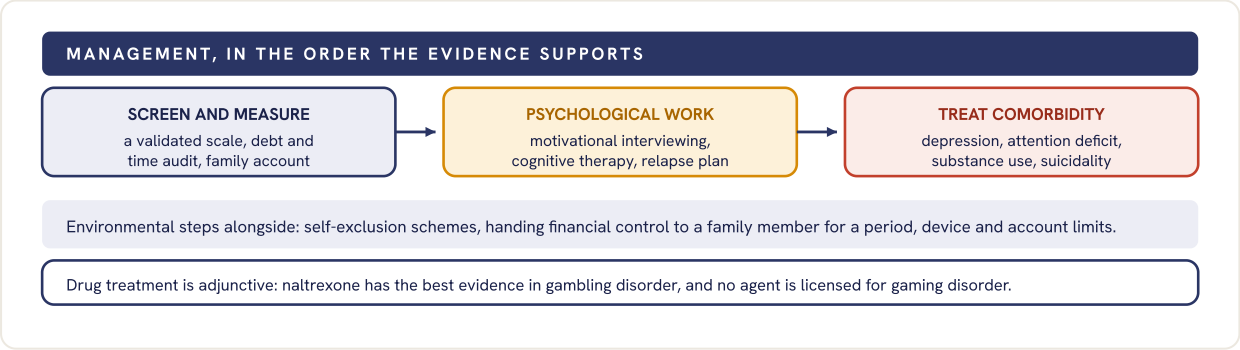


Fig 099.1 Screening, therapy and comorbidity run left to right, with environmental limits and the drug question carried as strips below.

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 203	II-20 p 63	II-92 p 257	II-31 p 94	II-40 p 111	II-83 p 220	II-16 p 50	II-18 p 55	II-71 p 169	II-94 p 260
II-S2	II-28 p 87	II-38 p 109	II-47 p 124	II-12 p 45	II-56 p 145	II-15 p 48	II-26 p 77	II-68 p 165	II-97 p 265	II-85 p 222
II-S3	II-52 p 135	II-91 p 255	II-54 p 139	II-48 p 125	II-86 p 236	II-75 p 208	II-62 p 154	II-17 p 53	II-98 p 267	II-09 p 29
II-S4	II-01 p 11	II-03 p 20	II-96 p 263	II-11 p 44	II-33 p 96	II-14 p 47	II-34 p 97	II-08 p 28	II-72 p 170	II-90 p 243
II-S5	II-29 p 91	II-53 p 138	II-22 p 67	II-10 p 41	II-61 p 153	II-06 p 26	II-35 p 99	II-51 p 129	II-84 p 221	II-89 p 242
II-S6	II-02 p 17	II-30 p 93	II-39 p 110	II-74 p 205	II-67 p 164	II-05 p 25	II-42 p 113	II-93 p 259	II-78 p 213	II-100 p 270
II-S7	II-36 p 104	II-45 p 121	II-46 p 122	II-55 p 143	II-04 p 23	II-58 p 147	II-07 p 27	II-77 p 211	II-87 p 239	II-99 p 269
II-S8	II-44 p 119	II-21 p 66	II-80 p 217	II-66 p 163	II-13 p 46	II-41 p 112	II-43 p 114	II-27 p 79	II-70 p 168	II-65 p 158
II-S9	II-19 p 61	II-60 p 151	II-23 p 68	II-32 p 95	II-25 p 75	II-82 p 219	II-63 p 156	II-76 p 209	II-88 p 241	II-95 p 261
II-S10	II-37 p 107	II-59 p 149	II-24 p 71	II-81 p 218	II-49 p 126	II-57 p 146	II-50 p 127	II-69 p 167	II-64 p 157	II-79 p 214

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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WEAVE SPRINT

This book tells you what to answer. Sprint teaches you the subject.

Weave 100 is a shortlist with model answers. Weave Sprint is the reading behind it: full chapters on every topic in every paper, with a colour memory code, a clickable contents and a complete index. The Sprint day is named at the foot of every map in this book.

4

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TAP OR SCAN TO OPEN

STUDY TOGETHER

A hundred questions is a shortlist, not a syllabus.

This counting started as a room full of residents comparing papers and arguing about what actually comes. That room is still open. Bring a question we missed, a paper we have not read, and the corrections. Every one of them makes the next count better.

Join the study group

https://chat.whatsapp.com/H7TsLGK2wFl4bSe2r0Zfzn?mode=gi_t

CREDITS

Who made this, and from what

Dr. Wilfred Dsouza	Counting, area weighting, selection, and the answer maps. Lead author of the Weave 100 series.
Dr. Niharika Reddy	Clinical review of the 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.

A frequency count is a strong prior, not a prophecy.

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