WEAVE SPRINT
PAPER II · VOLUME FOUR

*MD and DNB psychiatry, theory revision*

# Clinical Psychiatry

## *Anxiety, Trauma and Dissociation*

*Fear, obsession and the wound of trauma. Then the dissociative and conversion disorders, with the anxiolytic and hypnotic drugs that belong to them.*

**Dr. Wilfred D'souza · Dr. Niharika Reddy**

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WEAVE SPRINT · PAPER II

# Clinical Psychiatry

THE WEAVE SPRINT SERIES


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

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

ACKNOWLEDGEMENT

# A word of thanks

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

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

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

**Dr. Wilfred D'souza**

# Foreword

This fourth volume gathers the disorders in which fear is the engine. It moves from anxiety, obsession and trauma to the dissociative and conversion presentations, where the complaint arrives in the language of neurology and the examination is looking for what does not fit. It closes with the drugs given for distress, and the rules for not giving them for too long.

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

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

HOW TO USE THIS BOOK

# The colour memory code

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

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

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

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

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PART VII

# VII

# Anxiety, OCD and Trauma-Related Disorders

*Fear, obsession and the wound of trauma: the anxiety, obsessive-compulsive and stress-related disorders.*


01 Anxiety disorders

02 Obsessive-compulsive and related disorders

03 Trauma and stressor-related disorders

04 Apply and consolidate

PAPER II

# Anxiety, OCD and trauma-related disorders

Three sections . one complete notes document

This material gathers the anxiety, obsessive-compulsive and trauma-related disorders into one document, by syndrome and by treatment. The first band is **the anxiety disorders**: generalised anxiety disorder, panic disorder and Clark's cognitive model, agoraphobia, social anxiety disorder and the specific phobias, separation anxiety, the neurobiology of the fear circuit, the cognitive behaviour therapy of anxiety, and the differential diagnosis and medical mimics. The second band is **the obsessive-compulsive and related disorders**: obsessive-compulsive disorder and its symptom dimensions, the guideline-anchored management ladder, the Yale-Brown scale, the cortico-striato-thalamo-cortical circuit, clomipramine versus the serotonin reuptake inhibitors, exposure and response prevention, the treatment-resistant ladder to deep brain stimulation, and the related disorders from body dysmorphic disorder through hoarding, trichotillomania and excoriation to illness anxiety on the spectrum. The third band is **the trauma and stressor-related disorders**: post-traumatic stress disorder and its criteria, the evidence-based trauma-focused treatment, complex PTSD, the acute stress reaction and acute stress disorder, adjustment disorder, the neurobiology of fear conditioning, prolonged grief, and the attachment disorders of early neglect. The examinable skill is the safe, guideline-anchored diagnosis and treatment of each disorder: which disorder behind which fear, which scale and cut-off, which first-line agent for which anxiety disorder, the high-dose long-trial rule in obsessive-compulsive disorder, and the trauma-focused-psychotherapy-first algorithm in post-traumatic stress disorder.

**HOW TO USE THESE NOTES**

Read the three bands as one continuous account of how the anxiety, obsessive-compulsive and trauma-related disorders are diagnosed and treated. The **anxiety** band builds the frame: the individual anxiety disorders discriminated one from another, the fear-circuit neurobiology, the cognitive behaviour therapy owned here, and the medical mimics that must be excluded. The **obsessive-compulsive and related** band teaches obsessive-compulsive disorder in full, its cortico-striato-thalamo-cortical circuit, the Yale-Brown scale and the guideline-anchored management ladder (a high-dose serotonin reuptake inhibitor for a long trial, then clomipramine, then antipsychotic augmentation, then the refractory options), exposure and response prevention, and the related disorders across the spectrum. The **trauma and stressor** band teaches post-traumatic stress disorder and its trauma-focused treatment, complex PTSD and its distinction from a personality disorder, the acute stress and adjustment reactions, and the neurobiology of fear conditioning. Every management recommendation is guideline-anchored, led by the BAP guidelines with the Indian Psychiatric Society, NICE and CANMAT positions stated, on the where-to-treat, target-by-phase, modes-of-treatment spine; every named syndrome ends with a **Good to remember** box giving the criterion, the discriminator and the first step, and every named drug or psychotherapy ends with one giving the mechanism or principle, the signature caution and the one dose or first-line indication. Figures declare one axis and code it in colour: **petrol** marks structure and the neutral case, **coral** marks the trap or the danger, and **marigold** highlights the number to hold; the rating scales are reproduced as the actual instruments where the licence permits, credited to their sources, or typeset from their structure where it does not. Several boundaries are stated up front: the anxiolytic and hypnotic pharmacology is taught in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes and cross-referenced here; the deep antidepressant pharmacology in the Mood disorders: pharmacotherapy notes; the fear-circuit anatomy and receptor systems in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; deep brain stimulation and neurosurgery in the ECT and neurostimulation notes; the somatic-symptom and illness-anxiety boundary in the Somatic-symptom, sexual, impulse-control disorders and suicide notes; and the wider cognitive behaviour therapy technique in the Psychotherapies, clinical assessment, MSE and nosology notes. Exposure and response prevention, trauma-focused cognitive behaviour therapy, eye movement desensitisation and Clark's cognitive model of panic are owned and fully taught here. This is doctor-facing revision, so the full technical vocabulary is used, brand names lead with the Indian brand, and every dose, scale cut-off, criterion and first-line recommendation is verified against a source or omitted and flagged.

## Anxiety disorders

### 1 · Generalised anxiety disorder

Almost every anxious patient a psychiatrist sees carries the same engine under the bonnet: worry that will not switch off. **Generalised anxiety disorder** (GAD) is the disorder of chronic, **free-floating anxiety** and **excessive worry**, an **apprehensive expectation** spread across many domains of life rather than fixed to one object or situation, which the person finds hard to control and which drags a train of somatic and psychic symptoms behind it. Examiners like GAD because

it sits at the crossroads of the whole anxiety block: it is the free-floating baseline against which panic (episodic), phobia (cued) and obsessive-compulsive disorder (intrusive and ritualised) are contrasted, it carries the two scales you must know cold (the GAD-7 and the Hamilton Anxiety Rating Scale), and its management is the template first-line story, **SSRI first-line** with **pregabalin** and **buspirone** as options and the benzodiazepine held back, that repeats across the anxiety disorders.




**Fig 14.1** Anxiety disorders organise threat differently: diffuse future danger in generalised anxiety, abrupt bodily alarm in panic, social evaluation, specific objects or situations, and fear of difficult escape or unavailable help in agoraphobia.

**Why it matters, and the chapter mnemonic.** This topic seeds the master mnemonic for the whole anxiety block, **"WORRIED FEARS"**, unpacked in the mnemonic box below and paid off at consolidation. The amygdala and fear-circuit anatomy that underlies the sustained arousal is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and is not re-derived here; the deep pharmacology of the SSRIs and SNRIs lives in the Mood disorders: pharmacotherapy notes, and the anxiolytic story of pregabalin, buspirone and the benzodiazepines (with dependence and withdrawal) in the

Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. What this note owns is the diagnosis, the discrimination from normal worry and from the other anxiety disorders, the two scales and their cut-offs, and the guideline-anchored drug plan.

## GENERALIZED ANXIETY DISORDER 7-ITEM SCALE (GAD-7)

Spitzer RL, Kroenke K, Williams JBW, Lowe B, 2006

Over the **last 2 weeks**, how often have you been bothered by the following problems? *(Use the scale to the right to indicate your answer.)*

| Question | Not at all | Several days | More than half the days | Nearly every day |
|---|---|---|---|---|
| 1. Feeling nervous, anxious, or on edge | 0 | 1 | 2 | 3 |
| 2. Not being able to stop or control worrying | 0 | 1 | 2 | 3 |
| 3. Worrying too much about different things | 0 | 1 | 2 | 3 |
| 4. Trouble relaxing | 0 | 1 | 2 | 3 |
| 5. Being so restless that it is hard to sit still | 0 | 1 | 2 | 3 |
| 6. Becoming easily annoyed or irritable | 0 | 1 | 2 | 3 |
| 7. Feeling afraid, as if something awful might happen | 0 | 1 | 2 | 3 |

**Add columns (total):** Column totals summed give a total score of 0 to 21.

**Score interpretation:** 0-4 minimal anxiety; 5-9 mild anxiety; 10-14 moderate anxiety; 15-21 severe anxiety.

**Clinical threshold:** A score of 10 or greater is the common cut-off for probable generalized anxiety disorder (a cut-off of 8 or greater maximizes sensitivity and specificity).

If you checked off any problems, how **difficult** have these made it for you to do your work, take care of things at home, or get along with other people?

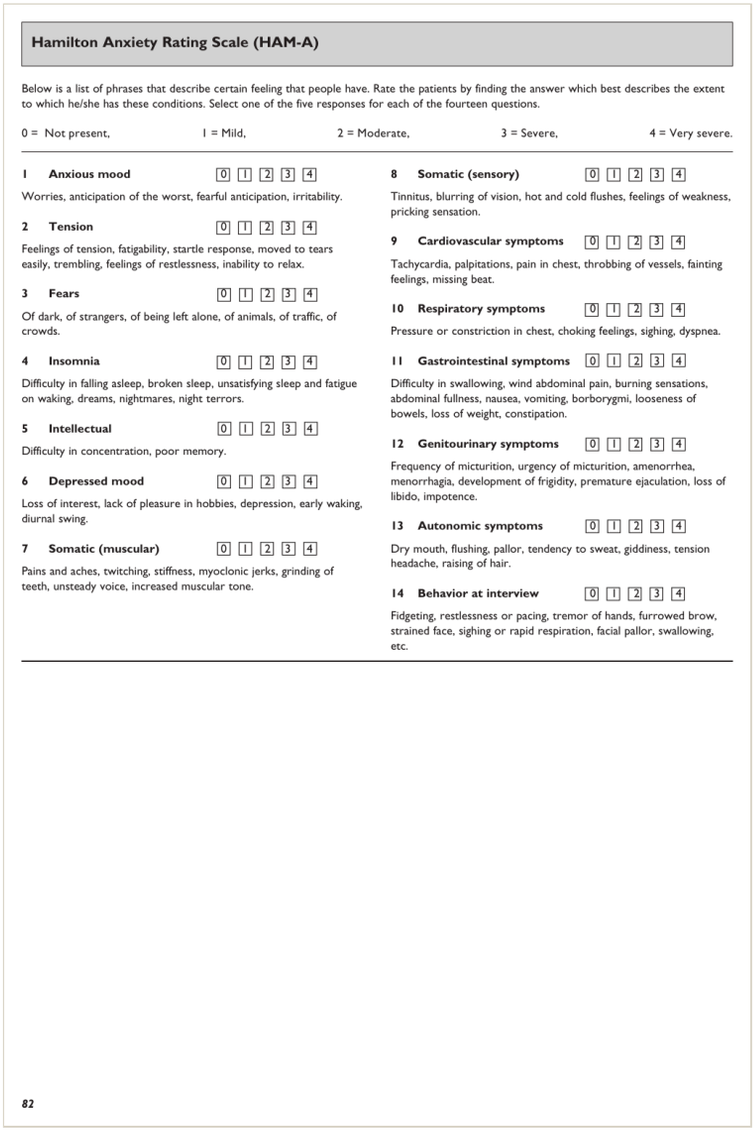
Not difficult at all □ Somewhat difficult □ Very difficult □ Extremely difficult □

**Source:** Spitzer RL, Kroenke K, Williams JBW, Lowe B. A brief measure for assessing generalized anxiety disorder: the GAD-7. Arch Intern Med. 2006;166(10):1092-1097.

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The GAD-7: 7 self-report items, each scored 0 to 3 over the last two weeks (total 0 to 21); the bands are 0 to 4 minimal, 5 to 9 mild, 10 to 14 moderate, 15 to 21 severe, and a score of 10 or more is the common cut-off for probable generalised anxiety disorder.

GAD-7 (Spitzer, Kroenke, Williams and Lowe, 2006); developed with an educational grant from Pfizer, no permission required to reproduce; free non-commercial educational use.



**Hamilton Anxiety Rating Scale (HAM-A)**

Below is a list of phrases that describe certain feeling that people have. Rate the patients by finding the answer which best describes the extent to which he/she has these conditions. Select one of the five responses for each of the fourteen questions.

0 = Not present, 1 = Mild, 2 = Moderate, 3 = Severe, 4 = Very severe.

**1 Anxious mood** [0] [1] [2] [3] [4]
Worries, anticipation of the worst, fearful anticipation, irritability.

**2 Tension** [0] [1] [2] [3] [4]
Feelings of tension, fatigability, startle response, moved to tears easily, trembling, feelings of restlessness, inability to relax.

**3 Fears** [0] [1] [2] [3] [4]
Of dark, of strangers, of being left alone, of animals, of traffic, of crowds.

**4 Insomnia** [0] [1] [2] [3] [4]
Difficulty in falling asleep, broken sleep, unsatisfying sleep and fatigue on waking, dreams, nightmares, night terrors.

**5 Intellectual** [0] [1] [2] [3] [4]
Difficulty in concentration, poor memory.

**6 Depressed mood** [0] [1] [2] [3] [4]
Loss of interest, lack of pleasure in hobbies, depression, early waking, diurnal swing.

**7 Somatic (muscular)** [0] [1] [2] [3] [4]
Pains and aches, twitching, stiffness, myoclonic jerks, grinding of teeth, unsteady voice, increased muscular tone.

**8 Somatic (sensory)** [0] [1] [2] [3] [4]
Tinnitus, blurring of vision, hot and cold flushes, feelings of weakness, pricking sensation.

**9 Cardiovascular symptoms** [0] [1] [2] [3] [4]
Tachycardia, palpitations, pain in chest, throbbing of vessels, fainting feelings, missing beat.

**10 Respiratory symptoms** [0] [1] [2] [3] [4]
Pressure or constriction in chest, choking feelings, sighing, dyspnea.

**11 Gastrointestinal symptoms** [0] [1] [2] [3] [4]
Difficulty in swallowing, wind abdominal pain, burning sensations, abdominal fullness, nausea, vomiting, borborygmi, looseness of bowels, loss of weight, constipation.

**12 Genitourinary symptoms** [0] [1] [2] [3] [4]
Frequency of micturition, urgency of micturition, amenorrhea, menorrhagia, development of frigidity, premature ejaculation, loss of libido, impotence.

**13 Autonomic symptoms** [0] [1] [2] [3] [4]
Dry mouth, flushing, pallor, tendency to sweat, giddiness, tension headache, raising of hair.

**14 Behavior at interview** [0] [1] [2] [3] [4]
Fidgeting, restlessness or pacing, tremor of hands, furrowed brow, strained face, sighing or rapid respiration, facial pallor, swallowing, etc.

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The Hamilton Anxiety Rating Scale (HAM-A): 14 clinician-rated items covering psychic and somatic anxiety, each scored 0 to 4 (total 0 to 56); commonly below 17 is mild, 18 to 24 mild to moderate and 25 to 30 moderate to severe (the bands vary by source).

HAM-A (Hamilton, 1959); in the public domain.

**Fig 14.2** The anxiety rating scales. The GAD-7 is the brief self-report screen for generalised anxiety, seven items each scored 0 to 3 for a total of 0 to 21, with 5, 10 and 15 marking mild, moderate and severe and a score of 10 or more the common threshold for probable generalised anxiety disorder. The Hamilton Anxiety Rating Scale is the clinician-rated severity measure, fourteen items spanning the psychic and the somatic symptoms of anxiety, each 0 to 4, for a total of 0 to 56. Both are shown here as their actual, freely reproducible forms; the GAD-7 keeps its original United States spelling.

## 1.1 The core concept: free-floating, excessive, hard-to-control worry

**What "generalised" means.** The defining quality is that the anxiety is *not tied to a specific trigger*. Unlike phobia (bound to a feared object) or panic disorder (episodic attacks), GAD is a persistent, diffuse free-floating anxiety, a general apprehensiveness "not restricted to any particular environmental circumstance" in the ICD-11 wording (ICD-11 CDDR). The cognitive core is excessive worry (apprehensive expectation) about multiple everyday domains, work, health, finances, the safety of family, minor chores, that is out of proportion to the actual likelihood or impact of the feared event.

**The control criterion.** The feature that separates GAD from the ordinary worry of a stressed but well person is that the patient *cannot control* the worry, cannot put it down to attend to the task in hand, and finds it pervasive, distressing and long-lasting. Everyday worry is manageable, focused and deferrable; GAD worry is uncontrollable, free-floating and self-perpetuating. This is the discriminator to lead with in any answer.

**GOOD TO REMEMBER**

- **Generalised anxiety disorder:** chronic, free-floating, excessive and uncontrollable worry (apprehensive expectation) spread across multiple life domains, not bound to a specific object or situation; the defining discriminator from normal worry is that it is excessive, pervasive and hard to control; the first diagnostic step is to exclude an organic mimic (hyperthyroidism, caffeine, substance withdrawal) before labelling it.

### 1.2 The symptom clusters: psychic worry plus a somatic tail

**Psychic and somatic together.** GAD is worry *with a body*. The psychic cluster is the apprehensive expectation itself, plus restlessness, feeling keyed up or on edge, difficulty concentrating or mind going blank, and irritability. The somatic cluster is dominated by **muscle tension** (with trembling, twitching, aches and soreness), fatigue and sleep disturbance, and a background of autonomic symptoms, sweating, nausea, gastrointestinal upset and an exaggerated startle. A teaching point examiners probe: overt autonomic hyperarousal (racing heart, breathlessness, dizziness) is *less* prominent in GAD than in panic disorder (Kaplan and Sadock 2024), because GAD is the sustained low-grade state rather than the acute surge.

**Muscle tension is the signature somatic marker.** Of the somatic features, muscle tension is the one most specific to GAD and the one most reliably present; it is the physical correlate of chronic vigilance and the symptom that best distinguishes GAD's body from the episodic autonomic storm of panic.

### 1.3 DSM-5-TR criteria: six months, three of six symptoms

**The criteria to state cold.** DSM-5-TR requires excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least **6 months**, about a number of events or activities; the person finds it difficult to control the worry; and the anxiety and worry are associated with **three or more of six** symptoms, more days than not for the past 6 months (DSM-5-TR 2022). The six are: restlessness or feeling keyed up or on edge; being easily fatigued; difficulty concentrating or mind going blank; irritability; muscle tension; and sleep disturbance. The disturbance must cause clinically significant distress or impairment, must not be attributable to a substance or another medical condition, and must not be better explained by another mental disorder.

**The paediatric relaxation.** A high-yield discriminator: in children, *only one* of the six associated symptoms is required, not three, reflecting that the worry-plus-body picture is thinner in a developing child.

| CRITERION | DSM-5-TR REQUIREMENT |
|---|---|
| **A. Worry** | **Excessive anxiety and worry (apprehensive expectation), more days than not for at least 6 months, about multiple events/activities** |
| **B. Control** | The individual finds it difficult to control the worry |
| **C. Symptoms** | **3 or more of 6: restlessness/keyed up, easily fatigued, poor concentration, irritability, muscle tension, sleep disturbance (only 1 required in children)** |
| **D. Impairment** | Clinically significant distress or social/occupational impairment |
| **E. Not organic** | Not due to a substance or another medical condition (e.g. hyperthyroidism) |
| **F. Not better explained** | Not better accounted for by another mental disorder (panic, social anxiety, OCD, PTSD, illness anxiety and others) |

DSM-5-TR criteria for generalised anxiety disorder; the two numbers to hold are the 6-month duration and 3-of-6 symptom threshold (only 1 in children) (DSM-5-TR 2022).

### 1.4 ICD-11 CDDR: free-floating anxiety and "at least several months"

**Where ICD-11 differs.** The ICD-11 CDDR frames GAD as either general apprehensiveness "not restricted to any particular environmental circumstance (free-floating anxiety)" or excessive worry (apprehensive expectation) about negative events across several domains of everyday life, accompanied by additional symptoms such as muscle tension, restlessness, difficulty relaxing, difficulty concentrating, irritability and sympathetic autonomic overactivity (ICD-11 CDDR). Crucially, ICD-11 does *not* impose the DSM's fixed 6-month clock: it requires the symptoms to be **not transient and to persist for at least several months, more days than not**. This softer, more clinically flexible duration is the classic ICD-11-versus-DSM-5 discriminator to name in a comparison answer.

**Onset and course.** ICD-11 notes the typical age of onset is the early-to-mid thirties (later than most anxiety disorders), that severity fluctuates between threshold and subthreshold forms, and that full remission is uncommon, GAD is chronic and relapsing, which is exactly why the guidelines argue for long continuation.

RULE **Duration is the ICD/DSM fork.** DSM-5-TR fixes GAD at 6 months more days than not with 3 of 6 symptoms; ICD-11 asks only for "at least several months" and does not set a hard 6-month threshold, so quote the framework you are asked about.

**GOOD TO REMEMBER**

- **ICD-11 GAD:** free-floating general apprehensiveness or excessive multi-domain worry plus muscle tension/autonomic and cognitive symptoms, not transient, persisting for at least several months more days than not; the defining contrast with DSM-5-TR is the absence of a fixed 6-month rule; typical onset is the early-to-mid thirties and full remission is uncommon.

### 1.5 The differential: excluding organic mimics and the other anxiety disorders

**Rule out the body first.** Before diagnosing GAD, exclude an organic cause of chronic anxiety: hyperthyroidism, phaeochromocytoma, hypoglycaemia, cardiac arrhythmia, and above all caffeine and substance withdrawal (alcohol and benzodiazepine withdrawal both produce a GAD-like picture). A first presentation of "anxiety" in an older patient, or one with prominent physical signs, warrants thyroid function and a substance history rather than a straight-to-SSRI reflex.

**Then separate it from its neighbours.** GAD is the free-floating baseline; the specific anxiety disorders are carved out by the *focus* of the fear, panic disorder (fear of the attacks themselves), social anxiety disorder (fear of negative evaluation), OCD (contamination or other obsessions), PTSD (trauma reminders), illness anxiety disorder (fear of serious illness), separation anxiety (separation from attachment figures). DSM criterion F requires that GAD not be better explained by any of these. Depression is the commonest comorbidity and can be hard to separate: in a depressive episode the worry is coloured by hopelessness and worthlessness, whereas GAD worry is future-oriented apprehension.

**COMMON TRAP**

Do not label chronic anxiety as GAD without excluding a medical mimic. Hyperthyroidism, a caffeine habit, and alcohol or benzodiazepine withdrawal each reproduce restlessness, tremor, poor sleep and autonomic arousal; missing them means treating the wrong disorder. Thyroid function, a substance and caffeine history, and attention to withdrawal timing come before the prescription.

### 1.6 The GAD-7: the screening and severity scale to know cold

**What it is.** The GAD-7 (Generalised Anxiety Disorder-7) is a seven-item self-report scale of general worry and fear symptoms over the last 2 weeks, each item scored 0 to 3 on a Likert scale (not at all, several days, more than half the days, nearly every day), giving a total range of **0 to 21** (Spitzer et al 2006; Kaplan and Sadock 2024). It is the workhorse instrument of primary-care anxiety screening and of tracking treatment response, and it is reproduced in full on the accompanying scale plate.

**The cut-offs.** The suggested severity bands are **5, 10 and 15** for mild, moderate and severe anxiety respectively. The single most useful clinical threshold to memorise is a score of **10**, the usual cut-off for probable GAD and the point at which further assessment is warranted. A two-item short form, the GAD-2, is used as a rapid case-finding question; NICE recommends being alert to GAD and considering the GAD-2 to ask about anxiety and the ability to control worry (NICE 2011). A caveat examiners like: the GAD-7 asks about the last 2 weeks, whereas the DSM diagnosis needs symptoms over 6 months, so a high GAD-7 supports but does not by itself make the diagnosis.

**GOOD TO REMEMBER**

- **GAD-7:** seven self-rated items over 2 weeks, each 0 to 3, total 0 to 21; severity bands at 5/10/15 for mild/moderate/severe; a score of 10 is the standard clinical cut-off for probable GAD; the 2-item GAD-2 is the rapid screen; it measures 2-week severity and screens, it does not replace the 6-month DSM diagnosis.

### 1.7 The Hamilton Anxiety Rating Scale (HAM-A): the clinician-rated outcome measure

**What it is.** The Hamilton Anxiety Rating Scale (HAM-A, also HARS) is a *clinician-rated* scale of 14 items, each scored 0 to 4, covering both the psychic and the somatic components of anxiety, for a maximum total of **56** (Maudsley Deprescribing Guidelines). It is the standard anxiety-severity and treatment-outcome measure in trials, the scale against which drug effects in GAD are reported, and it too is set out on the accompanying scale plate. Where the GAD-7 is a quick self-report screen, the HAM-A is the fuller clinician instrument used to quantify change.

**Reading it.** The 14 items split into a psychic-anxiety factor (anxious mood, tension, fears, insomnia, cognitive difficulty, depressed mood) and a somatic-anxiety factor (muscular, sensory, cardiovascular, respiratory, gastrointestinal, genitourinary, autonomic), so the profile shows which arm of the disorder dominates. Commonly used severity bands place mild anxiety below the high-teens and increasing severity above the mid-twenties, though the exact numeric cut-points vary by source and are not standardised the way the GAD-7 bands are, so quote the structure (14 items, max 56) with confidence and the precise band figures with appropriate caution.

**GOOD TO REMEMBER**

- **HAM-A (HARS):** a clinician-rated 14-item scale, each item 0 to 4, maximum 56, splitting into psychic-anxiety and somatic-anxiety factors; it is the standard outcome measure in GAD drug trials (drug effects are reported as points reduced on the HAM-A); contrast with the GAD-7, which is self-rated, 7 items and used to screen.

| 10 | 0 to 21 | 56 | 6 months |
|---|---|---|---|
| GAD-7 CLINICAL CUT-OFF FOR PROBABLE GAD (BANDS AT 5/10/15) | GAD-7 TOTAL SCORE RANGE (7 ITEMS, EACH 0 TO 3) | HAM-A MAXIMUM (14 ITEMS, EACH 0 TO 4) | DSM-5-TR DURATION; 3 OF 6 SYMPTOMS |

### 1.8 Management: SSRI or SNRI first-line, across the guidelines

**The BAP position.** Lead with the British Association for Psychopharmacology: an SSRI is the first-line pharmacological treatment for GAD, the efficacious agents named being citalopram, escitalopram, paroxetine and sertraline; if an SSRI is unsuitable, an SNRI (duloxetine or venlafaxine) or pregabalin is the alternative initial treatment, and higher pregabalin doses may achieve greater response (BAP 2014). This is the verified **SSRI first-line** anchor and must not be contradicted.

**What the other guidelines say.** The picture is strikingly consistent. The Indian Psychiatric Society CPG names cognitive behavioural therapy as the first-line psychological treatment (the Indian Psychiatric Society 2020), sitting alongside the same pharmacology. NICE offers an SSRI if drug treatment is chosen, considering sertraline first as the most cost-effective option, then an alternative SSRI or an SNRI, and pregabalin only if SSRIs and SNRIs cannot be tolerated (NICE 2011). CANMAT lists SSRIs (escitalopram, paroxetine, sertraline), SNRIs (duloxetine, venlafaxine XR), agomelatine and pregabalin as first-line, with CBT as a first-line psychological treatment of comparable magnitude (CANMAT 2014). The WFSBP likewise puts an SSRI or SNRI first-line with CBT as first-line psychotherapy (WFSBP 2022). The convergence is the exam point: *SSRI or SNRI first-line everywhere.*

| GUIDELINE | FIRST-LINE PHARMACOLOGY | KEY NOTE |
|---|---|---|
| **BAP 2014** | **SSRI (citalopram, escitalopram, paroxetine, sertraline)** | SNRI or pregabalin if SSRI unsuitable |
| **Indian Psychiatric Society 2020** | SSRI/SNRI, with CBT first-line psychological | CBT named as first-line therapy |
| **NICE 2011** | **SSRI, consider sertraline first (most cost-effective)** | Pregabalin only if SSRI/SNRI not tolerated |
| **CANMAT 2014** | SSRI, SNRI, agomelatine or pregabalin | CBT equal first-line to drugs |

First-line pharmacotherapy for GAD across the major guidelines; the shared message is SSRI or SNRI first, with pregabalin as an option and CBT a co-equal first-line psychological treatment.

RULE **SSRI or SNRI is first-line for GAD, full stop.** BAP, NICE, CANMAT, WFSBP and the Indian Psychiatric Society all put an SSRI (or SNRI) first, with pregabalin and buspirone as options; CBT is the co-equal first-line psychological treatment.

### 1.9 The options and the second line: pregabalin, buspirone and the trial rules

**Pregabalin.** Pregabalin (Indian brands Pregeb, Pregastar) is a calcium-channel alpha-2-delta ligand with a genuinely anxiolytic effect and a relatively rapid onset. It is an alternative initial treatment when SSRIs are unsuitable (BAP 2014) and an augmentation option after non-response to an SSRI or SNRI. Its advantages are that it is renally cleared (useful in hepatic impairment) and fast-acting; its cautions are weight gain in about a fifth of long-term users, discontinuation symptoms on abrupt withdrawal, and a real potential for misuse, which is why NICE requires a drug-abuse history to be evaluated before prescribing (NICE 2011).

**Buspirone.** Buspirone (Indian brand Buspin) is a 5-HT1A partial agonist, efficacious in the acute treatment of GAD, non-sedating and non-dependence-forming, which makes it a useful option, particularly where a benzodiazepine is being avoided. Its limitations are a slow onset (a couple of weeks, so no use as a rescue drug) and no evidence for relapse prevention.

**The adequate trial.** A shared rule: allow up to **12 weeks** to judge efficacy, but recognise that absence of any benefit by **4 weeks** signals that response to unchanged treatment is unlikely (BAP 2014). SSRIs and SNRIs are often started at half the usual dose in GAD and titrated up, because early activation is common. When a patient responds, continuation of at least a further 6 to 18 months (BAP: up to 18 further months; NICE: at least a year) reduces relapse.

GOOD TO REMEMBER

- **Pregabalin (Pregeb, Pregastar):** an alpha-2-delta calcium-channel ligand; alternative first-line or augmentation option in GAD, relatively rapid-acting and renally cleared; signature cautions are weight gain, discontinuation symptoms and misuse potential (check a drug-abuse history first).
- **Buspirone (Buspin):** a 5-HT1A partial agonist; non-sedating, non-dependence-forming option for acute GAD; signature caution is its slow onset (about 2 weeks, so not a rescue agent) and no relapse-prevention evidence.

### 1.10 The benzodiazepine: not first-line, and not for the long term

**The hard rule.** A benzodiazepine is explicitly *not* a first-line treatment for GAD and must not be used long-term. BAP reserves benzodiazepines only after non-response to an SSRI, SNRI, pregabalin and buspirone, for persistent, severe, distressing, impairing anxiety where other treatments have failed (BAP 2014). NICE is blunter: do not offer a benzodiazepine for GAD except as a short-term measure during a crisis, and no more than 2 to 4 weeks (NICE 2011). The reason is the class's tolerance, dependence and withdrawal liability, the full pharmacology of which is developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes: a meta-analysis shows the HAM-A advantage of benzodiazepines over placebo shrinks from about 8 points at 1 week to almost nothing by 3 months (Maudsley Deprescribing Guidelines).

**Propranolol.** The beta-blocker propranolol (Indian brands Ciplar, Inderal) blunts the peripheral autonomic symptoms (tremor, palpitations) and is useful for *performance* anxiety, but it is not a treatment for GAD itself and is explicitly not recommended for its long-term management (MOH Singapore 2015).

TRAP **Never give a benzodiazepine as long-term monotherapy for GAD.** It is not first-line, is limited to short crisis use (2 to 4 weeks), and its apparent benefit fades within weeks as tolerance and dependence develop; the exam-fatal error is to write it up as the maintenance answer.

### 1.11 The India lens: the treatment gap and the benzodiazepine problem

**Two genuine Indian realities.** First, most people with GAD in India never reach formal care, part of the very large anxiety-disorder treatment gap; anxiety is under-recognised, somatised (patients present with physical complaints rather than "worry"), and under-treated. Second, and connected, is the entrenched over-prescription of benzodiazepines: in Indian general and primary care, a benzodiazepine (often alprazolam) is frequently the default and only "anxiety" prescription, given long-term against every guideline. The Indian Psychiatric Society CPGs exist precisely to correct this, an SSRI or SNRI first-line with CBT, the benzodiazepine reserved and time-limited, and the anxiolytic used only as a short bridge while the antidepressant takes effect (the Indian Psychiatric Society 2020).

**Prescribing in Indian practice.** The first-line agents are cheap and familiar as Indian brands: escitalopram (Nexito, Rexipra) and sertraline lead, with pregabalin (Pregeb, Pregastar) and buspirone (Buspin) as options and propranolol (Ciplar, Inderal) for the performance-anxiety niche. Starting the SSRI low and warning the patient about early activation, then holding the course to 12 weeks before judging failure, is the practical antidote to the "one alprazolam strip forever" pattern.

**INDIA**

The single highest-yield Indian point on this topic is the benzodiazepine over-prescription problem. Anxiety is common, somatised and under-recognised, and the treatment gap is wide; into that gap has flowed long-term benzodiazepine (typically alprazolam) prescribing that runs directly against the Indian Psychiatric Society CPG. The correct Indian answer mirrors the global one: an SSRI or SNRI first-line (Nexito or Rexipra escitalopram, sertraline) with CBT, pregabalin (Pregeb, Pregastar) or buspirone (Buspin) as options, and the benzodiazepine only as a short, time-limited bridge, never the maintenance treatment.

**MNEMONIC**

### WORRIED FEARS

The chapter mnemonic, seeded here and paid off at consolidation. **WORRIED** holds the GAD picture: **W**orry (free-floating, uncontrollable), **O**n edge/restless, **R**umination and poor concentration, **R**elaxation impossible (muscle tension), **I**rritability, **E**asily fatigued, **D**isturbed sleep. **FEARS** holds the management: **F**irst-line SSRI/SNRI, **E**valuate for 12 weeks (4-week early-failure warning), **A**lternatives pregabalin and buspirone, **R**efer/CBT co-equal first-line, **S**hun the benzodiazepine as long-term monotherapy.

**HOLD THIS**

GAD is chronic, free-floating, uncontrollable worry across many domains, requiring 6 months and 3 of 6 symptoms in DSM-5-TR (only "several months" in ICD-11); the GAD-7 (self-rated, 0 to 21, cut-off 10) and HAM-A (clinician-rated, 14 items, max 56) are the scales; management is an SSRI or SNRI first-line (up to 12 weeks to judge), with pregabalin, buspirone and CBT as options, propranolol for performance anxiety, and the benzodiazepine never a long-term monotherapy.

## 2 · Panic disorder

**Panic disorder** is the disorder of the false alarm. A surge of autonomic terror arrives out of a calm sky, peaks in minutes, and the patient, feeling their heart pound and their breath shorten, concludes they are dying, suffocating or losing their mind. That misreading is the engine of the illness: the body's fear response is real, but the interpretation is catastrophic, and the fear of the next attack becomes a second illness layered on the first. Examiners test **panic disorder** on a tight set of exact points, the definition of a **panic attack** (an abrupt surge peaking within minutes, four or more of thirteen symptoms), the requirement of **recurrent** *unexpected* attacks plus a month of **anticipatory anxiety** or behaviour change, the medical mimics that must be excluded, and the management, where the one thing a wrong answer fails is starting an antidepressant without warning about early activation.

**Why it matters clinically.** This is a management/drug topic, so the marks sit in the drug, the dose, the line and the monitoring as much as in the phenomenology. Two facts anchor every answer. First, the diagnostic core is the *unexpected*, cued-by-nothing attack followed by persistent worry, which separates panic disorder from the situationally-bound attacks of the phobias. Second, the treatment core is that an SSRI is first-line but must be started low and slow with an

explicit warning, because these patients are exquisitely sensitive to the transient jitteriness of an antidepressant and will read it as another attack and stop the drug. The fear-circuit anatomy on which the alarm is built is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes, the wider CBT model in the Psychotherapies, clinical assessment, MSE and nosology notes, and the deep SSRI pharmacology in the Mood disorders: pharmacotherapy notes; they are not re-derived here.

### 2.1 What a panic attack is: an abrupt surge that peaks within minutes

**The unit of the disorder.** A panic attack is an abrupt surge of intense fear or intense discomfort that reaches a peak *within minutes*, during which four or more of a list of thirteen physical and cognitive symptoms occur (DSM-5-TR 2022). The word to hold is *abrupt*: the surge can rise from a calm state or an already-anxious state, and it crescendos fast rather than building over hours. Fewer than four symptoms is a **limited-symptom attack**, which still counts clinically but not as a full attack.

**The thirteen symptoms.** The list divides into autonomic and cognitive. The autonomic and somatic symptoms are palpitations or pounding heart, sweating, trembling, shortness of breath or smothering, feelings of choking, chest pain, nausea or abdominal distress, dizziness or faintness, chills or heat sensations, and paraesthesiae. The cognitive symptoms are the three that give panic its dread: derealisation or depersonalisation, fear of losing control or "going crazy", and fear of dying (DSM-5-TR 2022). It is the cognitive triad that turns a burst of arousal into terror.

> **WORKED EXAMPLE**
>
> A 28-year-old software engineer is brought to casualty at midnight convinced he is having a heart attack: chest tightness, a racing heart, tingling in both hands, a sense that the room is unreal and that he is about to die. It began abruptly while he was watching television and peaked in about five minutes. ECG, troponin and examination are normal. This is a full panic attack (well over four symptoms, including the cognitive fear-of-dying and derealisation), arising unexpectedly from a calm state. If this is his third such attack and he now avoids being alone at night for fear of another, the diagnosis is panic disorder, not cardiac disease.

### 2.2 Recurrent unexpected attacks: the diagnostic spine

**Recurrent and unexpected.** **Panic disorder** requires **recurrent** *unexpected* panic attacks (Criterion A) (DSM-5-TR 2022). *Recurrent* means more than one; *unexpected* means the attack comes with no obvious cue at the time it occurs, as when a person is relaxing or is woken from sleep by a **nocturnal panic attack**. This is the single discriminator that examiners protect: an attack triggered reliably by a feared situation is an *expected* attack and points to a phobia, not to panic disorder. Roughly half of patients with panic disorder also have expected attacks, so the presence of expected attacks does not rule the diagnosis out, but at least some attacks must come out of the blue.

**The one-month tail.** Criterion B requires that at least one attack has been followed by a month or more of one or both of: persistent concern or worry about further attacks or their consequences (the **anticipatory anxiety**), or a significant maladaptive change in behaviour

because of the attacks, such as avoiding exercise or unfamiliar places (DSM-5-TR 2022). Without this month-long tail there are panic attacks but not the disorder.

RULE **Unexpected attack plus a month of worry.** Panic disorder needs *recurrent unexpected* panic attacks (out of the blue, not situationally cued) plus at least one month of anticipatory worry or maladaptive avoidance; a situationally-triggered attack is a phobia's attack, not this diagnosis.

### 2.3 Anticipatory anxiety, catastrophic misinterpretation and the panic cycle

**The cognitive engine.** The dominant cognitive model, Clark's, holds that **panic disorder** arises from the **catastrophic misinterpretation** of benign bodily sensations: a normal palpitation is read as an impending heart attack, breathlessness as suffocation, dizziness as impending collapse or madness (Tasman Psychiatry; New Oxford Textbook of Psychiatry). The misinterpretation amplifies the very sensations it fears, which are then read as further proof of catastrophe, closing a vicious loop, the **panic cycle**, that drives the attack to its peak. This is why interoceptive exposure, deliberately inducing the feared sensations, is a core ingredient of panic-focused CBT: it disconfirms the catastrophic prediction.

**Anticipatory anxiety and phobic avoidance.** Between attacks the patient is left with **anticipatory anxiety**, a persistent apprehensive dread of the next attack, and often with *fear of the fear* itself. This can generalise into agoraphobia, avoidance of situations from which escape would be difficult or help unavailable if an attack struck, so that a third illness (avoidance) stacks on the anticipatory anxiety and the attacks. The three layers, the attack, the anticipatory anxiety, and the avoidance, are what a full answer names.

PEARL

Panic disorder is really three problems in a stack: the *panic attack* (the false autonomic alarm), the *anticipatory anxiety* (fear of the next alarm, the "fear of fear"), and the *phobic avoidance* that can crystallise into agoraphobia. The catastrophic misinterpretation of body sensations is the hinge that converts a burst of arousal into terror and keeps the cycle turning. Treatment must reach all three layers, which is why an SSRI (dampens the attacks) plus CBT (breaks the misinterpretation and the avoidance) is the fullest answer.

### 2.4 The mimics you must exclude before you diagnose

**Panic is a diagnosis of exclusion of the organic.** Criterion C requires that the disturbance is not due to a substance or another medical condition, and Criterion D that it is not better explained by another mental disorder (DSM-5-TR 2022). The medical mimics to actively exclude are **hyperthyroidism**, **hyperparathyroidism**, **phaeochromocytoma**, vestibular dysfunction, seizure disorders, and cardiopulmonary conditions such as arrhythmias, supraventricular tachycardia, asthma and COPD (DSM-5-TR 2022). Substance causes, intoxication with stimulants (cocaine, amphetamines, caffeine) or cannabis and withdrawal from alcohol or barbiturates, must also be considered.

**The red flags that push you toward the organic.** First onset after 45 years, or atypical features during the attack, vertigo, loss of consciousness, loss of bladder or bowel control, slurred speech

or amnesia, should make you suspect a medical or substance cause rather than primary panic disorder (DSM-5-TR 2022). A first "panic attack" in an older patient is a cardiac or endocrine event until proven otherwise.

TRAP **Missing the organic mimic.** A panic attack is a diagnosis of exclusion: hyperthyroidism, phaeochromocytoma, hypoglycaemia, an arrhythmia or supraventricular tachycardia, and stimulant or caffeine excess all mimic it. First onset after 45, or vertigo, syncope, incontinence, slurred speech or amnesia during the attack, are red flags for a medical cause, do not label them panic.

### 2.5 ICD-11 and the criterion-anchored discriminators

**ICD-11 CDDR.** ICD-11 places panic disorder (6B01) among the anxiety and fear-related disorders and, like DSM-5-TR, requires recurrent unexpected panic attacks that are not restricted to a particular stimulus or situation, together with persistent concern about their recurrence or their significance, or behaviours intended to avoid recurrence (ICD-11 CDDR). The systems align on the essentials: it is the *unexpected, uncued* attack plus the persistent apprehension that defines the disorder.

**The discriminator to state.** If panic attacks occur *only* in a specific context, in social situations (social anxiety disorder), before phobic objects (specific phobia or agoraphobia), triggered by obsessions (OCD), by trauma reminders (PTSD), or by separation (separation anxiety disorder), then only that disorder is diagnosed and not panic disorder (DSM-5-TR 2022). Panic disorder is the diagnosis when the attacks are, at least in part, cued by nothing at all.

GOOD TO REMEMBER

- **Panic disorder (the syndrome):** the defining criterion is *recurrent unexpected* panic attacks (an abrupt surge peaking within minutes, four or more of thirteen autonomic and cognitive symptoms) followed by at least one month of persistent anticipatory worry about further attacks or a maladaptive behaviour change; the discriminator from the phobias is that at least some attacks are *uncued*, and the first step before diagnosis is to exclude the organic mimics (hyperthyroidism, phaeochromocytoma, arrhythmia, stimulant/caffeine excess), especially with first onset after 45 or atypical neurological features.

### 2.6 Management overview: SSRI first-line, CBT, and where the guidelines converge

**Lead with BAP.** The British Association for Psychopharmacology (BAP) makes an **SSRI** the first-line pharmacological treatment for panic disorder (escitalopram, citalopram, fluoxetine, fluvoxamine, paroxetine or sertraline), and cognitive behaviour therapy the first-line psychological treatment, noting that drug and psychological approaches have broadly similar efficacy; for partial or non-response it recommends combining cognitive therapy with an antidepressant, which is more effective than drug treatment alone and may protect better against relapse (BAP 2014). This is the verified first-line position and anchors any answer: an SSRI or CBT, chosen by availability and patient preference, with the combination reserved for the incomplete responder.

**Then the other bodies.** The Indian Psychiatric Society clinical practice guideline makes SSRIs or SNRIs the first-line agents for the long-term management of panic disorder and recommends

CBT (psychoeducation, self-monitoring, systematic exposure to panic-inducing cues, countering anxious beliefs, relapse prevention), adding that a combination of CBT and medication gives the best outcomes; in the elderly it prefers SSRIs as safer, avoiding TCAs and benzodiazepines because of anticholinergic effects, orthostatic hypotension, ECG changes and paradoxical reactions (IPS 2017). NICE offers an SSRI licensed for panic (escitalopram, sertraline, citalopram, paroxetine) or venlafaxine as the first pharmacological option, offers CBT of 7 to 14 hours over four months as first-line, and explicitly says *do not* prescribe benzodiazepines, sedating antihistamines or antipsychotics for panic disorder (NICE 2011). CANMAT likewise places an SSRI or venlafaxine XR and CBT (with interoceptive and situational exposure) as first-line (CANMAT 2014). WFSBP grades the SSRIs and venlafaxine as first-line (Recommendation Grade 1) at **20 to 60 mg/day** (WFSBP 2008).

| GUIDELINE | FIRST-LINE PHARMACOLOGY | PSYCHOTHERAPY | BENZODIAZEPINE STANCE |
|---|---|---|---|
| **BAP 2014** | SSRI (escitalopram, citalopram, fluoxetine, fluvoxamine, paroxetine, sertraline) | CBT first-line; combine with antidepressant for partial/non-response | Not first-line |
| **Indian Psychiatric Society 2017** | SSRI or SNRI first-line for long-term management | CBT; CBT plus medication gives best outcomes | Short-acting oral, only for the acute attack with reassurance |
| **NICE 2011** | SSRI licensed for panic (escitalopram, sertraline, citalopram, paroxetine) or venlafaxine | CBT 7 to 14 hours over 4 months, first-line | **Do not prescribe (worse long-term outcome)** |
| **CANMAT 2014** | SSRI or venlafaxine XR, first-line | CBT with interoceptive and situational exposure, first-line | Second-line only |
| **WFSBP 2008** | SSRI or venlafaxine, first-line, 20 to 60 mg/day | CBT alone or combined with medication | Reserve for treatment-resistant, no substance-use history |

Guideline-level management of panic disorder; BAP-led, then the Indian Psychiatric Society, NICE, CANMAT and WFSBP, all converging on SSRI/SNRI plus CBT first-line and benzodiazepines out of the first line (verified from the drug-guideline supplement).

### 2.7 Starting the SSRI: start low, go slow, and warn about early activation

**The single most examinable prescribing point.** An **ssri panic** start must be at a *low* dose, lower than the usual antidepressant starting dose, and titrated slowly, because patients with panic disorder are unusually sensitive to the transient over-stimulation an antidepressant produces in the first days and weeks, the jitteriness syndrome of restlessness, insomnia, agitation and a paradoxical worsening of anxiety (Pohl et al., in Tasman Psychiatry; Maudsley Prescribing Guidelines; Stahl). Left unwarned, the patient reads this early activation as another panic attack, concludes the drug is harming them, and stops it. The instruction to memorise is therefore: *start low, go slow, and warn the patient in advance* that they may feel briefly more jittery before they feel better, that this is expected and self-limiting, and that it is not a sign of harm.

**How this looks in practice.** Begin, for example, sertraline at 25 mg/day rather than 50, or escitalopram at 5 mg/day, titrating upward after several days once early activation has settled; a short course of a benzodiazepine may be co-prescribed to cover the activation window in selected patients, then withdrawn (WFSBP 2008; MOH_SG 2015). The therapeutic effect is delayed, several weeks, so the patient must also be told the drug is not a rescue medication for an attack in progress but a preventive taken daily (NICE 2011).

TRAP **Starting the SSRI at a full dose with no warning.** Panic patients are hypersensitive to early SSRI activation (the jitteriness syndrome); a normal starting dose can trigger restlessness and a paradoxical surge of anxiety that the patient reads as another attack and stops the drug. Start at half the usual dose, titrate slowly, and warn in advance that a brief jittery phase is expected and self-limiting.

### 2.8 Dose, duration and stopping: the numbers to hold

**Target dose and trial length.** Once started low, the SSRI is titrated to the effective antidepressant range; WFSBP puts the first-line SSRI and venlafaxine range at **20 to 60 mg/day** (citalopram-equivalent), and panic disorder, like the other anxiety disorders, often needs the *upper* part of the range and an adequate trial before response is judged (WFSBP 2008; NICE 2011). NICE frames the antidepressant trial around a **12-week** course: if there is no improvement after a 12-week course of an SSRI, and only then, imipramine or clomipramine may be considered off-label as the next step (NICE 2011).

**Continuation and the taper.** After response, drug treatment is continued for *at least six months*, and when stopping the dose is reduced gradually over an extended period, a minimum of three months, to avoid discontinuation symptoms (BAP 2014). The relapse rate on abrupt withdrawal is high, so the slow taper is part of the treatment, not an afterthought. Patients should be told of both the delayed onset and the discontinuation risk from the outset, and monitored with a brief self-report panic-severity questionnaire (NICE 2011).

| STEP | ACTION | SOURCE |
|---|---|---|
| **Start** | **SSRI at half the usual dose (e.g. sertraline 25 mg/day, escitalopram 5 mg/day); warn about early activation** | Maudsley; Stahl |
| **Titrate** | Up to the effective range, 20 to 60 mg/day, often the upper end | WFSBP 2008 |
| **Judge response** | **Adequate trial; if no improvement after a 12-week course, consider imipramine or clomipramine (off-label)** | NICE 2011 |
| **Continue** | At least six months after response | BAP 2014 |
| **Stop** | **Taper gradually over a minimum of three months** | BAP 2014 |

The SSRI course in panic disorder: start low and warn, titrate to range, judge at 12 weeks, continue at least six months, taper over at least three (BAP 2014; NICE 2011; WFSBP 2008).

**GOOD TO REMEMBER**

- **SSRI in panic disorder (fluoxetine, sertraline, fluvoxamine, paroxetine, escitalopram; the antidepressant brand list applies):** the first-line agent across BAP, IPS, NICE, CANMAT and WFSBP; mechanism is serotonergic dampening of the fear circuit, the signature caution is early *activation* (the jitteriness syndrome), so *start at half the usual dose, titrate slowly and warn the patient in advance*; effective range 20 to 60 mg/day, judge response over a 12-week course, continue at least six months, and taper over a minimum of three months to avoid discontinuation.

### 2.9 CBT for panic: interoceptive exposure and the Clark model in practice

**What panic-focused CBT does.** CBT is first-line and, delivered as a structured 7-to-14-hour course over about four months, targets each layer of the disorder (NICE 2011; BAP 2014). Its ingredients are psychoeducation about the panic cycle, *interoceptive exposure* (deliberately inducing the feared bodily sensations, for example by hyperventilating or spinning, to disconfirm the catastrophic prediction), in-vivo exposure for any agoraphobic avoidance, and cognitive restructuring of the **catastrophic misinterpretation** at the heart of Clark's model (NICE 2011). Because it directly dismantles the misinterpretation and the avoidance, CBT tends to protect against relapse better than medication stopped early.

**Where it sits against the drug.** Drug and CBT have broadly similar acute efficacy; the fullest answer offers either as first-line and combines them for the partial responder (BAP 2014). The general CBT model and the wider exposure principle are developed in the Psychotherapies, clinical assessment, MSE and nosology notes.

**GOOD TO REMEMBER**

- **CBT for panic (the psychotherapy):** first-line, 7 to 14 hours over about four months; its defining active ingredient is *interoceptive exposure*, deliberately provoking the feared bodily sensations to disconfirm the catastrophic misinterpretation of Clark's model, plus cognitive restructuring and in-vivo exposure for agoraphobic avoidance; it has similar acute efficacy to an SSRI and better relapse protection, and combining CBT with an antidepressant is the recommended step for a partial responder.

### 2.10 The benzodiazepine: reserved, time-limited, never the first line

**The reserved role.** Every guideline pushes the benzodiazepine out of the first line. NICE says outright *do not* prescribe benzodiazepines for panic disorder, because they are associated with a worse long-term outcome (NICE 2011). CANMAT places them second-line, WFSBP reserves them for treatment-resistant cases with no substance-use history, and WHO permits them only for severe acute symptoms and only very short-term, 3 to 7 days (CANMAT 2014; WFSBP 2008; WHO mhGAP 2023). The Indian Psychiatric Society allows a short-acting, orally-dissolving benzodiazepine with reassurance for the *acute panic attack itself*, and MOH Singapore permits adding one to an antidepressant short-term to cover the early activation window, tapering it off within four weeks (IPS 2017; MOH_SG 2015). The through-line: a benzodiazepine is a bridge for the acute attack or the activation window, never the maintenance treatment and never a monotherapy.

**Why the caution is genuine.** The risks are dependence, tolerance, cognitive dulling, rebound anxiety and inter-dose panic, and a worse long-term course, which is why the drug is time-limited and tapered. The wider anxiolytic pharmacology, benzodiazepines, buspirone, pregabalin, the z-drugs, dependence and withdrawal, is developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes.

**INDIA**

Panic disorder sits inside a large, mostly untreated Indian anxiety burden, most sufferers never reach formal care, and it collides with the country's benzodiazepine over-prescription problem: alprazolam and clonazepam are dispensed freely, often long-term, as a first move for "ghabrahat" and palpitations, which is exactly what every guideline warns against. The Indian Psychiatric Society CPG is unambiguous, an SSRI or SNRI is first-line for long-term management and the benzodiazepine is only a short-acting, orally-dissolving bridge for the acute attack. Indian brands are cheap and available, sertraline (Serta, Zosert, Daxid), escitalopram (Nexito, Cipralex), paroxetine (Paxidep) and fluoxetine (Fludac); the correct Indian answer is a low-started, well-explained SSRI plus CBT, not a chronic benzodiazepine. A neat exam pearl: the lactate provocation (sodium lactate infusion) test can precipitate an attack in about 70% of panic-disorder patients, a classic Indian-textbook association.

**TRAP** **The chronic benzodiazepine.** A benzodiazepine as ongoing monotherapy for panic disorder is the classic error: it worsens the long-term outcome, breeds dependence and inter-dose rebound panic. It is at most a short-acting bridge for the acute attack or the SSRI activation window, tapered within a few weeks, never the maintenance treatment.

| 4 of 13 | 1 month | 12 weeks | 6 months |
|---|---|---|---|
| SYMPTOMS NEEDED FOR A FULL PANIC ATTACK (FEWER IS A LIMITED-SYMPTOM ATTACK) | OF ANTICIPATORY WORRY OR BEHAVIOUR CHANGE REQUIRED (CRITERION B) | SSRI COURSE BEFORE JUDGING NON-RESPONSE AND CONSIDERING A TCA | MINIMUM CONTINUATION AFTER RESPONSE; TAPER OVER AT LEAST 3 MONTHS |

**HOLD THIS**

Panic disorder is recurrent *unexpected* panic attacks (an abrupt surge peaking within minutes, four or more of thirteen symptoms) plus at least a month of anticipatory worry, driven by catastrophic misinterpretation of body sensations, and treated first-line with an SSRI started at half the usual dose with an explicit warning about early activation plus CBT with interoceptive exposure; the benzodiazepine is reserved, time-limited and never a monotherapy.

## 3 · Clark cognitive model of panic

David M. **Clark**'s **cognitive model of panic** (1986) is the single most examined psychological model in the anxiety chapter, because it does two things at once: it explains why a panic attack feels like a heart attack, and it hands the clinician the exact lever that **cognitive behaviour therapy** pulls to abolish it. The claim is deceptively simple. Panic disorder arises not from the bodily sensations of arousal themselves, which everyone gets, but from the **catastrophic misinterpretation** of those benign sensations as an imminent physical or mental disaster. A racing heart is read as *"I am having a heart attack"*, breathlessness as *"I am suffocating"*, dizziness as

*"I am about to collapse or go mad"*. That misreading feeds a self-amplifying loop that Clark drew as a **vicious cycle** (shown in the accompanying figure), and it is the loop, not any one symptom, that the model asks you to memorise.

**Why it matters.** The model is the reason panic disorder is one of the most treatable conditions in psychiatry: break the misinterpretation and the cycle collapses. It supplies the discriminator examiners want (panic disorder is a disorder of *interpretation*, not of the autonomic nervous system) and it maps one-to-one onto the CBT protocol that BAP, NICE and CANMAT all place first-line. The wider CBT technique, the general cognitive model and the exposure principle belong to the Psychotherapies, clinical assessment, MSE and nosology notes; this topic owns Clark's disorder-specific loop, the safety behaviours that keep it turning, and how CBT dismantles each link.

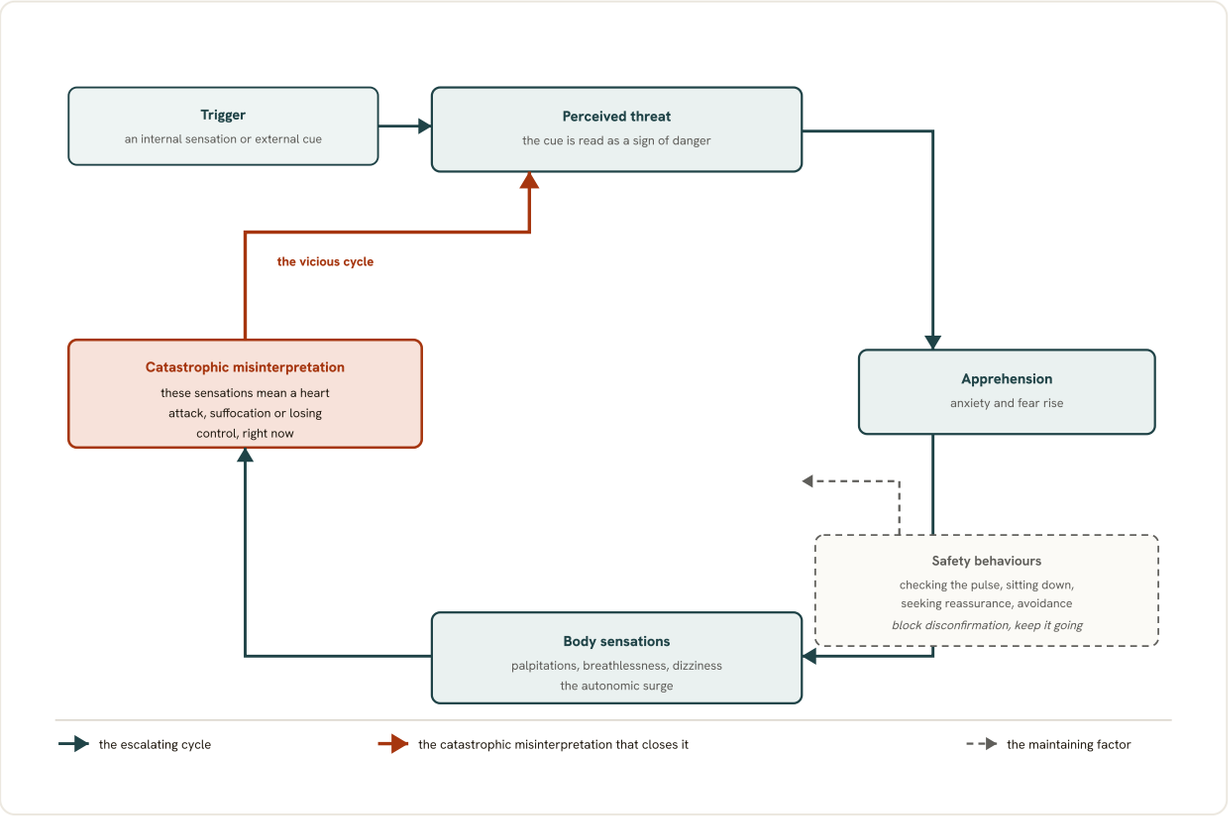



**Fig 14.3** Clark's cognitive model of panic. A trigger, most often an ordinary bodily sensation, is read as a sign of threat; the apprehension that follows amplifies the autonomic body sensations, which are then catastrophically misinterpreted as an imminent heart attack, suffocation or loss of control, and that misinterpretation feeds straight back into the perceived threat, closing a vicious cycle that escalates to a full panic attack in minutes. Safety behaviours, checking the pulse, sitting down, seeking reassurance and avoidance, feel protective but block the disconfirmation that would break the cycle, so they maintain the disorder. Cognitive behaviour therapy works by targeting the misinterpretation and dropping the safety behaviours, and the wider technique is taught in the Psychotherapies, clinical assessment, MSE and nosology notes.

### 3.1 The core claim: catastrophic misinterpretation of benign bodily sensations

**The single idea.** According to Clark, a panic attack occurs when an individual interprets normal or benign **bodily sensations** as evidence of an immediate catastrophe (Clark 1986, 1988, 1996; Tasman Psychiatry). The sensations are ordinary products of arousal, exercise, caffeine, a missed meal, or a fleeting emotion; what is pathological is the meaning imposed on them. **The evidence that seals it.** When panic is provoked experimentally (for example by lactate infusion or carbon-

dioxide inhalation), panic-disorder patients and healthy controls report the *same* physical sensations, yet only the patients who make a catastrophic interpretation go on to a full attack (Sanderson and Rego; Tasman Psychiatry). The bodily signal is shared; the disorder lives in the appraisal. **Why "catastrophic".** The misinterpretation is not merely negative but specifically catastrophic and *imminent*, forecasting death, collapse, loss of control or insanity within seconds, which is what drives the terror out of proportion to the trigger.

RULE **The interpretation, not the sensation, is the disorder.** Everyone gets palpitations; only the person who reads them as "heart attack" panics. Treatment targets the appraisal.

### 3.2 The vicious cycle: the loop you must be able to draw

**The five links.** Clark's loop runs: a *trigger* (internal, such as a bodily sensation or intrusive thought, or external, such as a crowded shop) leads to the *perceived threat*, which produces *apprehension or fear*, which drives further *bodily sensations* of autonomic arousal, which are then subjected to *catastrophic misinterpretation*, which feeds straight back into perceived threat, tightening the spiral until a full attack crests (the accompanying figure lays out the loop). **Why it is self-amplifying.** Each turn recruits more arousal: the thought "I am dying" is itself a fresh threat, so the sympathetic surge grows, the sensations intensify, and the interpretation looks ever more confirmed. This positive-feedback quality is the reason a panic attack escalates from first flutter to peak terror within about **10 minutes** and then, once the loop can no longer amplify, spontaneously subsides. **The internal trigger point.** Crucially the trigger is often internal and invisible to bystanders, which is why panic attacks appear to come "out of the blue"; the patient has misread a benign internal cue, not responded to an external danger.

TRAP **Do not confuse the trigger with the cause.** The out-of-the-blue attack is not causeless; it is an internal bodily sensation, misread, that the patient did not consciously register as the trigger.

### 3.3 Safety behaviours: why the belief never disconfirms itself

**What they are.** Safety behaviours are actions the patient performs to avert the feared catastrophe: gripping the trolley or a companion's arm to avoid collapsing, sitting down and breathing deeply to avoid a "heart attack", carrying water or medication, scanning for exits, or avoiding exercise and caffeine (Anxiety and Panic Therapist Guide, Treatments that Work). **Why they are pathogenic.** They feel protective but are the engine of chronicity, because they prevent *disconfirmation*: the patient survives the attack, attributes the survival to the safety behaviour rather than to the harmlessness of the sensations, and so the catastrophic belief is never tested and never falsified ("I only survived because I sat down"). **The link to avoidance and agoraphobia.** The same logic scales up: avoiding the supermarket, the bus or being alone are macro-level safety behaviours that entrench agoraphobic avoidance, so that the belief "these sensations are dangerous" is protected across a widening range of situations (the phobic-avoidance detail sits with the panic disorder and agoraphobia topics).

**PEARL**

Safety behaviours are the reason reassurance and self-monitoring apps often make panic worse, not better. A pulse oximeter, a "check my heart" habit, or a benzodiazepine kept in the pocket "just in case" all become safety behaviours: they hand the patient a fresh reason the catastrophe was averted, so the belief is preserved rather than disproved. The therapeutic move is to *drop* the safety behaviour and let the sensation prove itself harmless.

### 3.4 How CBT breaks the cycle

**The three levers.** Cognitive behaviour therapy for panic maps exactly onto the loop and attacks it at three points (Clark; BAP, Baldwin et al.; NICE). *Cognitive restructuring* identifies and challenges the catastrophic misinterpretation, replacing "palpitations mean a heart attack" with an accurate, non-catastrophic account of arousal. *Interoceptive exposure* deliberately induces the feared sensations (hyperventilating to voluntary breathlessness and dizziness, spinning to induce vertigo, running on the spot to raise the heart rate) so the patient experiences the sensation, makes no misinterpretation, and learns experientially that it is harmless. *Dropping safety behaviours and situational (in-vivo) exposure* remove the props so the belief is finally tested and disconfirmed. **The behavioural experiment.** The pivotal technique is the behavioural experiment: the patient predicts the catastrophe ("if I don't sit down I will faint"), then hyperventilates without the safety behaviour, and observes that the catastrophe does not occur, which corrects the belief far more powerfully than argument. **Dose and place.** CBT for panic is first-line, delivered in the optimal range of **7 to 14 hours** total, typically weekly over about **4 months** (NICE; BAP). The general CBT and exposure technique is developed in the Psychotherapies, clinical assessment, MSE and nosology notes.

**WORKED EXAMPLE**

A 28-year-old woman has recurrent attacks that begin with a pounding heart she reads as "I'm having a heart attack." In the session the therapist has her hyperventilate for two minutes; her heart races and she feels dizzy, exactly her attack, but with the prediction "I will collapse" written down first. She does not collapse. Repeated across sessions, with her hand off the wall (a dropped safety behaviour), the sensation stops meaning catastrophe. The catastrophic misinterpretation is disconfirmed at its source, and the vicious cycle has nowhere to turn.

### 3.5 Where the model sits in management, and the benzodiazepine trap

**First-line, and why.** Because the model is so directly testable, CBT built on Clark's framework is a first-line treatment for panic disorder with or without agoraphobia, on a par with an SSRI (BAP; NICE; CANMAT places CBT first-line psychologically). Where medication is used, an SSRI licensed for panic (escitalopram, sertraline, paroxetine or citalopram) or venlafaxine is first-line; a patient starting an SSRI must be warned about early activation and a transient worsening of anxiety, because in Clark's terms the initial jitteriness supplies fresh bodily sensations to misinterpret. **The benzodiazepine trap.** NICE explicitly says *do not prescribe benzodiazepines* for panic disorder: they are associated with a worse long-term outcome, and, read through Clark's

model, they act as a chemical safety behaviour that blocks the disconfirming learning CBT depends on. This matters acutely in the Indian setting.

**INDIA**

Panic disorder in India is routinely met with a benzodiazepine, often alprazolam, prescribed long-term in primary care and by non-specialists, against every guideline. Clark's model gives the sharpest argument against the habit: the tablet-in-the-pocket becomes a safety behaviour that preserves the very belief driving the disorder, so symptomatic relief today buys chronicity tomorrow, on top of the well-documented dependence problem. The Indian Psychiatric Society clinical practice guidelines for anxiety disorders reserve benzodiazepines for short-term use and place SSRIs and CBT first; against the wide anxiety treatment gap, teaching the panic cycle to the patient is itself a low-cost, transferable intervention that does not require a psychologist on site.

**TRAP** **The prescribed benzodiazepine is a safety behaviour.** Giving alprazolam "to carry just in case" relieves the attack but preserves the catastrophic belief, blocking the disconfirmation CBT needs and inviting dependence.

| 7 to 14 | 4 | 1986 |
|---|---|---|
| HOURS OF CBT FOR PANIC, THE FIRST-LINE DOSE (NICE, BAP) | MONTHS, THE TYPICAL CBT DELIVERY WINDOW | CLARK'S FOUNDATIONAL COGNITIVE-MODEL PAPER |

**GOOD TO REMEMBER**

- **Clark cognitive model of panic:** panic disorder is the *catastrophic misinterpretation of benign bodily sensations* as imminent physical or mental catastrophe; the loop is trigger, perceived threat, apprehension, bodily sensations, catastrophic misinterpretation, back to perceived threat.
- **The maintaining mechanism:** safety behaviours (and avoidance) prevent disconfirmation, so the catastrophic belief is never tested and the disorder becomes chronic.
- **The discriminator:** the disorder is one of *interpretation*, not of the autonomic nervous system; provoked sensations are identical in patients and controls, only the misinterpretation predicts an attack.

**GOOD TO REMEMBER**

- **CBT for panic (Clark protocol):** first-line, 7 to 14 hours over about 4 months; three levers are cognitive restructuring of the misinterpretation, interoceptive exposure to the feared sensations, and dropping safety behaviours with in-vivo exposure, driven by behavioural experiments.
- **Signature caution:** a benzodiazepine (and any "just in case" prop) acts as a safety behaviour that blocks the disconfirming learning, so it is not first-line and NICE advises against it in panic disorder.

**HOLD THIS**

Panic disorder is the catastrophic misinterpretation of benign bodily sensations, sustained by safety behaviours that stop the belief being disconfirmed; CBT (7 to 14 hours) breaks the vicious cycle by restructuring the misinterpretation, exposing the patient to the sensations, and removing the safety behaviours so the catastrophe is finally proven not to happen.

## 4 · Agoraphobia

**Agoraphobia** is not, despite its name, a simple **fear of public places**. It is a fear of *being trapped*: of finding oneself in a situation from which escape would be difficult or embarrassing, or in which help would not be available, should a panic attack or some other incapacitating or humiliating bodily event strike. The Greek *agora* was the marketplace, and the marketplace still captures the clinical picture, the crowd, the queue, the bus, the open square, but the driving cognition is about escape and rescue, not about the place itself. The patient reorganises life around this fear until, at its most severe, they are completely homebound. Examiners test **agoraphobia** on a tight set of exact points: the two-or-more-of-five situations of Criterion A, the *escape/help-unavailable* cognition of Criterion B that separates it from a specific phobia, its modern status as a diagnosis in its own right independent of panic disorder, and the treatment, where the single load-bearing fact is that *graded in-vivo exposure* is the core of care.

**Why it matters clinically.** This is a hybrid topic, so the marks sit both in the criterion-anchored discrimination and in the exposure-based management. Two facts anchor every answer. First, the diagnostic core is the cluster of at least two of five feared situations plus the specific cognition that escape or help would be impossible, which is exactly what separates agoraphobia from the single-situation specific phobia and from the socially-evaluative fear of social anxiety disorder. Second, both ICD-11 and DSM-5-TR now diagnose **agoraphobia** as a stand-alone disorder, *irrespective of the presence of panic disorder*, a deliberate break from the older nesting of "panic disorder with agoraphobia". The fear-circuit anatomy on which the alarm is built is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes, and the general CBT model and the wider exposure principle in the Psychotherapies, clinical assessment, MSE and nosology notes; they are not re-derived here.

### 4.1 What agoraphobia actually is: fear of being trapped, not fear of the place

**The core fear.** The essential feature is marked fear or anxiety triggered by the real or anticipated exposure to a range of situations, feared *because the person believes that escape might be difficult or that help might be unavailable* if panic-like or other incapacitating or embarrassing symptoms were to occur (DSM-5-TR 2022; ICD-11 CDDR). The place is incidental; the dread is of being stuck in it. "Panic-like symptoms" means any of the thirteen panic-attack symptoms (dizziness, faintness, fear of dying and the rest), and "other incapacitating or embarrassing symptoms" covers vomiting, incontinence, and, in older adults, a fear of falling.

**What the avoidance looks like.** The behaviour that follows is avoidance: the person actively rearranges their life to prevent contact with the feared situations, takes a job near home to avoid the bus, has food delivered to avoid the shop, insists on a companion, or sits near the exit at the cinema as a *safety behaviour*. Avoidance can be behavioural or cognitive (using distraction to endure), and at its most severe the person becomes entirely housebound, dependent on others for basic needs (DSM-5-TR 2022).

**WORKED EXAMPLE**

A 34-year-old woman has, over eight months, stopped taking the bus, stopped going to the crowded weekly market, and will now enter a shop only if her sister comes with her; she has not left the house alone in weeks. She describes no fear of the bus or the market as such, but a dread that she will "feel faint and trapped with no way out and no one to help". There is no current panic disorder, though she had a handful of panic attacks a year ago. Two or more of the five agoraphobic situations are feared (public transport, crowds, being outside the home alone), the cognition is the escape/help-unavailable one, the avoidance is marked and companion-dependent, and it has lasted well beyond six months. This is agoraphobia, diagnosable in its own right, panic disorder or not.

### 4.2 Criterion A: two or more of the five situations

**The five situations.** The diagnosis requires marked fear or anxiety about *two or more* of five situation clusters (DSM-5-TR 2022): (1) using **public transportation** (buses, trains, ships, planes); (2) being in **open spaces** (car parks, marketplaces, bridges); (3) being in **enclosed places** (shops, theatres, cinemas); (4) **standing in line or being in a crowd**; and (5) **being outside of the home alone**. The examples are not exhaustive, and the "two or more" threshold is the number examiners protect, because it is precisely what lifts the picture out of specific phobia.

**Consistency and proportion.** The fear must be evoked *nearly every time* the person meets the situation (Criterion C), must be actively avoided or endured with intense distress or need a companion (Criterion D), and must be out of proportion to the actual danger and to the sociocultural context (Criterion E) (DSM-5-TR 2022). That last clause matters cross-culturally: where it is socioculturally expected that a woman does not leave the house alone, that avoidance is not, by itself, agoraphobia.

| CRITERION | REQUIREMENT |
|---|---|
| **A** | Marked fear/anxiety about two or more of five situations (public transport, open spaces, enclosed places, crowds/queues, outside home alone) |
| **B** | **Feared because escape may be difficult or help unavailable if panic-like or incapacitating/embarrassing symptoms occur** |
| **C** | The situations almost always provoke fear or anxiety |
| **D** | Actively avoided, need a companion, or endured with intense fear |
| **E** | Out of proportion to actual danger and sociocultural context |
| **F** | **Persistent, typically six months or more** |
| **G** | Clinically significant distress or impairment |

The DSM-5-TR criteria for agoraphobia; the two-of-five rule (A) and the escape/help-unavailable cognition (B) are the load-bearing discriminators (DSM-5-TR 2022).

### 4.3 Criterion B: the escape-or-help cognition that defines it

**The discriminating thought.** Criterion B is what makes agoraphobia agoraphobia: the situations are feared *because of thoughts that escape might be difficult or help might not be available* in the event of developing panic-like symptoms or other incapacitating or embarrassing symptoms (DSM-5-TR 2022; ICD-11 CDDR). This single cognition is the examiner's favourite discriminator. If the situation is feared for another reason, fear the plane will crash, fear the dog will bite, then it is a *specific phobia*, not agoraphobia. If it is feared because of negative social evaluation, it is *social anxiety disorder*. The **exposure agoraphobia** treatment that follows is built directly on disconfirming this escape-or-help prediction.

**Where the fear centres.** ICD-11 puts it crisply: fear or anxiety in agoraphobia centres on *imminent perceived dangerous outcomes*, panic attacks, symptoms of panic, incapacitation, or embarrassing physical symptoms, that are anticipated to occur in multiple situations where obtaining help or escaping might be difficult (ICD-11 CDDR). It is worth establishing the specific feared outcome (a panic attack versus incontinence versus falling), because that shapes the exposure plan.

RULE **Two of five, feared for escape not for the object.** Agoraphobia = fear/avoidance of two or more of the five situations, feared *because escape or rescue would be impossible* if something dreadful happened in the body; that Criterion-B cognition, not the place, is the diagnosis.

### 4.4 The nosological shift: agoraphobia as a stand-alone diagnosis

**Independent of panic.** The examinable modern point is that both classifications now diagnose agoraphobia *independent of panic disorder*. DSM-5-TR states it outright: agoraphobia is diagnosed irrespective of the presence of panic disorder, and if a presentation meets criteria for both, *both* diagnoses are assigned (DSM-5-TR 2022). ICD-11 (6B02) similarly lists agoraphobia as its own category within the anxiety and fear-related disorders, with a "with panic attacks" specifier rather than a subordination to panic disorder (ICD-11 CDDR). This retired the old DSM-IV construct of "panic disorder with agoraphobia" and the notion that agoraphobia could not exist without a panic history.

**The panic relationship in numbers.** The link is real but not obligatory: the proportion of agoraphobia cases with panic attacks or panic disorder preceding onset ranges from about 30% in community samples to more than 50% in clinical samples, and agoraphobia without any preceding panic runs a mean age at onset of 25 to 29 years (DSM-5-TR 2022). Many patients avoid so completely that they no longer have attacks at all, which is exactly why tying the diagnosis to current panic was untenable.

PEARL

The single most exam-worthy fact about agoraphobia in ICD-11 and DSM-5-TR is that it is now a *free-standing* diagnosis, no longer a mere appendage of panic disorder. Where a patient meets both sets of criteria, code *both* (panic disorder *and* agoraphobia), not the old composite "panic disorder with agoraphobia". Heritability is high (about 61%), the highest and most phobia-specific of the phobias, another favourite one-liner.

### 4.5 Differential diagnosis: the boundaries an answer must draw

**Against specific phobia.** If only *one* agoraphobic situation is feared, diagnose specific phobia, situational type; two or more warrant agoraphobia (DSM-5-TR 2022). And if the situation is feared because of direct harm from the object (the plane crashing) rather than the escape/help cognition, it is again specific phobia.

**Against the others.** In *social anxiety disorder* the fear is of negative evaluation, so a fear confined to social scrutiny is social anxiety, not agoraphobia. In *PTSD* the person deliberately avoids trauma reminders, whereas in agoraphobia situations are avoided for fear of imminent bodily catastrophe with no escape (ICD-11 CDDR). In *separation anxiety disorder* the fear is of being apart from an attachment figure. And the organic and depressive mimics that colour every anxiety presentation, thyroid disease, arrhythmia, and secondary depression, are excluded as for panic disorder.

**GOOD TO REMEMBER**

- **Agoraphobia (the syndrome):** the defining criterion is marked fear/avoidance of *two or more of five* situations (public transport, open spaces, enclosed places, crowds or queues, being outside the home alone), feared specifically *because escape would be difficult or help unavailable* should panic-like or incapacitating/embarrassing symptoms strike (Criterion B); it must be persistent (six months or more) and out of proportion to sociocultural context; the discriminators are one-situation-only or object-directed fear (specific phobia), evaluative fear (social anxiety), trauma-reminder avoidance (PTSD); and, in the exam-critical shift, it is now diagnosed *independent of panic disorder*, with both coded if both are present.

### 4.6 Management overview: exposure-based CBT is the core

**The treatment principle.** The management of agoraphobia is, first and above all, *psychological and exposure-based*. CANMAT recommends cognitive behavioural therapy that uses *combined strategies including situational exposure* as first-line, noting that combined techniques outperform single techniques on agoraphobia measures (CANMAT 2014). NICE frames agoraphobia within panic-focused CBT: offer CBT for panic disorder *with or without agoraphobia* as first-line, delivered over about 7 to 14 hours across four months, with *in-vivo exposure for agoraphobic avoidance* as an explicit ingredient (NICE 2011). Where formal face-to-face therapy is impractical, minimal-intervention formats (self-help books, telephone or video delivery, internet-based CBT) are effective and cost-effective alternatives (CANMAT 2014).

**Where drugs sit.** Because agoraphobia so often travels with panic, the pharmacological backbone is the panic backbone: an SSRI is the first-line agent for the anxiety/panic component (the antidepressant class shared across the anxiety disorders, fluoxetine, sertraline, fluvoxamine, paroxetine, escitalopram), started low and titrated, with the tricyclics clomipramine (Anafranil) or imipramine as second-line agents that have meta-analytic efficacy for both panic and agoraphobia (CANMAT 2014). Medication is combined with exposure where agoraphobia is severe or rapidly worsening, or where CBT alone is likely to be insufficient (comorbid moderate-to-severe depression, frequent severe panic, or suicidal ideation) (CANMAT 2014).

| LINE | TREATMENT | SOURCE |
|---|---|---|
| **First-line** | CBT with combined situational (in-vivo) exposure and cognitive restructuring | CANMAT 2014; NICE 2011 |
| **First-line (drug)** | SSRI for the panic/anxiety component (start low, titrate, warn about early activation) | NICE 2011; BAP 2014 |
| **Second-line (drug)** | **Clomipramine or imipramine (efficacy for both panic and agoraphobia)** | CANMAT 2014 |
| **Access alternative** | Low-intensity or internet-based CBT, guided self-help | CANMAT 2014 |
| **Severe/rapid** | Combined pharmacotherapy plus psychotherapy | CANMAT 2014 |

Guideline-level management of agoraphobia; exposure-based CBT is first-line, the SSRI carries the panic component, and the tricyclics clomipramine and imipramine are second-line (CANMAT 2014; NICE 2011).

### 4.7 Graded in-vivo exposure: how the core treatment actually works

**What exposure agoraphobia treatment does.** Graded in-vivo exposure is the active ingredient. The patient and therapist build a hierarchy of feared situations from least to most distressing (standing at the front door, walking to the gate, a short bus ride, the crowded market) and the patient enters them *repeatedly and in a graded way*, remaining until the anxiety falls, so that the escape-or-help catastrophe (Criterion B) fails to materialise and is disconfirmed (CANMAT 2014; NICE 2011). The exposures must be *real and in-vivo*, prolonged, and repeated across settings; the safety behaviours (the companion, the exit seat, the water bottle) are progressively dropped, because it is only exposure *without* the safety crutch that fully extinguishes the fear.

**For those who decline formal therapy.** For patients with persistent agoraphobic distress or avoidance who fear or decline formal CBT, the guideline is still exposure: provide instruction and support to engage in *graded exposure exercises* on their own or with minimal facilitation (CANMAT 2014). Where in-vivo entry is genuinely impractical, virtual-reality or interoceptive exposure can substitute or supplement, and where panic drives the avoidance, interoceptive exposure (deliberately inducing the feared body sensations) is added, as in panic-focused CBT.

TRAP **Exposure with the safety behaviours left in.** Sending a patient into the feared situation while they keep the companion, the exit seat and the escape route intact teaches nothing, the catastrophe is never disconfirmed. Grade the hierarchy, keep exposures prolonged and repeated, and *systematically drop the safety behaviours*; and never let avoidance itself go unaddressed while only prescribing a drug.

### 4.8 The Indian frame: the treatment gap, the benzodiazepine problem, and the IPS line

**Genuine Indian context.** Agoraphobia sits inside a large, mostly untreated anxiety burden in India: the anxiety, OCD and PTSD treatment gap means most sufferers never reach formal psychological care, and structured exposure-based CBT, the very treatment that is first-line, is scarce outside a handful of centres. Into that gap falls India's **benzodiazepine over-prescription**

problem: alprazolam and clonazepam are dispensed freely and often long-term for "ghabrahat", panic and phobic avoidance, which is precisely the wrong move, a benzodiazepine suppresses the very anxiety the patient needs to feel and habituate to during exposure, and can actively blunt the therapeutic effect of exposure work. The Indian Psychiatric Society CPGs keep the line clean: an SSRI or SNRI for the panic/anxiety component and CBT with systematic exposure as the treatment, not a chronic anxiolytic (IPS 2017). Indian brands are cheap and available: sertraline (Serta, Zosert, Daxid), escitalopram (Nexito, Cipralex), paroxetine (Paxidep), fluoxetine (Fludac), and clomipramine (Anafranil) for the second line. The correct Indian answer is graded in-vivo exposure plus a low-started, well-explained SSRI, never a standing benzodiazepine.

**GOOD TO REMEMBER**

- **Graded in-vivo exposure (the psychotherapy):** the first-line, core treatment of agoraphobia; its mechanism is repeated, graded, prolonged entry into the feared situations so the escape-or-help catastrophe of Criterion B is disconfirmed and the fear extinguishes; the signature caution is that *safety behaviours must be dropped* and a concurrent benzodiazepine avoided (both prevent habituation); combined CBT (exposure plus cognitive restructuring) outperforms single techniques, with the SSRI carrying the panic component and clomipramine or imipramine as the second-line drug.

| | | | |
|---|---|---|---|
| **2 of 5** | **6 months** | **7 to 14 h** | **61%** |
| SITUATIONS FEARED, REQUIRED FOR CRITERION A | MINIMUM DURATION FOR THE DIAGNOSIS (CRITERION F) | CBT WITH IN-VIVO EXPOSURE OVER ABOUT 4 MONTHS (NICE) | HERITABILITY, THE MOST PHOBIA-SPECIFIC OF THE PHOBIAS |

**HOLD THIS**

Agoraphobia is fear and avoidance of two or more of five situations (public transport, open spaces, enclosed places, crowds or queues, being outside the home alone), feared *because escape would be difficult or help unavailable* if a panic-like or incapacitating symptom struck; it is now diagnosed independent of panic disorder (code both if both present), and its first-line treatment is graded in-vivo exposure within CBT, with an SSRI for the panic component and the benzodiazepine kept firmly out.

## 5 · Social anxiety disorder

**Social anxiety disorder** (still widely called **social phobia**) is the disorder of a marked, persistent **fear of scrutiny** and of negative evaluation in social situations. Where generalised anxiety disorder is free-floating and panic is episodic and cued-by-nothing, this fear is *bound to the social gaze*: the patient dreads acting in a way, or showing anxiety symptoms, that others will judge as humiliating, embarrassing or offensive, and so avoids or endures with dread the very situations, conversations, meetings, eating in front of others, public speaking, in which that scrutiny might fall on them. Examiners like it because it carries a clean, high-yield distinction, the **generalised** subtype versus the **performance-only** specifier, and because its management is the anxiety-block template with one signature twist: the beta-blocker for discrete **performance anxiety**.

**Why it matters, and how this note is framed.** This is a hybrid topic: it teaches the diagnosis and its discrimination, then the guideline-anchored drug and psychotherapy plan. The core

discriminator to lead with is the *object* of the fear, negative evaluation by others, which is what separates social anxiety disorder from specific phobia (feared object), agoraphobia (fear that escape is impossible or help unavailable) and separation anxiety (fear of separation from an attachment figure). The amygdala and fear-circuit substrate is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; the deep SSRI pharmacology in the Mood disorders: pharmacotherapy notes; the general CBT and exposure model in the Psychotherapies, clinical assessment, MSE and nosology notes; and the benzodiazepine, pregabalin and buspirone story in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. What this note owns is the phenomenology, the subtype split, the criteria, the LSAS, and the management.

### 5.1 The core concept: fear of scrutiny and of negative evaluation

**The essential feature.** The heart of the disorder is a marked, intense fear or anxiety about one or more social situations in which the individual is exposed to *possible scrutiny by others* (DSM-5-TR 2022). The dreaded outcome is not the situation itself but the judgement it invites: the person is concerned they will act in a way, or show visible anxiety, blushing, trembling, sweating, stumbling over words, that will be negatively evaluated as humiliating, embarrassing, or leading to rejection or offence. This fear of negative evaluation is the cognitive engine, and naming it is what earns the marks.

**The three feared arenas.** The scrutiny is dreaded across three kinds of situation: social *interactions* (having a conversation, meeting unfamiliar people), being *observed* (eating or drinking in front of others), and *performing* before others (giving a speech). Blushing is described as the hallmark physical response of the disorder (Kaplan and Sadock 2024). A useful clinical texture: some patients avoid public toilets when others are present (paruresis, "shy bladder"), and self-medication with alcohol before a social event is common.

**GOOD TO REMEMBER**

- **Social anxiety disorder (social phobia):** a marked, persistent fear of one or more social situations involving possible scrutiny, driven by the fear of negative evaluation (that one will be judged humiliating, embarrassing, weak or offensive); the defining discriminator from the other anxiety disorders is that the feared object is *the judgement of others*; blushing is the hallmark physical sign, and the first diagnostic step is to establish that the fear is of negative evaluation and out of proportion to the actual social threat.

### 5.2 Generalised versus performance-only: the subtype split to know cold

**The one specifier DSM-5-TR keeps.** DSM-5-TR replaced the old "generalised" subtype with a single specifier, *performance only*: the fear is **restricted to speaking or performing in public** (DSM-5-TR 2022). By default, without the specifier, the fear spans a broad range of social and interactional situations, the **generalised** picture, which is more chronic, more impairing and more comorbid. The **performance-only** group, by contrast, do not fear or avoid non-performance social situations; their impairment falls in professional lives that demand public performance, musicians, dancers, athletes, and in roles requiring regular public speaking.

**Why the split drives treatment.** The distinction is not cosmetic: it steers the prescription. Generalised social anxiety needs a serotonergic antidepressant and CBT. Discrete **performance anxiety** can be covered situationally, as needed, with a beta-blocker taken before the feared performance, an option that has no place in the generalised form. This is exactly why the subtype question is examinable.

| FEATURE | GENERALISED (DEFAULT) | PERFORMANCE-ONLY SPECIFIER |
|---|---|---|
| **Situations feared** | **Broad: interactions, observation and performance** | Restricted to speaking/performing in public |
| **Non-performance social situations** | Feared and avoided | **Not feared or avoided** |
| **Typical impairment** | Pervasive; more chronic and comorbid | Focused on public-performance roles |
| **First-line treatment** | SSRI (or SNRI) plus CBT | **Situational beta-blocker for the discrete event** |

The generalised picture versus the DSM-5-TR performance-only specifier; the split is high-yield because it changes the drug (serotonergic antidepressant versus as-needed beta-blocker).

RULE **Subtype sets the drug.** Generalised social anxiety disorder is treated with an SSRI (or SNRI) plus CBT; the discrete performance-only picture can be covered situationally with a beta-blocker before the event, which has no role in the generalised form.

### 5.3 DSM-5-TR criteria: marked fear, negative evaluation, six months

**The criteria to state cleanly.** DSM-5-TR requires (A) marked fear or anxiety about one or more social situations involving possible scrutiny; (B) the individual fears they will act, or show anxiety symptoms, that will be negatively evaluated; (C) the situations almost always provoke fear or anxiety; (D) they are avoided or endured with intense fear; (E) the fear is out of proportion to the actual threat and to the sociocultural context; (F) it is persistent, **typically lasting 6 months or more**; (G) it causes clinically significant distress or impairment; (H) it is not attributable to a substance or another medical condition; and (I) it is not better explained by another mental disorder (DSM-5-TR 2022). The specifier is *performance only*.

**The paediatric qualifier.** In children the anxiety must occur in *peer settings* and not only during interactions with adults, and the fear may be expressed by crying, tantrums, freezing, clinging or failing to speak. That "peer settings" caveat is a favourite discriminator.

| CRITERION | DSM-5-TR REQUIREMENT |
|---|---|
| **A. Fear** | **Marked fear/anxiety about social situation(s) with possible scrutiny (interaction, observation, performance)** |
| **B. Feared outcome** | **Fear of acting or showing anxiety that will be negatively evaluated (humiliating, embarrassing, rejecting, offensive)** |

| CRITERION | DSM-5-TR REQUIREMENT |
|---|---|
| **C. Consistency** | The situations almost always provoke fear or anxiety (in children, may show as crying/tantrums/freezing) |
| **D. Avoidance** | Situations avoided or endured with intense fear or anxiety |
| **E. Proportion** | Out of proportion to the actual threat and to the sociocultural context |
| **F. Duration** | **Persistent, typically 6 months or more** |
| **G. Impairment** | Clinically significant distress or social/occupational impairment |
| **H. Not organic** | Not due to a substance or another medical condition |
| **I. Not better explained** | Not better explained by panic disorder, body dysmorphic disorder, autism spectrum disorder and others |
| **Specifier** | **Performance only: fear restricted to speaking or performing in public** |

DSM-5-TR criteria for social anxiety disorder; the numbers to hold are the 6-month typical duration and the single performance-only specifier (DSM-5-TR 2022).

### 5.4 ICD-11 CDDR: marked and excessive fear, "at least several months"

**Where ICD-11 frames it.** ICD-11 (6B04) requires marked and excessive fear or anxiety that occurs *consistently* in one or more social situations, social interaction, being observed, or performing, with the concern that one will act, or show anxiety symptoms, that will be negatively evaluated (humiliating, embarrassing, leading to rejection or offence); the situations are consistently avoided or endured with intense fear; the symptoms are not transient and persist for **at least several months**; and they cause significant distress or impairment (ICD-11 CDDR). As across the anxiety block, ICD-11 does *not* impose the DSM's fixed 6-month clock, asking instead for "at least several months", the classic ICD-versus-DSM contrast to name.

**The ICD-11 specifier.** ICD-11 offers a "with panic attacks" qualifier rather than a performance-only one, so if you are asked about the performance-only distinction, that is a DSM-5-TR construct; ICD-11 codes the panic-attack overlay instead.

TRAP **Do not miss the medical and psychiatric mimics.** Blushing, tremor and palpitations before scrutiny can equally be hyperthyroidism, a phaeochromocytoma or excess caffeine; and fear of being looked at can be body dysmorphic disorder (fear centred on a perceived defect) or the social withdrawal of autism, not social anxiety, so exclude the organic mimic and check that the fear is genuinely of *negative evaluation*.

**GOOD TO REMEMBER**

- **ICD-11 social anxiety disorder (6B04):** marked, excessive, consistent fear of social situations with concern about negative evaluation, consistent avoidance, persisting at least several months, with distress or impairment; the defining contrast with DSM-5-TR is the absence of a fixed 6-month rule and a "with panic attacks" specifier in place of DSM's performance-only one.

### 5.5 The Liebowitz Social Anxiety Scale (LSAS): the severity instrument

**What it is.** The Liebowitz Social Anxiety Scale (**lsas**), developed by Michael Liebowitz in 1987, is the standard instrument for assessing social anxiety. It is a **24-item** scale that rates both *fear* and *avoidance* across 24 social and performance situations, each dimension scored **0 to 3**, splitting into a social-interaction subscale and a performance subscale (AIIMS Clinical Methods 2024; Kaplan and Sadock 2024). It exists in clinician-administered and self-rated forms; it is copyright-protected, so permission is needed to use it, and it is set out on the accompanying scale plate rather than reproduced here.

**How it is used.** The LSAS is the workhorse outcome measure in social anxiety trials, drug effects are reported as points reduced on the LSAS, and it is what tracks response over a treatment course. For quick case-finding, NICE instead recommends the 3-item Mini-Social Phobia Inventory (Mini-SPIN) or two screening questions, referring for full assessment if positive (NICE 2013). The through-line: Mini-SPIN to screen, LSAS to grade severity and follow response.

**GOOD TO REMEMBER**

- **LSAS (Liebowitz 1987):** a 24-item scale rating fear and avoidance across 24 situations, each 0 to 3, with social-interaction and performance subscales; it is the standard severity and outcome measure in social anxiety disorder (drug effects are reported as LSAS point reductions); contrast with the 3-item Mini-SPIN, which is the rapid screen.

### 5.6 Management: SSRI first-line and CBT, across the guidelines

**Lead with BAP.** The British Association for Psychopharmacology (BAP) makes an SSRI the first-line pharmacological treatment for social anxiety disorder, the efficacious agents named being escitalopram, fluoxetine, fluvoxamine, paroxetine and sertraline; it also offers cognitive behavioural therapy (cognitive therapy plus exposure) as an efficacious acute treatment of broadly similar efficacy to pharmacotherapy, and names the SNRI venlafaxine, the MAOI phenelzine and the RIMA moclobemide as alternative antidepressants (BAP 2014). This is the verified **SSRI first-line** anchor and must not be contradicted. BAP adds a pointed negative: *do not* prescribe atenolol or buspirone for generalised social anxiety disorder, as neither is efficacious (BAP 2014).

**Then the other bodies.** The convergence is striking. NICE puts individual CBT specifically developed for social anxiety disorder (the Clark and Wells or Heimberg model) as the *first-line* treatment for adults, and offers an SSRI (escitalopram or sertraline) if the adult declines psychological therapy and prefers medication, with an alternative SSRI (fluvoxamine or paroxetine) or the SNRI venlafaxine at the next step, and a MAOI (phenelzine or moclobemide) after that (NICE 2013). CANMAT lists first-line agents as escitalopram, fluvoxamine (including

CR), paroxetine (including CR), sertraline, venlafaxine XR or pregabalin, with CBT (cognitive restructuring plus exposure) as the gold-standard first-line psychotherapy (CANMAT 2014). The Indian Psychiatric Society does publish CPGs across the anxiety disorders and champions SSRI-plus-CBT for the anxiety block, but a dedicated social-anxiety-disorder recommendation could not be confirmed in the verified supplement, so its precise position is flagged under gaps rather than asserted here. The shared exam message: *SSRI first-line and CBT everywhere*.

| GUIDELINE | FIRST-LINE | KEY NOTE |
|---|---|---|
| **BAP 2014** | **SSRI (escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline); CBT** | Do not use atenolol or buspirone for generalised SAD |
| **NICE 2013** | **Individual CBT (Clark & Wells / Heimberg); SSRI (escitalopram or sertraline) if medication preferred** | Do not routinely offer benzodiazepines/TCAs/antipsychotics |
| **CANMAT 2014** | SSRI/venlafaxine XR/pregabalin; CBT first-line | Beta-blockers not for generalised SAD; may help discrete performance |
| **Indian Psychiatric Society** | SSRI/SNRI with CBT (anxiety-block position) | Dedicated SAD recommendation unconfirmed (see gaps) |

First-line treatment for social anxiety disorder; the shared message is an SSRI (or SNRI) plus CBT, with the beta-blocker reserved for discrete performance situations only.

RULE **SSRI plus CBT is first-line for social anxiety disorder.** BAP names an SSRI (escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline) first-line with CBT of similar efficacy; NICE puts disorder-specific CBT first and an SSRI if medication is preferred; CANMAT agrees. Venlafaxine, phenelzine and moclobemide are the antidepressant alternatives.

GOOD TO REMEMBER

- **SSRI in social anxiety disorder:** the mechanism is serotonergic (serotonin reuptake blockade with downstream receptor adaptation); it is first-line for the generalised disorder (escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline per BAP), needs an adequate trial before response is judged, and, once effective, is continued for at least a further 6 months because social anxiety disorder is chronic with high symptom re-emergence on stopping.

### 5.7 Propranolol for discrete performance anxiety, and what NOT to use

**The one situational role.** For discrete **performance anxiety**, a beta-blocker taken before the feared event is a recognised as-needed option. Propranolol (Indian brands Ciplar, Inderal), a non-selective beta-adrenergic blocker, is long-established as useful particularly for the performance situation, because it damps the peripheral autonomic symptoms, hand tremor and tachycardia, that the performer both feels and fears will be seen (Kaplan and Sadock 2024). The critical caveat: it has *no* effect on anticipatory anxiety or on the generalised disorder, so its use is strictly the discrete, foreseeable performance, taken before going on.

**The important negatives.** The beta-blocker is explicitly *not* a treatment for generalised social anxiety disorder: BAP says do not prescribe atenolol for it, and CANMAT lists propranolol and

atenolol among agents not recommended for the generalised disorder while conceding beta-blockers "may still help discrete performance situations" (BAP 2014; CANMAT 2014). Buspirone, too, is not efficacious as monotherapy for generalised social anxiety disorder (BAP 2014). And the benzodiazepine is not first-line and should not be a maintenance treatment: NICE says do not routinely offer benzodiazepines (alongside TCAs, anticonvulsants and antipsychotics) for social anxiety disorder in adults (NICE 2013).

**WORKED EXAMPLE**

A 24-year-old violinist has crippling tremor and a pounding heart only in the minutes before a solo recital; away from the stage she is socially at ease and has no other feared situations. This is the performance-only picture. The correct move is a beta-blocker, propranolol taken before the performance, to blunt the tremor and tachycardia she fears will be visible; it will not touch anticipatory dread and is not a daily treatment. Contrast a 30-year-old who avoids conversations, meetings, eating out and phone calls across his whole life, the generalised disorder, where the answer is an SSRI plus CBT, and a beta-blocker would be the wrong tool.

**GOOD TO REMEMBER**

- **Propranolol (Ciplar, Inderal) for performance anxiety:** a non-selective beta-blocker that damps the peripheral autonomic signs (tremor, tachycardia) of discrete performance anxiety when taken before the event; the signature caution is that it does nothing for anticipatory anxiety or for generalised social anxiety disorder, for which it is not recommended; check for asthma and bradycardia before prescribing.

### 5.8 CBT: the first-line psychotherapy and the partial-response rule

**What disorder-specific CBT does.** CBT is the first-line psychological treatment; NICE specifies *individual* CBT developed for social anxiety disorder, following the Clark and Wells or the Heimberg model, over group CBT, which it found less clinically and cost effective (NICE 2013). Its ingredients are cognitive restructuring of the fear of negative evaluation and the self-focused, distorted self-image, plus graded exposure to feared social situations, and it targets the safety behaviours (over-rehearsing, avoiding eye contact, gripping the glass) that maintain the belief that catastrophe was only just averted. Delivered well, it produces durable benefit that can outlast medication.

**The partial-response rule.** A clean examinable step: for an adult whose symptoms have only *partially* responded to an SSRI after **10 to 12 weeks**, NICE says add individual CBT to the SSRI; for a partial response to CBT, consider adding a pharmacological intervention (NICE 2013). So the initial trial length before judging partial response is 10 to 12 weeks, and the response to incomplete benefit is to combine, not to abandon.

**GOOD TO REMEMBER**

- **CBT for social anxiety disorder:** the principle is cognitive restructuring of the fear of negative evaluation plus graded exposure and the dropping of safety behaviours (Clark and Wells or Heimberg model); it is first-line and individual is preferred over group (NICE); the signature rule is that partial response to an SSRI at 10 to 12 weeks is managed by *adding* CBT, not switching away.

**INDIA**

Social anxiety disorder sits inside India's large, mostly untreated anxiety burden, most sufferers never reach formal care, and it is easily mistaken for "shyness" or a personality trait and left undiagnosed for years. It also collides with the country's benzodiazepine over-prescription problem: alprazolam and clonazepam are dispensed freely, often long-term, for "nervousness" before social or performance situations, which is exactly what NICE and BAP warn against, the benzodiazepine is not first-line and not a maintenance treatment. The correct Indian answer is a low-cost, widely available SSRI plus CBT for the generalised disorder, sertraline (Serta, Zosert, Daxid), escitalopram (Nexito, Cipralex), paroxetine (Paxidep) or fluoxetine (Fludac), with propranolol (Ciplar, Inderal) reserved for the discrete performer. The Indian Psychiatric Society CPGs anchor the SSRI-plus-CBT approach for the anxiety disorders; a dedicated social-anxiety-disorder recommendation could not be confirmed in the verified supplement and is flagged rather than invented.

| 24 | 6 months | 10 to 12 weeks |
|---|---|---|
| LSAS ITEMS (FEAR + AVOIDANCE, EACH RATED 0 TO 3) | DSM-5-TR TYPICAL DURATION FOR SOCIAL ANXIETY DISORDER | SSRI TRIAL BEFORE ADDING CBT FOR PARTIAL RESPONSE (NICE) |

**HOLD THIS**

Social anxiety disorder is fear of negative evaluation under scrutiny: SSRI (escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline) plus CBT first-line for the generalised disorder, propranolol before the event for discrete performance anxiety only, and never a benzodiazepine as maintenance.

## 6 · Specific phobias

The **specific phobia** is the purest of the anxiety disorders: a fear that is not free-floating, not episodic and not social, but bound tight to one circumscribed object or situation, so that the anxiety switches on the moment the thing is met and switches off the moment it is gone. It is also the commonest anxiety disorder in the community and the anxiety disorder examiners most like to test as a discrimination exercise, because the whole diagnosis turns on a single feature, the **circumscribed phobic stimulus**, and because it carries two facts that appear nowhere else in the anxiety block: the diphasic vasovagal response of the **blood-injection-injury** subtype, and the **applied-tension technique** that is its bespoke treatment.

**Why it matters, and what this note owns.** Specific phobia is the disorder against which the other anxiety disorders are carved, GAD is diffuse, panic is episodic and unexpected, social anxiety fears evaluation, agoraphobia fears escape being difficult, whereas specific phobia is cued and only cued. What this note owns is the subtype classification (with the fainting subtype and its coping skill), the DSM-5-TR and ICD-11 criteria, and the exposure-based treatment that is the treatment of choice. The general CBT model and the wider exposure principle behind all of this are developed in the Psychotherapies, clinical assessment, MSE and nosology notes and are not re-derived here; the amygdala and fear-circuit anatomy that lights up to a phobic stimulus lives in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes.

### 6.1 The core concept: a cued, circumscribed, out-of-proportion fear

**The defining quality.** The essence of a specific phobia is that the fear is *circumscribed* to a particular object or situation, the phobic stimulus, and is out of proportion to the actual danger it poses (DSM-5-TR 2022; ICD-11 CDDR). Unlike the other anxiety disorders, no specific dysfunctional cognition defines it: the person is simply and immediately afraid of the thing itself. The fear is evoked *nearly every time* the stimulus is met (it is reliable and cued, not "out of the blue"), it comes on immediately rather than being delayed, and it drives active avoidance, or, where the situation cannot be avoided, endurance with intense distress.

**The old name.** The specific phobia was called simple phobia in earlier classifications (DSM-III-R and ICD-10 "simple phobia"), and the terms are interchangeable in exam answers; "specific" replaced "simple" to signal the tie to a specific stimulus rather than any simplicity of the condition.

**GOOD TO REMEMBER**

- **Specific phobia (simple phobia):** a marked, out-of-proportion fear cued reliably and immediately by one circumscribed object or situation, leading to active avoidance or distressed endurance; the defining discriminator is that the fear is *bound to the stimulus* (cued and only cued), with no defining cognition, which separates it from the diffuse worry of GAD, the unexpected attacks of panic, and the evaluation fear of social anxiety.

### 6.2 The five subtypes: animal, natural environment, blood-injection-injury, situational, other

**The DSM-5-TR specifiers.** Specific phobia is coded by the type of phobic stimulus, and DSM-5-TR recognises five families (DSM-5-TR 2022): **animal** (spiders, insects, dogs), **natural environment** (heights, storms, water, darkness), **blood-injection-injury** (blood, needles, invasive medical or dental procedures, injury), **situational** (aeroplanes, lifts, enclosed places, driving), and **other** (situations that might lead to choking or vomiting; in children, loud sounds or costumed characters). When more than one stimulus is feared, each is coded separately, and this is the rule rather than the exception: the average patient with specific phobia fears three objects or situations, and roughly three-quarters fear more than one.

**The classic eponyms.** The Greek-derived names are worth recognising: acrophobia (heights), zoophobia (animals), claustrophobia (enclosed spaces), algophobia (pain), xenophobia (strangers), and, of the fainting subtype, the fear of blood, injection and injury. The list of named phobias is virtually endless, but the exam-relevant work is done by the five subtypes, not the eponyms.

| SUBTYPE | TYPICAL STIMULI | AUTONOMIC SIGNATURE AND PEARL |
|---|---|---|
| **Animal** | Spiders, insects, dogs, snakes | Sympathetic arousal; earliest onset, more common in younger children and in females |
| **Natural environment** | Heights, storms, water, darkness | Sympathetic arousal; younger onset for water/weather |
| **Blood-injection-injury** | Blood, needles, injury, medical/dental procedures | **Diphasic vasovagal response with fainting; the only subtype with equal sex ratio and a familial pattern; treat with applied tension** |
| **Situational** | Aeroplanes, lifts, enclosed places, driving | Sympathetic arousal; older onset (flying, heights); overlaps with panic/agoraphobia |
| **Other** | Choking, vomiting; in children loud sounds, costumed figures | Heterogeneous residual category |

The five DSM-5-TR specific-phobia subtypes; the one that carries all the special facts is blood-injection-injury (DSM-5-TR 2022). ICD-11 6B03 sets no subtype specifiers, its only specifier being "with panic attacks".

**GOOD TO REMEMBER**

- **Subtypes:** animal, natural environment, blood-injection-injury, situational and other; the average patient fears three stimuli and each is coded separately; the blood-injection-injury subtype is the exam favourite because it alone shows the diphasic vasovagal faint, has an equal sex ratio, runs in families, and is treated with applied tension rather than exposure alone.

### 6.3 The blood-injection-injury subtype: the diphasic vasovagal response

**Why it is unique.** Every other phobia produces plain sympathetic arousal, a racing heart and rising blood pressure that stay up until escape. The blood-injection-injury subtype does something different and diagnostic: it produces a **diphasic** (biphasic) autonomic response. There is a brief initial acceleration of heart rate and a rise in blood pressure, followed by an abrupt *deceleration* of heart rate and a *fall* in blood pressure, a vasovagal reflex that commonly ends in fainting (vasovagal syncope) at the sight of blood, a needle or an injury (DSM-5-TR 2022). This is the one anxiety presentation in which the patient does not run but falls: the fainting is the presenting feature and the reason the person avoids donating blood, having dental work, or attending for injections.

**The other quirks.** Two more facts hang off this subtype and are heavily tested. First, sex ratio: whereas specific phobias overall run about 2:1 female-to-male, the blood-injection-injury subtype affects men and women roughly equally (ICD-11 CDDR). Second, it has the strongest familial pattern of the phobias, a heritable tendency to the vasovagal faint. Recognising the diphasic pattern matters clinically because the ordinary phobia instruction, "relax", would lower blood pressure further and precipitate the faint the patient fears.

RULE **Blood-injection-injury faints; the rest run.** Only the blood-injection-injury subtype shows the diphasic vasovagal response (a brief pressor phase then a bradycardic hypotensive drop to syncope); every other subtype shows sustained sympathetic arousal, so recognising the faint identifies the subtype and dictates applied tension rather than relaxation.

GOOD TO REMEMBER

- **Blood-injection-injury phobia:** the criterion-defining feature is the diphasic vasovagal response, an initial brief rise in heart rate and blood pressure followed by a vagally mediated fall in both, ending in fainting at the sight of blood/needles/injury; it uniquely has an equal sex ratio and the strongest familial loading, and it is the one subtype where relaxation is contraindicated and applied tension is the treatment of choice.

### 6.4 Applied tension: the bespoke treatment for the fainting subtype

**What it is.** Applied tension is the coping skill developed by Lars-Goran Ost and colleagues specifically for the blood-injection-injury subtype (Ost, Sterner and Fellenius 1989). The patient is taught to repeatedly *tense* the large muscle groups of the arms, legs and trunk for about 10 to 15 seconds, release to normal (not to relaxation) for 20 to 30 seconds, and repeat five times, applying the technique at the first sign of the pressure drop when confronting the phobic stimulus. The tensing raises blood pressure, counteracts the vasovagal fall, and thereby prevents the faint that maintains the avoidance.

**Why it beats plain exposure here.** Ost concluded that applied tension should be the treatment of choice for blood phobia, and a dismantling study showed that tension alone was almost as effective as full applied tension (tension plus exposure), while exposure alone was considerably less effective (Craske, Antony and Barlow 2006). The teaching point is that in this one subtype the coping skill, keeping the blood pressure up, matters more than habituation, which is exactly why the generic "relax during exposure" instruction is wrong here.

COMMON TRAP

Do not teach relaxation to a blood-injection-injury phobic. Relaxation lowers blood pressure and, in a patient whose problem is a vasovagal pressure drop to syncope, it precipitates the very faint being treated. The correct skill is applied tension (repeated large-muscle tensing to raise blood pressure), not relaxation; confusing the two is the classic exam and clinical error for this subtype.

GOOD TO REMEMBER

- **Applied tension (Ost):** the treatment of choice for the blood-injection-injury subtype; repeated tensing of large muscle groups (about 10 to 15 seconds on, 20 to 30 seconds off, five cycles) raises blood pressure and aborts the vasovagal faint; the mechanism is counteracting the pressure drop, not habituation, so it is used *instead of* relaxation and is combined with graded exposure to the feared stimulus.

### 6.5 DSM-5-TR criteria: marked fear, immediate, avoided, out of proportion, six months

**The criteria to state cold.** DSM-5-TR requires a marked fear or anxiety about a specific object or situation (Criterion A); the phobic stimulus almost always provokes immediate fear or anxiety (B);

the stimulus is actively avoided or endured with intense fear (C); the fear is out of proportion to the actual danger and the sociocultural context (D); the fear, anxiety or avoidance is persistent, typically **6 months or more** (E); it causes clinically significant distress or impairment (F); and it is not better explained by another mental disorder (G) (DSM-5-TR 2022). In children the fear may present as crying, tantrums, freezing or clinging, and the child need not recognise the fear as excessive.

**The two discriminators inside the criteria.** Criterion B ("almost always" and "immediate") is what separates a true phobia from an occasional situational nerviness: the person who is anxious on only one flight in five does not qualify. Criterion D moves the judgement of "out of proportion" to the clinician, taking cultural context into account (a fear of the dark may be reasonable where night-time assault is common).

| CRITERION | DSM-5-TR REQUIREMENT |
|---|---|
| **A. Marked fear** | **Marked fear/anxiety about a specific object or situation (in children: crying, tantrums, freezing, clinging)** |
| **B. Immediate/reliable** | **The phobic stimulus almost always provokes immediate fear or anxiety** |
| **C. Avoidance** | Actively avoided, or endured with intense fear or anxiety |
| **D. Out of proportion** | Fear out of proportion to actual danger and to sociocultural context (clinician judges) |
| **E. Duration** | **Persistent, typically 6 months or more** |
| **F. Impairment** | Clinically significant distress or functional impairment |
| **G. Not better explained** | Not better explained by another disorder (agoraphobia, OCD, PTSD, separation anxiety, social anxiety) |

DSM-5-TR criteria for specific phobia; the numbers to hold are the 6-month duration and the "almost always / immediate" reliability of Criterion B (DSM-5-TR 2022).

**GOOD TO REMEMBER**

- **Specific phobia (DSM-5-TR):** marked fear of a circumscribed stimulus that almost always and immediately provokes anxiety (Criterion B), is avoided or endured with distress, is out of proportion to the real danger, persists 6 months or more, and impairs functioning; the first diagnostic step is to confirm the fear is *cued and reliable* and then to exclude that it is better explained by agoraphobia, OCD, PTSD, separation or social anxiety (Criterion G).

### 6.6 ICD-11 CDDR and the differential: what to exclude

**Where ICD-11 sits.** The ICD-11 CDDR essential features mirror DSM closely: marked and excessive fear or anxiety that consistently occurs on exposure or anticipated exposure to one or more specific objects or situations, out of proportion to the actual danger; the stimulus is actively avoided or endured with intense fear; the pattern is not transient, persisting for at least **several**

**months**; it is not better accounted for by another disorder; and it causes significant distress or impairment (ICD-11 CDDR). As across the anxiety block, ICD-11 uses the softer "at least several months" rather than DSM's fixed 6-month clock.

**The differential.** The work is in Criterion G. If the avoided situations are feared because escape would be difficult or help unavailable in a panic, it is agoraphobia. If the fear is of contamination or driven by an obsession, it is OCD, not a phobia (the hand-washer who avoids door handles has OCD). If the avoidance is of trauma reminders, it is PTSD. If it is fear of scrutiny and negative evaluation, it is social anxiety disorder. If it is fear of separation from an attachment figure, it is separation anxiety. And medical mimics of situational fear (for example genuine cardiac or vestibular disease presenting as fear of exertion or heights) are excluded first.

**GOOD TO REMEMBER**

- **ICD-11 specific phobia:** marked, excessive, out-of-proportion fear consistently cued by one or more specific stimuli, actively avoided or endured with intense fear, not transient (at least several months), with distress or impairment; the discriminator from its neighbours is the *reason* for the fear, of the stimulus itself in phobia, versus escape difficulty (agoraphobia), obsessions (OCD), trauma reminders (PTSD), evaluation (social anxiety) or separation (separation anxiety).

### 6.7 Epidemiology and course: the commonest anxiety disorder, earliest onset

**The numbers.** Specific phobia is the most prevalent anxiety disorder, with a 12-month community prevalence of about **8 to 12 per cent** in the United States (around 6 per cent in Europe, lower at 2 to 4 per cent in Asian, African and Latin American surveys) (DSM-5-TR 2022). It is roughly **twice as common in women** as in men across most subtypes, the exception being blood-injection-injury, which is equal. Onset is early, most commonly in childhood between 7 and 10 years of age, and specific phobias that persist from childhood rarely remit spontaneously.

**Course and comorbidity.** Animal and natural-environment phobias tend to the earliest onset; situational phobias such as flying and heights begin later. Patients report high lifetime rates of co-occurring depressive and other anxiety disorders, and in most cases the specific phobia comes *first* and precedes the other disorder, which is why it is a useful early marker.

| 8 to 12% | 6 months | 2:1 | 7 to 10 yrs |
|---|---|---|---|
| 12-MONTH US PREVALENCE, THE COMMONEST ANXIETY DISORDER | DSM-5-TR MINIMUM DURATION (ICD-11: SEVERAL MONTHS) | FEMALE-TO-MALE RATIO (EQUAL IN BLOOD-INJECTION-INJURY) | TYPICAL AGE OF ONSET |

### 6.8 Treatment: graded in-vivo exposure is the treatment of choice

**Behavioural first, not drugs.** The treatment of choice for specific phobia is *exposure-based* behaviour therapy, not medication; meta-analyses show very large effect sizes that are stable for at least a year (Craske, Antony and Barlow 2006). The workhorse is graded in-vivo exposure: the patient builds an **exposure hierarchy** (feared tasks rated 0 to 100 for anxiety, ordered from least to most provoking) and then repeatedly, systematically confronts the real phobic stimulus, starting low and climbing the ladder, until the feared catastrophe fails to occur and new, non-fearful learning takes hold. Modern practice reframes the goal not as staying until fear declines

but as staying long enough to *learn* that the feared outcome does not happen and that anxiety can be tolerated (inhibitory-learning model).

**How fast it works.** Specific phobia is unusually treatable: animal and blood phobias often respond to a single, prolonged session of exposure lasting two to three hours (Ost). No routine drug is indicated; a short-acting benzodiazepine or beta-blocker is at most an occasional aid for a discrete one-off exposure (for example a single flight) and is never the treatment, because it undermines the corrective learning that exposure depends on.

TRAP **Do not reach for a drug (or a benzodiazepine) as the treatment for a specific phobia.** The evidence-based treatment is graded in-vivo exposure; medication is not first-line, and a benzodiazepine taken to get through exposure blunts the fear-extinction learning that makes exposure work, so it undermines the very treatment.

GOOD TO REMEMBER

- **Graded in-vivo exposure:** the treatment of choice for specific phobia; the principle is repeated, systematic confrontation with the real phobic stimulus up a rated exposure hierarchy until the feared outcome is disconfirmed and anxiety is tolerated (inhibitory learning); it can work in a single 2 to 3 hour session for animal and blood phobias, and drugs are not first-line.

### 6.9 Systematic desensitisation and flooding: the two ends of the exposure spectrum

**Systematic desensitisation.** Systematic desensitisation is Joseph Wolpe's original method (Psychotherapy by Reciprocal Inhibition, 1958): the patient is taught deep muscle relaxation, an anxiety hierarchy is constructed, and the phobic stimuli are then paired, one graded step at a time, with the incompatible relaxation response, usually imaginally at first. Wolpe's mechanism was **reciprocal inhibition** (counter-conditioning): the relaxation response inhibits the anxiety response so the stimulus can be faced without fear. It is *gradual* and pairs each step with relaxation. Note the practical implication for the fainting subtype: because desensitisation uses relaxation, blood-injection-injury phobia is instead paired with applied tension.

**Flooding.** Flooding is the opposite pole: the patient is exposed to the most feared stimulus at full intensity from the outset, with no hierarchy and no graded climb, and stays in the situation until the anxiety, which cannot be sustained indefinitely, extinguishes. It is faster but more distressing and less tolerable, so graded exposure and desensitisation are generally preferred. The three methods sit on one spectrum, gradual-with-relaxation (systematic desensitisation), gradual real-life (graded in-vivo exposure), and all-at-once (flooding), and share the single active ingredient of exposure with prevention of avoidance.

MNEMONIC

**SAFE-D**

The specific-phobia essentials. **S**pecific circumscribed stimulus (cued, immediate, Criterion B); **A**void or endure with distress; **F**ive subtypes (animal, natural environment, blood-injection-injury, situational, other); **E**xposure is the treatment (graded in-vivo, systematic desensitisation, flooding); **D**iphasic vasovagal faint marks blood-injection-injury, treated with applied tension.

**GOOD TO REMEMBER**

- **Systematic desensitisation (Wolpe):** graded exposure up an anxiety hierarchy paired with deep relaxation; mechanism is reciprocal inhibition (relaxation inhibits anxiety); signature caution is that its reliance on relaxation makes it unsuitable, as-is, for the blood-injection-injury subtype, where applied tension replaces relaxation.
- **Flooding:** ungraded exposure to the most feared stimulus at full intensity from the outset until anxiety extinguishes; faster than graded methods but poorly tolerated, so it is not first-choice; the active ingredient it shares with all exposure is sustained contact with prevention of avoidance.

### 6.10 The India lens: a common, treatable disorder lost in the treatment gap

**The genuine Indian point.** Specific phobia is common, it presents early, and it is one of the most treatable conditions in psychiatry, a few sessions of exposure can resolve a fear that has constrained a life for decades. Yet in India it sits squarely inside the anxiety treatment gap: most people with a phobia never reach care, the fear is normalised or somatised, and when they do present the reflex prescription in general and primary care is a benzodiazepine (typically alprazolam), which is neither first-line nor a treatment for phobia at all and only undermines the exposure learning that would actually help. The correct Indian answer mirrors the global one: graded in-vivo exposure is the treatment, applied tension for the fainting subtype, and the benzodiazepine is at most a rare, single-exposure aid, never the plan. Where a drug is genuinely considered a beta-blocker such as propranolol (Indian brands Ciplar, Inderal) can blunt peripheral autonomic symptoms for a discrete situational exposure, but it too is an adjunct, not the treatment.

**INDIA**

The highest-yield Indian point is that specific phobia is common, early-onset and highly treatable with a short course of exposure, yet it is routinely lost in the anxiety treatment gap and met, when it does present, with a long-term benzodiazepine (usually alprazolam) that runs against the Indian Psychiatric Society CPG stance on anxiety and actively undermines the exposure-based learning that cures phobia. The right answer stays behavioural: graded in-vivo exposure and systematic desensitisation, applied tension for the blood-injection-injury faint, propranolol (Ciplar, Inderal) at most as an autonomic adjunct for a one-off situational exposure, and the benzodiazepine reserved and time-limited if used at all.

**HOLD THIS**

Specific (simple) phobia is a cued, out-of-proportion fear of one circumscribed stimulus that almost always and immediately provokes anxiety (DSM-5-TR: 6 months; ICD-11: several months), classified in DSM-5-TR into five subtypes (animal, natural environment, blood-injection-injury, situational, other); its treatment is exposure-based behaviour therapy, graded in-vivo exposure first, with systematic desensitisation (Wolpe, reciprocal inhibition) and flooding on the same spectrum, and the blood-injection-injury subtype alone shows the diphasic vasovagal faint and is treated with applied tension, never relaxation.

## 7 · Separation anxiety disorder

**A childhood diagnosis that grew up.** **Separation anxiety disorder** is the excessive, developmentally inappropriate fear of separation from an **attachment figure**. The single highest-yield fact for the exam is a nosological one: both the ICD-11 and the DSM-5 now list it as a **lifespan anxiety** disorder inside the anxiety and fear-related grouping, no longer walled off in the childhood-onset chapter. Examiners test the placement change, the criterion-anchored definition, and the boundary with normal developmental separation anxiety.

Keep the discrimination sharp. The apprehension is focused specifically on **separation from an attachment figure** (a parent in a child; a spouse, partner or one's own child in an adult), which is what sets it apart from panic disorder, agoraphobia and generalised anxiety. Treatment mirrors the wider anxiety toolkit, so the management detail lives in the neighbouring topics; this note is deliberately brief and criterion-led.

### 7.1 Definition and the core fear

**Attachment-focused, not situation-focused.** The disorder is characterised by marked, excessive and developmentally inappropriate fear or anxiety about separation from those to whom the person is emotionally bonded, with catastrophic beliefs about what separation will bring (harm, illness, an untoward event, never being reunited). Attachment figures are usually parents in children and a spouse, romantic partner or one's own children in adults.

### 7.2 The ICD-11 placement change (the exam hook)

**Out of the childhood chapter, into anxiety.** In DSM-IV and ICD-10, separation anxiety sat among disorders first diagnosed in infancy, childhood or adolescence (ICD-10 F93.0, "separation anxiety disorder of childhood"). The **icd-11 placement** moves it to code 6B05 within anxiety and fear-related disorders, and DSM-5 places it among the anxiety disorders. The reclassification reflects that the disorder can begin, or persist, in adulthood.

### 7.3 Lifespan reframe: no upper age bar

**Adults get the diagnosis too.** Unlike earlier editions, DSM-5 dropped the requirement for onset before 18 years and recognises that some adults report onset after 18. This is the substantive change behind the "lifespan" framing: an adult with excessive fear of being apart from a spouse or child, meeting criteria, is diagnosed here rather than being forced into a residual category.

### 7.4 The symptom set and threshold

**Three of eight.** DSM-5-TR requires at least three of eight symptoms: recurrent excessive distress on anticipated or actual separation; worry about losing the attachment figure or about harm to them; worry that an untoward event will cause separation; reluctance or refusal to leave home, school or work; fear of being alone; reluctance or refusal to sleep away from the attachment figure; nightmares about separation; and physical complaints (nausea, stomach ache, headache) on separation.

| AXIS | DSM-5-TR | ICD-11 (6B05) |
|---|---|---|
| Chapter | Anxiety disorders (not childhood chapter) | Anxiety and fear-related disorders |
| Age scope | No onset-before-18 rule; adults included | Lifespan; adult onset recognised |
| Symptom threshold | At least 3 of 8 | Marked fear plus characteristic features |
| Duration, children/adolescents | At least 4 weeks | Not transient (at least several months) |
| Duration, adults | At least 6 months (typically) | At least several months |
| Impairment | Distress or functional impairment | Distress or functional impairment |

The two systems agree on substance; DSM-5-TR carries the explicit symptom count and duration thresholds most often examined.

### 7.5 Duration criterion

**Four weeks in the young, six months in adults.** DSM-5-TR asks for symptoms lasting at least **4 weeks** in children and adolescents and typically at least **6 months** in adults, the longer adult window added to avoid over-diagnosing transient fears (for example after a bereavement or a move). ICD-11 phrases this as symptoms that are "not transient," persisting for at least several months.

### 7.6 Boundary with normal developmental separation anxiety

**Normal is time-limited and age-expected.** Developmentally normal separation anxiety appears around **6 months** and declines between two and three years of age; it is a healthy marker of attachment. The disorder is diagnosed only when the fear is out of proportion to the child's developmental level, persists across repeated separations, and produces genuine distress or impairment (school refusal, inability to sleep alone, somatic complaints on parting).

RULE **Focus of fear decides the diagnosis.** If the apprehension is specifically about separation from an attachment figure, it is separation anxiety disorder, whatever the patient's age.

TRAP **Do not treat it as childhood-only.** Reflexively excluding the diagnosis in an adult (or over-calling normal toddler clinginess as a disorder) are the two classic errors since the placement change.

WORKED EXAMPLE

A 34-year-old woman cannot let her husband travel for work; she rings him repeatedly, is convinced he will be harmed while apart, cannot fall asleep until he is home, and has skipped her own commitments to stay near him for the past eight months. The apprehension is separation-focused, adult-onset, and beyond six months with clear impairment. This is separation anxiety disorder, not generalised anxiety, whose worry would be diffuse rather than pinned to one attachment figure.

**INDIA**

Anxiety disorders carry a large untreated **treatment gap** in India, and much of what does reach treatment is met with benzodiazepines: the **benzodiazepine over-prescription** problem is well recognised, with clonazepam and alprazolam handed out as first-line and long-term anxiolytics against guidance. The **IPS Clinical Practice Guidelines** and the international consensus are unambiguous that an SSRI (available here under brands such as sertraline, escitalopram and fluoxetine), with CBT or exposure, is first-line for the anxiety disorders, and that a benzodiazepine is neither first-line nor for long-term use. Separation anxiety disorder, presenting through a clingy child brought for "school refusal" or an adult brought by an exasperated spouse, is easily missed or sedated rather than diagnosed.

| 3 of 8 | 4 wk | 6 mo |
|---|---|---|
| DSM-5-TR SYMPTOM THRESHOLD | MINIMUM DURATION IN CHILDREN | TYPICAL MINIMUM DURATION IN ADULTS |

**MNEMONIC**

### A-P-A-R-T

**A**ttachment-figure fear is the core; **P**lacement now under anxiety in ICD-11 and DSM-5; **A**dults included (lifespan, no age bar); **R**equires three of eight symptoms; **T**ime threshold four weeks in children, about six months in adults.

**PEARL**

School refusal is a presentation, not a diagnosis. Separation anxiety disorder is one of its commonest drivers in younger children; social anxiety disorder takes over as the leading cause into adolescence.

**GOOD TO REMEMBER**

- **Separation anxiety disorder:** defining criterion is excessive, developmentally inappropriate fear focused specifically on separation from an attachment figure, at least three of eight symptoms, four weeks in children and about six months in adults; first step is to check the fear is separation-focused and to place it, correctly, as a lifespan anxiety disorder in ICD-11 (6B05) and DSM-5, no longer a childhood-only diagnosis.

**HOLD THIS**

Separation anxiety disorder is now a lifespan anxiety disorder: ICD-11 and DSM-5 moved it out of the childhood chapter, dropped the onset-before-18 rule, and it is defined by attachment-figure-focused fear meeting three of eight symptoms.

For the shared anxiolytic pharmacology and the benzodiazepine dependence problem, see the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; for SSRI dosing and adverse effects, the Mood disorders: pharmacotherapy notes; for the general CBT and exposure model, the Psychotherapies, clinical assessment, MSE and nosology notes; and for the childhood

attachment disorders that sit nearby, the Child behavioural, emotional and other disorders notes and the Child psychiatry: assessment, treatment, services and protection notes.

### 8 · The neurobiology of anxiety

The **neurobiology of anxiety** is the machinery of a survival system that has become a symptom. Fear is an adaptive, fast, hard-wired response to a present threat; anxiety is its slower, sustained, anticipatory cousin, aimed at a threat that is diffuse, future or imagined. Both run on the same core circuit, a threat-detection network built around the **amygdala**, wired to fire the body's alarm before the thinking cortex has finished deliberating, and normally reined back in by the prefrontal cortex and hippocampus. When the alarm fires too readily, extinguishes too poorly, or is regulated too weakly, the survival system becomes an anxiety disorder. Examiners test this topic as the anatomy of a loop and the chemistry that tunes it: know the amygdala-centred **fear circuit**, its two sensory roads, its outputs to the hypothalamus, the brainstem and the periaqueductal grey, its cortical and hippocampal brakes, and the three transmitter systems, GABA, serotonin and noradrenaline, that the anxiolytic drugs act on.

**Why it matters clinically.** Every drug that works in the anxiety disorders acts somewhere on this circuit, so the neurobiology is not decoration, it is the rationale for the treatment. A benzodiazepine calms anxiety because it potentiates **gaba** inhibition across the amygdala and its targets; an SSRI works because **serotonin anxiety** pathways from the raphe tune the fear circuit over weeks; propranolol blunts performance anxiety by blocking the peripheral noradrenergic surge that the **locus coeruleus** drives. Two ideas anchor the whole answer. First, the amygdala is the hub: it evaluates threat and organises the fear response, and it is normally held in check by the prefrontal cortex, so anxiety disorders are, in circuit terms, an over-active amygdala under-regulated by a weak cortical brake. Second, the circuit has a fast, crude, subcortical route and a slow, accurate, cortical route, so the alarm can fire before you consciously know why. The pathway anatomy and receptor systems are developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; this topic applies that anatomy to the **fear circuitry** of clinical anxiety and refers to the accompanying figure for the map.

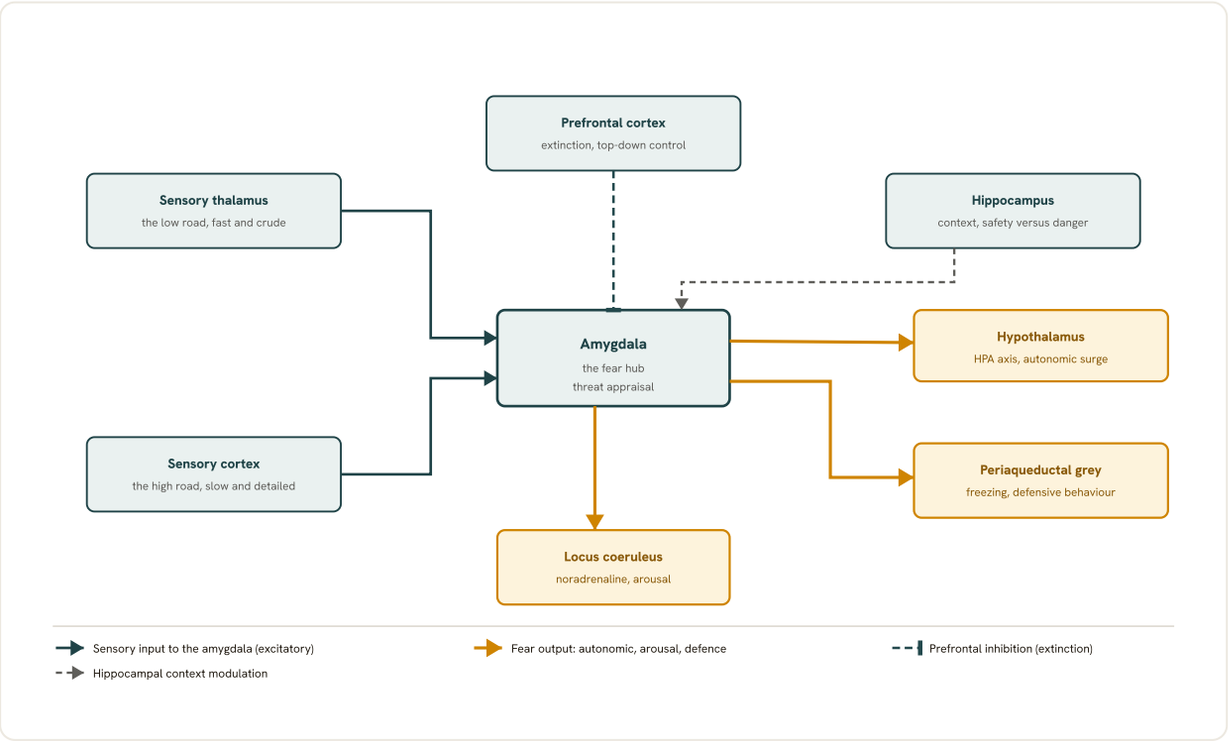



**Fig 14.4** The fear circuit. The amygdala is the hub of the fear response: it receives a fast, crude input from the sensory thalamus (the low road) and a slower, detailed input from the sensory cortex (the high road), and it drives the fear state through the hypothalamus (the HPA axis and the autonomic surge), the periaqueductal grey (freezing and defensive behaviour) and the locus coeruleus (noradrenergic arousal). The prefrontal cortex exerts top-down inhibition, the basis of fear extinction, and the hippocampus supplies the context that separates safety from danger. A hyperresponsive amygdala with weak prefrontal control is the core lesion across the anxiety disorders. The receptor systems and the pathway anatomy are taught in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes.

## 8.1 Fear versus anxiety: the same circuit, two timescales

**The distinction to open with.** *Fear* is the response to a present, identifiable threat, phasic, fast, and organised largely by the central nucleus of the amygdala; *anxiety* is the response to a potential, diffuse or future threat, sustained, slower to build and slower to settle, and organised more by the bed nucleus of the stria terminalis (BNST), the amygdala's "extended" partner (Kaplan & Sadock CTP 2024). The BNST mediates the longer-duration apprehension elicited by less explicit or poorly-defined cues and contexts, which is why it is called the substrate of sustained anxiety rather than acute fear. Holding this apart matters because the clinical disorders are diseases of the anxiety end, a threat-detection system left switched on when no discrete threat is present.

**One machine, one vulnerability.** Because fear and anxiety share the amygdala hub, a system tuned to over-respond will produce both the acute false alarm (a panic attack) and the chronic apprehensive tension (generalised worry) in the same patient, which is why the anxiety disorders cluster and co-occur.

## 8.2 The amygdala as the hub: the lateral nucleus takes the input

**The sensory gateway.** The amygdala is an almond-shaped cluster of subnuclei (the amygdaloid complex) in the medial temporal lobe, and it is the pivot of the fear circuit. Its **lateral nucleus** (LA) is the primary sensory interface: it receives incoming sensory information from both the

sensory thalamus and the sensory cortex, and single LA neurons respond to auditory, visual and somatic stimuli, with the conditioned stimulus (the cue) and the unconditioned stimulus (the threat) converging there (Kaplan & Sadock CTP 2024). The LA is where a neutral cue first acquires threat value through **fear conditioning**, a form of synaptic plasticity (long-term potentiation) in the amygdala.

**Distribution inside the complex.** From the LA the signal is distributed to the basal and accessory basal nuclei and to the central nucleus (CE); the direct LA-to-CE projection can by itself drive conditioning to a simple cue, while the LA-to-basal projections build the longer-term fear memory (Kaplan & Sadock CTP 2024). The CE is the output nucleus, the amygdala's motor for the fear response.

PEARL

Think of the amygdala as a two-door hub. The *lateral nucleus* is the *in-door*, where thalamic and cortical sensory streams arrive and a cue is tagged as dangerous. The *central nucleus* is the *out-door*, from which the fear response is broadcast to the hypothalamus, brainstem and periaqueductal grey. Everything else, the fast road, the slow road, the cortical brake, is wiring around those two doors.

### 8.3 LeDoux's two roads: the low road and the high road

**The fast, crude subcortical route.** Joseph LeDoux mapped two parallel inputs to the amygdala. The *low road* runs from the sensory **thalamus** directly to the lateral amygdala, bypassing the cortex; it is fast and imprecise, a "quick and dirty" pathway that supports rapid conditioning to simple stimuli and permits an unconscious fear response before the stimulus is consciously identified (Kaplan & Sadock CTP 2024). Lesions of the auditory cortex do not prevent conditioning to a single tone, which is the experimental signature of the low road.

**The slow, accurate cortical route.** The *high road* runs from the sensory thalamus to the primary sensory and association **cortex** and then to the lateral amygdala; it is slower but detailed, and it is required for conditioned responding to more complex sensory stimuli and for the higher cognitive appraisal of the threat (Kaplan & Sadock CTP 2024). The two roads together explain the lived experience of fear: you flinch at the coiled shape on the path (low road) a fraction of a second before the cortex tells you it is a garden hose (high road).

WORKED EXAMPLE

A patient with a phobia of snakes freezes and gasps at a curved stick glimpsed in long grass, then within a second recognises it as a stick and feels foolish. The freeze-and-gasp is the low road, the sensory thalamus firing the amygdala directly and triggering the fear response before identification. The relief a moment later is the high road, cortical processing correcting the appraisal. In an anxiety disorder the low road is set too sensitive and the high-road correction is too weak or too slow to abort the alarm.

### 8.4 The output: central nucleus to hypothalamus, brainstem and periaqueductal grey

**The three efferent targets to name.** The central nucleus (CE) is the interface between the amygdala and the body's fear response, projecting to the medulla, midbrain and **hypothalamus** to drive the autonomic, endocrine and behavioural components (Kaplan & Sadock CTP 2024). Three CE outputs are the high-yield trio. To the *hypothalamus*, specifically the paraventricular nucleus (PVN), the amygdala drives release of corticotropin-releasing hormone and thereby the HPA-axis stress response; the projection is bisynaptic and GABAergic. To the **locus coeruleus** in the brainstem, the amygdala drives the noradrenergic surge that produces tachycardia, hypertension and hypervigilance. To the periaqueductal grey (PAG) in the midbrain, it drives the somatic and behavioural defence responses, freezing and avoidance (Gorman's neuroanatomical hypothesis, in Tasman Psychiatry).

**Sustained anxiety takes the BNST branch.** Both the CE and the BNST can directly activate the hypothalamus and brainstem; the BNST branch mediates the longer, context-driven anxiety states while the CE drives the phasic fear response (Kaplan & Sadock CTP 2024). Lesioning a specific CE efferent produces a selective deficit, PAG lesions blunt the somatobehavioural response, lateral hypothalamic lesions blunt the cardiovascular one, which is why the fear response is described as a set of parallel outputs from a single hub.

MNEMONIC

#### HELP the amygdala expresses fear: Hypothalamus, brainstem/LC, PAG

The central nucleus fires fear through its efferents: **H**ypothalamic PVN (CRH and the HPA stress axis), the brainstem **L**ocus coeruleus (noradrenaline, tachycardia, hypervigilance) and the **P**eriaqueductal grey (freezing and avoidance). Naming these three CE outputs is the fastest way to earn the "fear expression" marks.

### 8.5 The prefrontal brake and hippocampal context: how the alarm is switched off

**The cortical brake.** The medial and orbital prefrontal cortex (PFC) modulates the amygdala through extensive reciprocal projections and is the substrate of *fear regulation* and *extinction*, the learned reduction of a fear response once a cue is no longer dangerous (Kaplan & Sadock CTP 2024). The ventromedial PFC (the infralimbic region in the rat) drives extinction: it acts through the amygdala's GABAergic intercalated cells to inhibit central-nucleus output, so a strong vmPFC brake means a fear that can be extinguished. A weak vmPFC brake, or an over-driving dorsal anterior cingulate, means fear that persists, which is the circuit signature of the anxiety disorders and of PTSD.

**Hippocampal context.** The hippocampus supplies the *context* of the fear memory, whether the cue is dangerous *here and now* or only in the original setting, and it is central to contextual fear conditioning and to distinguishing a safe context from a dangerous one (Kaplan & Sadock CTP 2024). When hippocampal contextual gating fails, fear generalises to safe situations, the over-generalisation that characterises PTSD and generalised anxiety.

RULE **Over-active amygdala, under-active prefrontal brake.** The core circuit lesion of the anxiety disorders is an amygdala that fires too readily on threat and a ventromedial prefrontal cortex that regulates and extinguishes that fear too weakly; the hippocampus normally fixes the fear to its correct context, and when it fails, fear over-generalises to safe situations.

### 8.6 The GABA system: the brain's brake and the benzodiazepine target

**Inhibition across the circuit. gaba** (gamma-aminobutyric acid) is the principal inhibitory transmitter of the brain and the physiological brake on the fear circuit: GABAergic interneurons within the amygdala, the GABAergic intercalated cells through which the vmPFC inhibits CE output, and GABAergic tone throughout the limbic system all restrain the alarm (Kaplan & Sadock CTP 2024). A deficiency or reduced sensitivity of GABA-A signalling is a leading model of pathological anxiety.

**Why the benzodiazepine works, and its ceiling.** The benzodiazepine binds an allosteric site on the GABA-A receptor and potentiates GABA's action, increasing the frequency of chloride-channel opening, so it acutely amplifies the brake and calms anxiety within minutes. That immediacy is exactly why it is over-used and why it is *not* a first-line maintenance treatment: it does not correct the circuit, it only sedates it, and chronic use brings tolerance, dependence and rebound anxiety. The detailed anxiolytic pharmacology (benzodiazepines, buspirone, pregabalin, the z-drugs, dependence and withdrawal) is developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes.

GOOD TO REMEMBER

- **GABA and the benzodiazepine (the transmitter and its drug):** GABA is the brain's chief inhibitory transmitter and the physiological brake on the amygdala and fear circuit; the benzodiazepine potentiates GABA at an allosteric GABA-A site (increasing chloride-channel opening frequency), giving a fast anxiolytic effect, the signature caution is that it sedates rather than corrects the circuit, so tolerance, dependence and rebound anxiety follow chronic use and it is never a first-line maintenance drug for an anxiety disorder.

### 8.7 The serotonin system: the slow tuner and the SSRI target

**Raphe modulation of the fear circuit. serotonin anxiety** pathways ascend from the dorsal raphe nucleus of the brainstem to the amygdala, prefrontal cortex, hippocampus and hypothalamus, where serotonin tonically modulates the excitability of the fear circuit (Kaplan & Sadock CTP 2024; New Oxford Textbook of Psychiatry). The relationship is complex rather than simply inhibitory, but the clinical fact is that enhancing serotonergic transmission dampens pathological anxiety over weeks, and the postsynaptic 5-HT1A receptor is a key mediator (the partial agonist buspirone acts here).

**Why the SSRI is first-line and slow.** The SSRI raises synaptic serotonin at once, but the anxiolytic benefit is delayed by several weeks, the lag reflecting adaptive receptor changes and a re-tuning of the circuit rather than acute inhibition. This delayed, circuit-remodelling action is precisely why the SSRI corrects where the benzodiazepine only sedates, and why the SSRI (and SNRI) is first-line across the anxiety disorders and PTSD. The deep antidepressant pharmacology

(the SSRIs, SNRIs, clomipramine, doses, adverse effects, interactions) is developed in the Mood disorders: pharmacotherapy notes.

TRAP **Expecting the SSRI to work like a benzodiazepine.** The serotonergic anxiolytic effect is delayed by weeks and can transiently *worsen* anxiety in the first days (early activation); reaching for a benzodiazepine because "the SSRI is not working yet", or stopping the SSRI too early, is the classic error, warn the patient about the lag and the early jitteriness from the outset.

GOOD TO REMEMBER

- **Serotonin and the SSRI (the transmitter and its drug):** serotonin ascends from the dorsal raphe to tune the amygdala, prefrontal cortex and hippocampus (the 5-HT1A receptor a key mediator); the SSRI raises synaptic serotonin at once but re-tunes the fear circuit only over several weeks, the signature caution is the delayed onset with early activation, and the principle is that it *corrects* the circuit where a benzodiazepine only sedates, which is why the SSRI/SNRI is first-line for the anxiety disorders and PTSD.

### 8.8 The noradrenaline system: the locus coeruleus and the alarm's amplifier

**The brainstem alarm centre.** The **locus coeruleus** (LC) is the principal noradrenergic nucleus of the brain, in the pons, and it is the amplifier of the fear response: activation of the LC raises noradrenaline throughout the forebrain and drives the peripheral autonomic surge, tachycardia, raised blood pressure, sweating and hyperarousal, that the patient feels as the physical face of anxiety (Kaplan & Sadock CTP 2024; Tasman Psychiatry). The amygdala's central nucleus projects to the LC, and the LC in turn feeds arousal back into the circuit, a positive loop that panic disorder in particular exploits.

**The pharmacological proof.** The noradrenergic model is not merely theoretical: yohimbine, an alpha-2 antagonist that increases LC firing and noradrenaline release, provokes panic in vulnerable patients, while clonidine, an alpha-2 agonist that suppresses the LC, reduces arousal (Tasman Psychiatry). Clinically the same physiology explains why propranolol (Ciplar, Inderal), a beta-blocker, blunts the peripheral tremor and tachycardia of *performance* anxiety, and why prazosin (Minipress, Prazopress), an alpha-1 antagonist, dampens the noradrenergic hyperarousal that drives the nightmares of PTSD.

GOOD TO REMEMBER

- **Noradrenaline and the locus coeruleus (the transmitter and its nucleus):** the LC is the pontine noradrenergic alarm centre that amplifies the amygdala's fear output into the peripheral autonomic surge (tachycardia, hypertension, hyperarousal); yohimbine (alpha-2 antagonist) provokes panic and clonidine (alpha-2 agonist) suppresses it, the clinical corollary is that propranolol blunts the peripheral surge of performance anxiety and prazosin dampens the noradrenergic hyperarousal behind PTSD nightmares.

### 8.9 Putting the circuit together: how the transmitters map onto the anatomy

**One picture, three dials.** The fear circuit is one anatomical loop with three chemical dials. GABA is the inhibitory brake wired through the amygdala and the prefrontal intercalated cells; serotonin is the slow tuner descending from the raphe onto the whole circuit; noradrenaline from

the locus coeruleus is the amplifier of the output. An anxiety disorder is, in this scheme, too little brake, mis-tuning of the serotonergic set-point, and too much amplifier, on a hub (the amygdala) that is over-reactive and a cortex (vmPFC) that regulates it too weakly. Every anxiolytic drug is best understood as adjusting one of these dials.

| NODE / SYSTEM | ROLE IN THE FEAR CIRCUIT | CLINICAL / DRUG CORRELATE |
|---|---|---|
| **Lateral nucleus of amygdala (LA)** | Sensory in-door; thalamic + cortical input converge; site of fear conditioning (LTP) | Over-sensitive threat detection in anxiety disorders |
| **Central nucleus of amygdala (CE)** | **Out-door; organises the fear response via efferents** | Lesion abolishes conditioned fear expression |
| **Low road (thalamo-amygdala)** | Fast, crude, pre-conscious alarm; bypasses cortex (LeDoux) | Explains reflex fear before identification |
| **High road (thalamo-cortico-amygdala)** | Slow, accurate cortical appraisal of threat (LeDoux) | Weak or slow high-road correction sustains the alarm |
| **Hypothalamus (PVN)** | **CE efferent; CRH release, HPA stress axis** | Neuroendocrine stress response |
| **Locus coeruleus** | CE efferent; noradrenaline, autonomic surge, hyperarousal | **Yohimbine provokes, propranolol/prazosin blunt** |
| **Periaqueductal grey (PAG)** | CE efferent; freezing and defensive avoidance behaviour | Somatobehavioural fear output |
| **Ventromedial PFC** | **The brake; drives extinction via GABAergic intercalated cells** | Weak brake = fear that will not extinguish (PTSD) |
| **Hippocampus** | Supplies context of the fear memory | Failure = fear over-generalises to safe settings |
| **GABA** | Inhibitory brake across the circuit | Benzodiazepine target (fast, sedating) |
| **Serotonin (raphe)** | Slow tonic tuner of the whole circuit; 5-HT1A | SSRI target (slow, corrective, first-line) |
| **Noradrenaline (LC)** | Amplifier of the fear output | Beta-blocker / prazosin / clonidine target |

The amygdala-centred fear circuit node by node, mapped to its transmitter systems and drug correlates; the accompanying figure draws the same loop as a map (input roads, amygdala hub, efferent targets, cortical and hippocampal brakes). Grounded in Kaplan & Sadock CTP 2024 and Tasman Psychiatry.

## 8.10 Definitions to fix the vocabulary

### Amygdala

Almond-shaped medial-temporal complex of subnuclei; the hub that evaluates threat and organises the fear response.

### Lateral nucleus (LA)

The amygdala's sensory in-door; thalamic and cortical input converge here and cues acquire threat value.

### Central nucleus (CE)

The amygdala's output nucleus; its efferents to hypothalamus, LC and PAG express the fear response.

### Low road

LeDoux's fast thalamo-amygdala route that bypasses the cortex; pre-conscious, crude, quick.

### High road

LeDoux's slower thalamo-cortico-amygdala route; accurate cortical appraisal of the threat.

### BNST

Bed nucleus of the stria terminalis; the "extended amygdala" that mediates sustained, context-driven anxiety rather than phasic fear.

### Extinction

Learned reduction of a conditioned fear once the cue is no longer dangerous; driven by the ventromedial PFC.

**INDIA**

India carries a large, largely untreated anxiety burden: the great majority of people with an anxiety, OCD or PTSD disorder never reach formal care, a treatment gap widened by stigma, scarcity of psychiatrists and the framing of anxiety as physical "ghabrahat" or "gas". Into that gap steps the country's **benzodiazepine over-prescription** problem: alprazolam and clonazepam are dispensed freely, often long-term and as a first move, by general practitioners and over the counter, which is exactly the wrong dial to turn on this circuit, the sedating brake that breeds dependence rather than the corrective serotonergic re-tuning. The neurobiology gives the corrective the Indian Psychiatric Society CPG endorses: an SSRI or SNRI is first-line for the anxiety disorders because it re-tunes the fear circuit, with the benzodiazepine reserved as a short bridge only. The relevant Indian brands are cheap and available, escitalopram (Nexito, Cipralex), sertraline (Serta, Zosert, Daxid), fluoxetine (Fludac) and paroxetine (Paxidep) for the serotonergic drugs, propranolol (Ciplar, Inderal) for the noradrenergic blunting of performance anxiety, and prazosin (Minipress, Prazopress) for PTSD nightmares; the correct answer treats the circuit, not merely the symptom.

| 2 | 3 | 3 |
|---|---|---|
| SENSORY ROADS TO THE AMYGDALA (LEDOUX'S FAST LOW ROAD, SLOW HIGH ROAD) | CENTRAL-NUCLEUS EFFERENT TARGETS TO NAME (HYPOTHALAMUS, LOCUS COERULEUS, PAG) | TRANSMITTER DIALS ON THE CIRCUIT (GABA BRAKE, SEROTONIN TUNER, NORADRENALINE AMPLIFIER) |

**HOLD THIS**

The fear circuit is an amygdala hub fed by LeDoux's fast subcortical low road and slow cortical high road, expressing fear through the central nucleus onto the hypothalamus, locus coeruleus and periaqueductal grey, and normally braked by the ventromedial prefrontal cortex and contextualised by the hippocampus; anxiety is that circuit over-reactive and under-regulated, tuned by three dials, the GABA brake (benzodiazepine target), the serotonergic tuner (SSRI target, slow and corrective) and the noradrenergic amplifier from the locus coeruleus (beta-blocker and prazosin target).

## 9 · Cognitive behaviour therapy for anxiety

**Cognitive behaviour therapy for anxiety** is the psychological treatment that the whole anxiety block leans on: across generalised anxiety disorder, panic, social anxiety and the phobias, every major guideline names **cbt for anxiety** as a first-line option of broadly similar efficacy to drug treatment. This note owns the disorder-specific CBT model, the cognitive model of anxiety, **cognitive restructuring**, the **behavioural experiment**, and the three classes of exposure, including **interoceptive exposure**, with the safety-behaviour idea running through all of them; the wider CBT technique (the general cognitive model, the wider exposure principle) is cross-referenced to the Psychotherapies, clinical assessment, MSE and nosology notes.




**Fig 14.5** Exposure therapy repeatedly approaches feared-but-safe cues while reducing avoidance and safety behaviours, allowing new inhibitory safety learning to compete with the older threat association.

**Why it matters, and how this note is framed.** Examiners reward the candidate who can do two things: state the *cognitive model* of anxiety (that it is the appraisal of a situation, not the situation itself, that drives the fear, and that avoidance and safety behaviours keep the belief alive by preventing disconfirmation), and name the *techniques* with their disorder-specific twist,

interoceptive exposure for the "fear of fear" in panic, dropping safety behaviours and shifting attention outward in social anxiety, exposure and response prevention for OCD (owned separately in the OCD notes). The single sharpest fact to hold is that CBT works by *disconfirming a catastrophic prediction* through direct experience, which is exactly what a behavioural experiment is engineered to do.

### 9.1 The cognitive model of anxiety: appraisal, not the event, drives the fear

**The basic assumption.** The cognitive model holds that people's beliefs and appraisals of events are at least as important as the events themselves in determining their emotional reactions; problematic anxiety is frequently the product of unrealistic, unhelpful beliefs about the world, the self and others (Kaplan and Sadock 2024). The classic sequence is *thoughts and beliefs lead to emotions and behaviours*, Aaron Beck's belief-disconfirmation model, so the therapeutic target is the faulty appraisal, not the feared object directly. In anxiety specifically, the appraisals share a family resemblance: probability overestimation (the feared outcome is far more likely than it is) and catastrophic thinking (its consequences would be unbearable), plus all-or-nothing thinking, overgeneralisation and selective attention to confirming evidence.

**How maintenance works.** The disorder is held in place by a vicious cycle: an ambiguous cue is appraised as threatening, which raises anxiety and arousal, which is itself read as further evidence of danger, and the person then *avoids* or deploys *safety behaviours* that abort the situation before the catastrophe can be shown not to happen. Because the feared outcome never gets a chance to be disconfirmed, the belief survives intact. CBT breaks this loop by getting the patient to stay in contact with the feared situation, or sensation, long enough to learn that the prediction was wrong.

**GOOD TO REMEMBER**

- **Cognitive behaviour therapy for anxiety:** the principle is that anxiety flows from a distorted *appraisal* (probability overestimation and catastrophic thinking) rather than the event itself, and is maintained by avoidance and safety behaviours that prevent the belief from being disconfirmed; the mechanism of change is disconfirmation of the catastrophic prediction through cognitive restructuring and exposure; it is first-line, of broadly similar efficacy to pharmacotherapy across the anxiety disorders.

### 9.2 The shape of a course: assessment, formulation, psychoeducation, homework

**How a course of CBT is built.** CBT begins with a structured assessment of the presenting complaint using validated measures (and disorder-relevant constructs such as worry-related metacognitions in GAD or anxiety sensitivity in panic), repeated over treatment to track outcome. The next step is psychoeducation: the patient is given a shared cognitive-behavioural formulation of their specific diagnosis and a treatment rationale, then therapist and patient collaboratively set targets, for example building the exposure hierarchy or eliciting the core fear (Kaplan and Sadock 2024). CBT is time-limited, structured and collaborative, and leans heavily on homework and self-monitoring between sessions so that skills generalise beyond the consulting room.

**How long it takes.** The course is short by psychotherapy standards. For panic disorder the optimal dose is **7 to 14 hours** of treatment, delivered weekly, typically over about four months (NICE). For GAD, a high-intensity individual CBT course is **12 to 15 weekly sessions** of one hour (NICE). This brevity is part of why guidelines can place CBT co-equal with drugs and why low-intensity and internet-delivered versions are pushed hard to widen access.

GOOD TO REMEMBER

- **Structure of CBT for anxiety:** assessment then shared cognitive-behavioural formulation and psychoeducation, collaborative goal-setting, and between-session homework and self-monitoring; it is brief and time-limited (panic 7 to 14 hours over about four months; GAD 12 to 15 weekly one-hour sessions per NICE), which is why it sits co-equal with pharmacotherapy as first-line.

### 9.3 Cognitive restructuring: Socratic dialogue, thought records, the downward arrow

**What it is.** Cognitive restructuring is the set of procedures for identifying and modifying the unhelpful appraisals. The three traditional techniques are (1) Socratic dialogue, a guided series of questions that helps the patient surface and challenge a fear-related belief and uncover disconfirming evidence; (2) the downward-arrow technique, repeatedly asking "and what would that mean?" to reach the deeper core belief; and (3) the thought record, in which the patient logs automatic thoughts, lists the evidence for and against, spots the cognitive error, and generates a more realistic, balanced alternative (Kaplan and Sadock 2024). The therapist does not simply reassure or argue; the patient is walked to the new conclusion so that they own it.

**What it targets in anxiety.** The restructuring is aimed squarely at the two anxiety appraisals, overestimating how likely the bad outcome is and catastrophising how bad it would be. In panic disorder that means challenging the catastrophic misinterpretation of a bodily sensation ("this pounding heart means I am having a heart attack"); in social anxiety it means challenging the prediction of humiliation and the distorted self-image seen "from the outside". Restructuring alone can reduce anxiety, but its power is greatest when it sets up a prediction that exposure can then test.

RULE **Restructure, then test.** Cognitive restructuring makes the catastrophic belief explicit and generates a specific prediction; exposure and behavioural experiments then supply the disconfirming experience, and it is the direct experience, not the argument, that shifts the belief.

GOOD TO REMEMBER

- **Cognitive restructuring:** identifying and modifying anxious appraisals (probability overestimation, catastrophising) via Socratic dialogue, thought records and the downward-arrow technique; the signature caution is that it is collaborative and guided, not reassurance or debate, and it works best when it produces a testable prediction that exposure can then disconfirm.

### 9.4 The behavioural experiment: testing a prediction against reality

**The single most important technique.** A behavioural experiment is the point where cognitive and behavioural methods fuse. Its goal is to test a belief about the nature and probability of a feared consequence under conditions that optimise exposure to, and encoding of, disconfirming

information (Kaplan and Sadock 2024). Before the experiment the patient makes a *detailed, specific prediction* of what they believe will happen, then carries out the relevant action and observes the actual result. Each time the catastrophe fails to occur, the patient is confronted with evidence against the belief. The worked panic example is canonical: a patient who fears they will "go crazy" if they feel dizzy is asked to spin in a chair, slowly then faster, and discovers that intense dizziness comes and goes without madness.

**Why it beats reassurance.** Homework assignments and behavioural experiments let the patient practise new behaviours and test the therapist's alternative explanations for themselves; even experiments that "fail" reveal more about the condition (Shorter Oxford Textbook). Because the learning is experiential and self-generated, it sticks in a way that being told "your heart is fine" never does, which is why the behavioural experiment is the engine of durable change in CBT for anxiety.

| STEP | WHAT HAPPENS |
|---|---|
| **Identify the belief** | Elicit the specific catastrophic prediction (e.g. "if I feel dizzy I will collapse") |
| **Make it testable** | **Operationalise a concrete, falsifiable prediction with the patient** |
| **Design the experiment** | Choose an action that will expose the prediction to disconfirming evidence, drop safety behaviours |
| **Run it** | Carry out the action; the patient stays in contact and observes what actually happens |
| **Review the evidence** | **Compare prediction with outcome; update the belief from direct experience** |

The anatomy of a behavioural experiment; the load-bearing steps are making the belief testable and then reviewing prediction against reality (Kaplan and Sadock 2024).

**GOOD TO REMEMBER**

- **Behavioural experiment:** a planned test in which the patient makes a specific prediction about a feared consequence, carries out an action (with safety behaviours dropped), and compares prediction with outcome; the principle is experiential disconfirmation of the catastrophic belief, and the signature caution is that the prediction must be concrete and falsifiable, and safety behaviours removed, or the experiment cannot disconfirm anything.

### 9.5 Exposure: the three classes, done without safety behaviours

**What exposure is.** Exposure therapy means intentionally confronting a feared but not actually dangerous object, situation, thought, memory or physical sensation, in order to reduce the fear and avoidance attached to it. Contemporary exposure divides into three classes: *in vivo*, *imaginal* and interoceptive, all conducted while encouraging the patient to *prevent safety behaviours*, the strategies that avert the feared outcome but, in doing so, block the new learning (Kaplan and Sadock 2024). Older systematic desensitisation paired graded imaginal or in vivo exposure with relaxation; that relaxation is now regarded as unnecessary and possibly counterproductive, and prolonged in vivo exposure has become the standard.

**The three classes.** *In vivo* exposure directly confronts feared objects, activities and situations, usually graded up a collaboratively agreed hierarchy (the snake photographed, then caged, then handled). *Imaginal* exposure has the patient vividly picture the feared scenario, thought or memory (its main use is confronting feared thoughts and traumatic memories, and it underpins imagery rescripting in PTSD). **Interoceptive exposure**, the most recent, deliberately induces the feared bodily sensations under control, hyperventilating for breathlessness and tingling, spinning for dizziness, straw-breathing for air-hunger, to teach that the sensations are not dangerous; it is the signature technique for panic disorder's "fear of fear".

| CLASS | WHAT IS CONFRONTED | SIGNATURE USE |
|---|---|---|
| **In vivo** | Real feared objects, activities and situations, graded up a hierarchy | **Specific phobia, agoraphobic avoidance, social situations** |
| **Imaginal** | Feared thoughts, images and memories, vividly pictured | Traumatic memories (PTSD), feared consequences; imagery rescripting |
| **Interoceptive** | **Feared bodily sensations, induced on purpose (spin, hyperventilate, straw-breathe)** | **Panic disorder: the "fear of fear" / anxiety sensitivity** |

The three classes of exposure; all are run while dropping safety behaviours, and interoceptive exposure is the panic-specific technique for anxiety sensitivity (Kaplan and Sadock 2024).

TRAP **Safety behaviours and reassurance sabotage exposure.** If the patient keeps a hidden crutch (clutching a water bottle, sitting near the exit, silent reassurance-seeking) or the exposure is too brief to let anxiety fall, the catastrophic belief is never disconfirmed, so the exposure looks like it "failed" when the design was at fault.

GOOD TO REMEMBER

- **Exposure (in vivo, imaginal, interoceptive):** graded confrontation of the feared stimulus without safety behaviours to allow new learning; in vivo for real situations and phobias, imaginal for feared thoughts and traumatic memories, interoceptive for the feared bodily sensations of panic; the signature caution is that safety behaviours, avoidance and reassurance-seeking must be dropped, and prolonged real (in vivo) exposure now replaces relaxation-paired systematic desensitisation.

### 9.6 Interoceptive exposure and the CBT model of panic

**The panic cycle.** The cognitive-behavioural model of panic (the Clark model) frames a panic attack as a vicious cycle: the person is hypervigilant for a bodily sensation, misinterprets it catastrophically ("this racing heart means a heart attack; this unreality means I am going mad"), which raises arousal and intensifies the sensation, spiralling into panic; agoraphobic avoidance then grows as an attempt to escape the situations where attacks might strike (Kaplan and Sadock 2024). The therapeutic implication is direct: break the catastrophic misinterpretation and the loop cannot close.

**Where interoceptive exposure fits.** CBT for panic has four components: psychoeducation about the panic cycle, cognitive restructuring of the catastrophic misinterpretation, **interoceptive**

**exposure** to the feared sensations to reduce the "fear of fear", and in vivo exposure to any avoided (agoraphobic) situations. Dismantling studies point to cognitive restructuring combined with in vivo or interoceptive exposure as the active ingredients, and adding interoceptive exposure is particularly helpful; in one meta-analysis the largest effect size was for cognitive therapy plus interoceptive exposure (**d = 0.88**), ahead of CBT without it and of medication alone. Between **41 and 87 per cent** of patients are panic-free after CBT, and CBT gains hold better than medication after treatment stops.

**WORKED EXAMPLE**

A 28-year-old with panic disorder is terrified that the dizziness and derealisation during an attack mean she is about to faint or lose her mind, so she sits down, grips a chair and sips water whenever they start (safety behaviours). Restructuring surfaces the prediction "if I let the dizziness build I will faint". The interoceptive exposure is to hyperventilate for a minute, then spin, deliberately bringing on the dizziness and unreality, without sitting, gripping or sipping. Over repeated trials she learns the sensations crest and pass without fainting or madness; the "fear of fear" falls, and in vivo exposure then returns her to the supermarket and the bus she had been avoiding.

**GOOD TO REMEMBER**

- **Interoceptive exposure in panic:** deliberate induction of feared bodily sensations (hyperventilation, spinning, straw-breathing) to break the catastrophic misinterpretation that drives the panic cycle and reduce the "fear of fear"; it is the panic-specific exposure class, one of the active ingredients of panic CBT, and the signature caution is that it fails if the patient keeps safety behaviours or the induced sensations are cut short.

### 9.7 Social anxiety CBT: dropping safety behaviours, shifting attention outward

**The disorder-specific twist.** In social anxiety disorder the Clark and Wells model adds two signature moves to the standard restructuring-plus-exposure package: a stronger emphasis on *identifying and eliminating safety behaviours* (over-rehearsing sentences, avoiding eye contact, gripping a glass to hide a tremor) and on *shifting attention outward* from the distorted, self-focused image the patient believes others can see (Kaplan and Sadock 2024). Exposures are integrated with cognitive challenge: the patient makes an explicit prediction, drops the safety behaviours, focuses attention on the other person rather than the self, and reviews the outcome afterwards, often with video feedback that contradicts the "I looked ridiculous" belief.

**Why it works.** Network meta-analysis puts individual CBT ahead of group CBT and of psychodynamic, supportive and mindfulness therapies for social anxiety, and the Clark and Wells version shows large effects against fluoxetine, exposure alone and group CBT. This is why NICE makes disorder-specific *individual* CBT the first-line treatment for social anxiety disorder, developed further in the Social anxiety disorder note.

**PEARL**

The two Clark-and-Wells refinements that lift social-anxiety CBT above generic exposure are *dropping safety behaviours* and *redirecting attention outward*. Self-focused attention manufactures the very "distorted self-image" the patient dreads others see; video feedback, showing the patient they did not visibly shake or blush as badly as they believed, is a behavioural experiment that disconfirms it directly.

**GOOD TO REMEMBER**

- **Social anxiety CBT (Clark and Wells):** cognitive restructuring of the fear of negative evaluation plus graded exposure, with two signature additions, systematically dropping safety behaviours and shifting attention outward from the self-focused image, tested with video-feedback experiments; individual CBT is first-line and outperforms group CBT for social anxiety disorder (NICE).

### 9.8 Evidence and place in the anxiety guidelines

**What the guidelines say.** The convergence is the exam point. NICE, BAP, CANMAT and the Indian Psychiatric Society all place **cbt for anxiety** as a first-line psychological treatment across generalised anxiety disorder, panic disorder and social anxiety disorder, of broadly similar efficacy to pharmacotherapy, with combination (CBT plus an SSRI or SNRI) reserved for partial or non-response. NICE makes CBT (or applied relaxation) one of two equivalent first-choice high-intensity options in GAD, first-line for panic (7 to 14 hours), and the first-line individual treatment for social anxiety disorder; BAP and CANMAT agree. For OCD the psychological arm is *exposure and response prevention*, taught in the OCD management notes; for PTSD it is trauma-focused CBT, taught in the PTSD notes.

**The one durability advantage.** A recurring finding is that CBT gains are more durable than medication gains once treatment stops (in panic, CBT alone held better than CBT plus imipramine after discontinuation), and cognitive therapy with exposure may reduce relapse better than drug treatment alone. That relapse-protection edge, plus lower dropout than medication, is why guidelines are keen to expand access through group, guided self-help and internet-delivered CBT.

| DISORDER | CBT STATUS | DISORDER-SPECIFIC CBT INGREDIENT |
|---|---|---|
| **Generalised anxiety disorder** | **First-line (co-equal with drugs); 12 to 15 sessions** | Worry-focused restructuring; no standard in vivo target |
| **Panic disorder** | **First-line; 7 to 14 hours** | **Interoceptive exposure for the "fear of fear"** |
| **Social anxiety disorder** | First-line individual CBT (over group) | Drop safety behaviours, shift attention outward (Clark and Wells) |
| **Specific phobia / agoraphobia** | Exposure is treatment of choice | Graded in vivo exposure up a hierarchy |
| **OCD** | First-line psychotherapy (see OCD notes) | Exposure and response prevention |

CBT across the anxiety block; the through-line is first-line status with a disorder-specific technique, and the durability edge over medication after stopping is the recurring evidence point.

**INDIA**

India has a wide anxiety, OCD and PTSD treatment gap, most sufferers never reach formal care, and where care is reached it too often means long-term benzodiazepine prescribing, alprazolam and clonazepam dispensed for "nervousness", which runs directly against the Indian Psychiatric Society CPGs. CBT is the evidence-based alternative that the gap most needs, yet trained CBT therapists are scarce outside metros. This is exactly where low-intensity and internet-delivered CBT, guided self-help and task-shifting to non-specialists matter: they are the realistic route to scaling a first-line psychological treatment in a low-resource setting, and they let the SSRI-plus-CBT model displace the benzodiazepine habit rather than reinforce it.

| 3 | 7 to 14 hours | 0.88 | 10 |
|---|---|---|---|
| CLASSES OF EXPOSURE (IN VIVO, IMAGINAL, INTEROCEPTIVE) | CBT DOSE FOR PANIC DISORDER (NICE) | EFFECT SIZE, COGNITIVE THERAPY PLUS INTEROCEPTIVE EXPOSURE IN PANIC | GAD-7 CLINICAL CUT-OFF (SCREEN THE DISORDER CBT TREATS) |

**HOLD THIS**

CBT for anxiety works by disconfirming a catastrophic prediction through direct experience: cognitive restructuring makes the belief testable, the behavioural experiment and the three exposure classes (in vivo, imaginal, interoceptive, all with safety behaviours dropped) supply the disconfirming evidence, and it is first-line and co-equal with drugs across the anxiety disorders, with interoceptive exposure the signature technique for panic.

## 10 · Anxiety: differential diagnosis and medical mimics

Once the anxiety disorders have been learned one at a time, the examiner turns the question round: a patient walks in with palpitations, tremor, sweating and a racing sense of dread, and you must say *which* anxiety disorder this is, and first of all whether it is an anxiety disorder at all. This is the discrimination step. The **differential diagnosis of anxiety** has two limbs that must both be secured: separating the anxiety disorders from one another by the *focus of the fear*, and

separating a primary anxiety disorder from an organic cause, the **medical mimics**. The accompanying figure maps the anxiety disorders by the focus of the fear, and the figure alongside sorts the medical mimics by the feature that gives each away; this note is the text that unpacks both.

**Why it matters, and the trap that fails candidates.** The single discriminator that separates each anxiety disorder is a one-line fact the examiner expects you to produce instantly, and the classic error, the trap that turns a pass into a fail, is to reach for a psychiatric label while missing an organic mimic sitting in plain sight (**hyperthyroidism**, a **phaeochromocytoma**, an arrhythmia, hypoglycaemia, or a caffeine habit). The neurobiology of the fear circuit that these mimics hijack is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; the anxiolytic and withdrawal pharmacology that produces the substance-related pictures below lives in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; what this note owns is the discrimination itself.

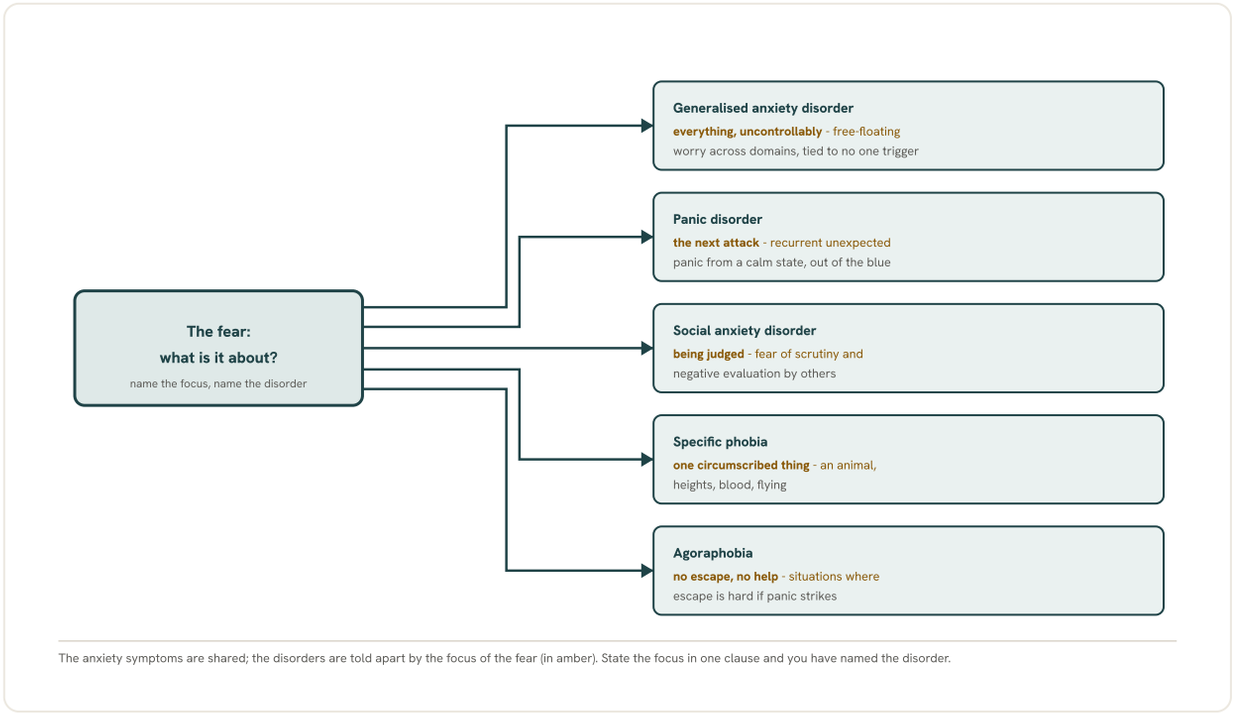



**Fig 14.6** Discriminating the anxiety disorders by the focus of the fear. The somatic symptoms of anxiety are shared across all of these disorders, so they cannot separate them; what separates them is what the fear is about. Generalised anxiety fears everything and nothing in particular; panic fears the next attack; social anxiety fears being judged; specific phobia fears one circumscribed object or situation; agoraphobia fears being trapped where escape would be difficult or help unavailable. Name the focus and you have named the disorder.

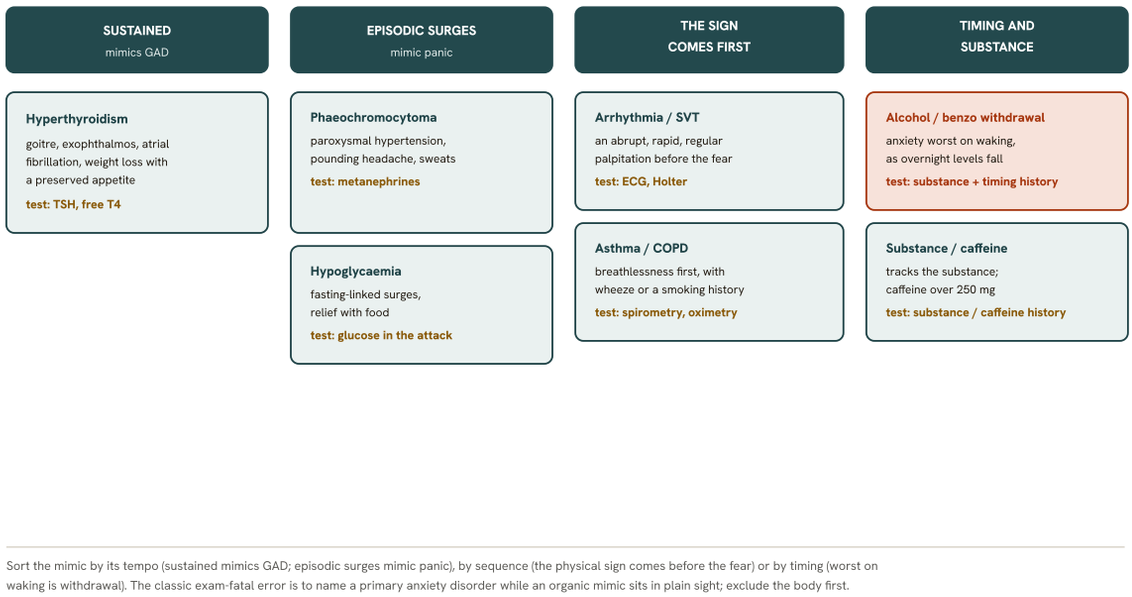



**Fig 14.7** The medical mimics of anxiety, sorted by the feature that gives each away. Rather than a flat list, the mimics fall into four groups by their discriminating principle. A sustained driver (thyrotoxicosis) reproduces the steady picture of generalised anxiety; episodic paroxysmal surges (a phaeochromocytoma, hypoglycaemia) reproduce panic; where the physical sign comes first, an arrhythmia (a palpitation preceding the fear) or asthma and COPD (breathlessness first) is the answer; and anxiety that is worst on waking is alcohol or benzodiazepine withdrawal, while anxiety that tracks a substance or a heavy caffeine load is intoxication. Excluding the body, and choosing the test by the tempo, comes before any anxiety-disorder label.

## 10.1 The organising principle: one discriminator per disorder, the body ruled out first

**Two questions, in order.** Faced with an anxious patient, ask first "is this organic?" and only then "which anxiety disorder is it?". The order is deliberate: an SSRI started for what is actually thyrotoxicosis or a phaeochromocytoma is not merely useless but leaves a dangerous lesion untreated. The organic screen (thyroid function, a substance and caffeine history, attention to withdrawal timing, and where the story is episodic and physical, catecholamines and an ECG) comes before the psychiatric formulation, not after it.

**Then the focus of the fear.** Once the body is cleared, the anxiety disorders are told apart not by the *symptoms* of anxiety (which are shared) but by *what the fear is about*. GAD fears everything and nothing in particular; panic fears the next attack; social anxiety fears being judged; specific phobia fears one circumscribed thing; agoraphobia fears being trapped where escape is hard. Name the focus and you have named the disorder.

> **RULE** **Exclude the body before you name the disorder.** Thyroid function, a caffeine and substance history, and (where symptoms are episodic and physical) an ECG and catecholamines precede any anxiety-disorder label; the focus of the fear then names the disorder.

## 10.2 Discriminating the anxiety disorders: the focus of the fear

**The one-liner for each.** The examiner wants the discriminating focus stated in a single clause. GAD is free-floating anxiety, chronic uncontrollable worry across many domains, not tied to any one trigger. Panic disorder is defined by *recurrent unexpected panic attacks* that come out of the

blue from a calm state, with the fear being of the attacks themselves (anticipatory anxiety about the next surge). Social anxiety disorder is a *fear of scrutiny and negative evaluation* in social or performance situations. Specific phobia is fear of a *circumscribed object or situation* (an animal, heights, blood, flying). Agoraphobia is fear of situations *where escape would be difficult or help unavailable* should a panic-like or incapacitating event strike, the fear is about escape and rescue, not the place itself.

| DISORDER | THE ONE DISCRIMINATOR (FOCUS OF THE FEAR) | COURSE/CUE |
|---|---|---|
| **Generalised anxiety disorder** | **Free-floating, uncontrollable worry across many domains, not bound to any trigger** | Chronic, persistent |
| **Panic disorder** | **Fear of the attacks themselves; recurrent unexpected panic from a calm state** | Episodic, out of the blue |
| **Social anxiety disorder** | **Fear of scrutiny and negative evaluation by others** | Cued by social/performance settings |
| **Specific phobia** | Fear of one circumscribed object or situation | Cued by the single phobic stimulus |
| **Agoraphobia** | **Fear of situations where escape is difficult or help unavailable** | Cued by crowds, transport, open/enclosed spaces |

The anxiety-disorders differentiation grid: each disorder is separated by the focus of the fear, not by the anxiety symptoms, which are shared (ICD-11 CDDR; DSM-5-TR 2022).

**GOOD TO REMEMBER**

- **Differential of the anxiety disorders:** the discriminator is the focus of the fear, GAD (everything, free-floating), panic (the next attack), social anxiety (negative evaluation), specific phobia (one circumscribed object), agoraphobia (being unable to escape or get help); state the focus in one clause and you have named the disorder.

### 10.3 Organic anxiety: the category and why episodic-versus-sustained matters

**The concept.** Organic anxiety disorder (organic anxiety disorder in ICD terms) is anxiety arising secondarily from a medical disorder, the fear-circuit output is real but the driver is bodily. The teaching pivot, drawn straight from the Shorter Oxford Textbook, is the *tempo* of the mimic. Sustained, low-grade organic drivers (thyrotoxicosis) reproduce the GAD picture; *episodic, paroxysmal* drivers (phaeochromocytoma, hypoglycaemia, paroxysmal arrhythmia) reproduce panic disorder or a phobic disorder, because they come in discrete surges (Shorter Oxford Textbook of Psychiatry). Matching the tempo of the mimic to the tempo of the psychiatric picture is how you decide which physical test to send.

**The pointer to organicity.** Features that should push you towards a physical cause rather than a primary anxiety disorder: a first presentation of anxiety in an older patient, prominent and disproportionate physical signs, anxiety without the cognitive content of worry or dread, a fluctuating episodic course with autonomic storms, and any hard sign on examination (goitre, exophthalmos, atrial fibrillation, sustained hypertension).

### 10.4 Hyperthyroidism: the sustained mimic of GAD

**The picture.** In hyperthyroidism (thyrotoxicosis) the patient is irritable and restless, with tremor and tachycardia, exactly the somatic tail of GAD, and it is the commonest and most examinable organic mimic (Shorter Oxford Textbook of Psychiatry). Because the thyroid drive is continuous, the resulting anxiety is *sustained*, so thyrotoxicosis is the classic mimic of generalised anxiety disorder rather than of panic.

**The pointing feature, the test, the discriminator.** The pointing features are the hard thyroid signs, an enlarged thyroid (goitre), atrial fibrillation, exophthalmos, heat intolerance and weight loss despite a good appetite. The screening test is thyroid function (a suppressed TSH with raised free T4). The discriminator from a primary anxiety disorder is that thyrotoxic patients lose weight while eating well and show the eye and gland signs, and their anxiety resolves when the thyroid is treated (Shorter Oxford Textbook of Psychiatry).

**GOOD TO REMEMBER**

- **Hyperthyroidism (thyrotoxicosis):** the sustained organic mimic of GAD (irritability, restlessness, tremor, tachycardia); pointing features are goitre, exophthalmos, atrial fibrillation and weight loss with preserved appetite; the screening test is thyroid function (suppressed TSH, raised free T4); the discriminator is the physical thyroid signs and resolution on treating the thyroid.

### 10.5 Phaeochromocytoma: the paroxysmal mimic of panic

**The picture.** A phaeochromocytoma is a catecholamine-secreting tumour that releases adrenaline and noradrenaline in bursts, producing paroxysmal episodes of palpitations, pounding headache, sweating and a surge of dread, indistinguishable at the bedside from a panic attack. Because the secretion is *episodic*, the phaeochromocytoma mimics panic disorder or a phobic disorder rather than GAD (Shorter Oxford Textbook of Psychiatry; New Oxford Textbook of Psychiatry).

**The pointing feature, the test, the discriminator.** The pointing feature is *paroxysmal hypertension*, blood pressure that spikes with each attack, together with the headache-palpitations-sweating triad. The screening tests are plasma or 24-hour urinary metanephrines and catecholamines. The discriminator from panic disorder is the objectively recorded hypertensive surge during an attack and the presence of the pounding headache, panic disorder does not raise the blood pressure to those levels or produce that headache. The classic exam pairing is that phaeochromocytoma and hypoglycaemia both cause *episodic* symptoms and therefore mimic panic or phobia (Shorter Oxford Textbook of Psychiatry).

**GOOD TO REMEMBER**

- **Phaeochromocytoma:** a catecholamine-secreting tumour producing paroxysmal palpitations, pounding headache and sweating that mimic panic; the pointing feature is paroxysmal hypertension; the screening test is plasma/urinary metanephrines and catecholamines; the discriminator is the recorded hypertensive surge and the headache during attacks.

### 10.6 Arrhythmia and supraventricular tachycardia, and hypoglycaemia

**Cardiac: the palpitation that came first.** A paroxysmal arrhythmia, above all a supraventricular tachycardia (SVT), produces sudden palpitations, breathlessness and a sense of doom that read as panic; paroxysmal tachycardia is on the standard organic-mimic list (New Oxford Textbook of Psychiatry). The pointing feature is a very rapid, regular, abruptly-starting-and-stopping palpitation that *precedes* the fear rather than following it, and, in panic disorder, mitral valve prolapse is a recognised association. The screening test is an ECG, and ideally a rhythm strip or ambulatory (Holter) monitor capturing an episode. The discriminator from panic is the temporal order and the rate: in SVT the tachycardia comes first and is faster and more regular than the sinus tachycardia of panic, which follows the psychological alarm.

**Hypoglycaemia: the episodic autonomic surge.** Recurrent hypoglycaemia produces episodic tremor, sweating, palpitations and anxiety and, like phaeochromocytoma, mimics panic or a phobic disorder because it is episodic (Shorter Oxford Textbook of Psychiatry; New Oxford Textbook of Psychiatry). The pointing feature is the relationship to fasting and the relief with food; the screening test is a paired capillary/venous glucose during an attack (Whipple's triad); the discriminator is the low glucose recorded during symptoms and their resolution with carbohydrate.

**GOOD TO REMEMBER**

- **SVT/arrhythmia:** abrupt rapid regular palpitations that come *before* the fear; test with ECG/Holter; discriminator is the tachycardia preceding the anxiety and being faster and more regular than panic's sinus tachycardia.
- **Hypoglycaemia:** episodic tremor, sweating and anxiety mimicking panic; test with glucose during symptoms; discriminator is the low glucose recorded in the attack and relief with food.

### 10.7 Asthma and COPD: the breathless mimic

**The picture.** Bronchospasm in asthma and hypoxia or hypercapnia in COPD produce breathlessness, chest tightness and a frightened, air-hungry arousal that is easily mislabelled panic, and the two genuinely co-occur and feed each other (breathlessness triggers panic, panic worsens breathlessness). The pointing feature is that the *breathlessness comes first* and is accompanied by wheeze, cough, a documented trigger (exertion, allergen, cold air) or a smoking history, rather than arising from a calm state. The screening test is peak expiratory flow or spirometry (and pulse oximetry in an acute episode). The discriminator from panic disorder is the demonstrable airflow obstruction and hypoxia, and the response to a bronchodilator, panic produces hyperventilation with a normal or low CO2, not true obstruction.

**GOOD TO REMEMBER**

- **Asthma/COPD:** breathlessness and chest tightness mimicking panic, with the dyspnoea coming first plus wheeze/cough/trigger or smoking history; test with peak flow/spirometry and oximetry; discriminator is demonstrable airflow obstruction and hypoxia responding to a bronchodilator, unlike the normocapnic hyperventilation of panic.

### 10.8 Substance and caffeine intoxication

**Stimulant and substance-induced anxiety.** Substance-induced anxiety is anxiety arising during intoxication with a stimulant, cocaine, amphetamine or other sympathomimetic, or from other agents, and it must be excluded before a primary diagnosis is made. The pointing feature is the temporal link to the substance and any intoxication signs (dilated pupils, tachycardia, hypertension, agitation); the screening test is a substance history and, where available, a urine drug screen; the discriminator is that the anxiety tracks the substance and clears as the drug is eliminated.

**Caffeine, the ubiquitous and forgotten mimic.** Caffeine anxiety (caffeinism, caffeine intoxication) is the mimic most often missed because it is legal and everyday. DSM defines caffeine intoxication as recent consumption usually in excess of **250 mg** of caffeine (about 2 to 3 cups of brewed coffee) with **five or more** of restlessness, nervousness, excitement, insomnia, flushed face, diuresis, gastrointestinal disturbance, muscle twitching, rambling thought, tachycardia, and psychomotor agitation (DSM-5-TR 2022). The pointing feature is a high daily caffeine load; the screening test is a careful dietary caffeine history (brewed coffee about 100 mg/cup, instant about 60 mg, tea about 45 mg, cola 25 to 50 mg per can); the discriminator is that the anxiety and insomnia settle on cutting caffeine down.

**COMMON TRAP**

The classic error is to diagnose a primary anxiety disorder and miss an organic mimic. Before labelling GAD or panic, exclude hyperthyroidism (goitre, exophthalmos, weight loss), a phaeochromocytoma and hypoglycaemia (episodic surges, paroxysmal hypertension in the phaeochromocytoma), an arrhythmia or SVT (palpitation preceding the fear), asthma or COPD (breathlessness first), and above all a caffeine habit or stimulant use, each reproduces the anxiety picture, and each is treated at its source, not with an SSRI.

**GOOD TO REMEMBER**

- **Substance-induced anxiety:** anxiety during stimulant/sympathomimetic intoxication; pointing feature is the temporal link plus intoxication signs; screen with substance history and urine drug screen; discriminator is clearance as the drug is eliminated.
- **Caffeine intoxication:** more than 250 mg caffeine plus 5 or more of restlessness, nervousness, insomnia, flushed face, diuresis, GI upset, twitching, tachycardia; screen with a caffeine dietary history; discriminator is resolution on reducing caffeine.

### 10.9 Alcohol and benzodiazepine withdrawal: the morning mimic

**The picture.** Withdrawal from alcohol or from a benzodiazepine reproduces GAD-like anxiety with tremor, sweating, tachycardia and insomnia, and it is a leading cause of "treatment-resistant anxiety" in a dependent patient who is really in daily micro-withdrawal (Shorter Oxford Textbook of Psychiatry). The pointing feature that Gelder emphasises is *timing*: anxiety that is worst on waking in the morning, when overnight alcohol and short-half-life benzodiazepine levels have fallen, points to withdrawal (and, separately, morning-worst anxiety also raises depressive disorder). The screening test is a detailed alcohol and sedative history with quantities

and timing; the discriminator from a primary anxiety disorder is the diurnal, dose-linked pattern of the anxiety and, on withdrawal from certain drug classes, hard signs such as pupillary changes.

**GOOD TO REMEMBER**

- **Alcohol/benzodiazepine withdrawal:** a GAD-like tremulous, sweaty, tachycardic anxiety that is worst on waking; the pointing feature is the morning timing as overnight levels fall; screen with a detailed substance-and-timing history; the discriminator is the diurnal, dose-linked pattern rather than the persistent free-floating worry of primary anxiety.

| MIMIC | POINTING FEATURE | SCREENING TEST | DISCRIMINATOR FROM PRIMARY ANXIETY |
|---|---|---|---|
| **Hyperthyroidism** | **Goitre, exophthalmos, AF, weight loss with appetite** | Thyroid function (TSH, free T4) | Physical thyroid signs; sustained (mimics GAD); resolves on treating thyroid |
| **Phaeochromocytoma** | **Paroxysmal hypertension, pounding headache** | Plasma/urinary metanephrines, catecholamines | Recorded BP surge in attack; episodic (mimics panic) |
| **Arrhythmia / SVT** | Abrupt rapid regular palpitation before the fear | ECG, Holter/rhythm strip | Tachycardia precedes anxiety; faster, more regular than panic |
| **Hypoglycaemia** | Fasting-related surges, relief with food | Glucose during symptoms (Whipple's triad) | Low glucose recorded in attack; episodic (mimics panic) |
| **Asthma / COPD** | Breathlessness first, wheeze, smoking history | Peak flow/spirometry, oximetry | Airflow obstruction and hypoxia; responds to bronchodilator |
| **Substance / caffeine intoxication** | High stimulant or caffeine load, intoxication signs | Substance history, urine drug screen, caffeine history | Anxiety tracks the substance; clears on cessation |
| **Alcohol / benzodiazepine withdrawal** | **Anxiety worst on waking, dose-linked** | Detailed substance and timing history | Diurnal, dose-linked pattern; withdrawal signs |

The anxiety-versus-medical-mimic grid: pointing feature, screening test and the discriminator from a primary anxiety disorder for each organic mimic (Shorter Oxford Textbook of Psychiatry; New Oxford Textbook of Psychiatry).

| | | |
|---|---|---|
| **250 mg** | **5** | **100 mg** |
| CAFFEINE INTOXICATION THRESHOLD (PLUS 5 OR MORE SYMPTOMS) | DSM SYMPTOMS REQUIRED FOR CAFFEINE INTOXICATION | CAFFEINE PER CUP OF BREWED COFFEE (INSTANT ABOUT 60) |

**TRAP** **Missing an organic mimic is the exam-fatal error.** Episodic autonomic storms with paroxysmal hypertension are a phaeochromocytoma until proven otherwise; sustained tremor with weight loss is thyrotoxicosis; anxiety worst on waking is alcohol or benzodiazepine withdrawal, not "resistant GAD".

### 10.10 The India lens: the treatment gap and the benzodiazepine problem behind the differential

**Two genuine Indian realities.** First, the anxiety, OCD and PTSD treatment gap is wide, anxiety is under-recognised, heavily somatised (patients present with palpitations, tremor and "gas" rather than "worry"), and under-treated, so the organic mimics are precisely the physical complaints that get an anxious patient sent round the physicians for years without either an anxiety diagnosis or a thyroid test. Second, the entrenched over-prescription of benzodiazepines (typically alprazolam) means many "anxious" Indian patients are in chronic benzodiazepine dependence, so *benzodiazepine withdrawal* is itself a common, iatrogenic mimic of anxiety in Indian practice. The Indian Psychiatric Society CPGs exist to correct both, rule out the organic causes, treat the primary disorder with an SSRI or SNRI first-line, and reserve the benzodiazepine as a short, time-limited bridge rather than the standing prescription (the Indian Psychiatric Society 2020).

**INDIA**

The highest-yield Indian point on this discrimination topic is that the benzodiazepine over-prescription problem creates its own mimic: a large number of Indian patients labelled "anxiety" are actually in daily alprazolam micro-withdrawal, which reads as morning-worst anxiety. The correct Indian workup mirrors the global one, exclude hyperthyroidism (a cheap TSH), a phaeochromocytoma and hypoglycaemia where episodic, and a caffeine or substance load, then treat the primary disorder with an SSRI or SNRI (escitalopram Nexito or Rexipra, sertraline) rather than adding yet another benzodiazepine strip.

**RULE** **Tempo tells you which mimic.** Sustained anxiety with weight loss and tremor is thyrotoxicosis (mimics GAD); episodic autonomic surges are phaeochromocytoma or hypoglycaemia (mimic panic); a palpitation preceding the fear is an arrhythmia; morning-worst anxiety is withdrawal.

**HOLD THIS**

Before naming any anxiety disorder, exclude the organic mimics, thyrotoxicosis (sustained, mimics GAD), phaeochromocytoma and hypoglycaemia (episodic, mimic panic), arrhythmia/SVT (palpitation first), asthma/COPD (breathlessness first), caffeine and substance intoxication, and alcohol/benzodiazepine withdrawal (worst on waking); then separate the anxiety disorders by the focus of the fear, GAD (everything), panic (the next attack), social anxiety (negative evaluation), specific phobia (one object), agoraphobia (no escape).

## Obsessive-compulsive and related disorders

### 11 · OCD: clinical features, dimensions and insight

Where anxiety disorders are about a feared object or a feared situation, **obsessive-compulsive disorder** is about a feared *thought* and the ritual built to undo it. **Obsessive-compulsive disorder** (**ocd**) is defined by **obsessions** (intrusive, unwanted, ego-dystonic thoughts, images or urges) and **compulsions** (repetitive behaviours or mental acts performed to neutralise the obsession or its dreaded consequence). It is the anchor diagnosis of the obsessive-compulsive and

related disorders grouping, which both DSM-5-TR and ICD-11 lifted out of the anxiety disorders into a spectrum of its own, and which the accompanying figure maps.

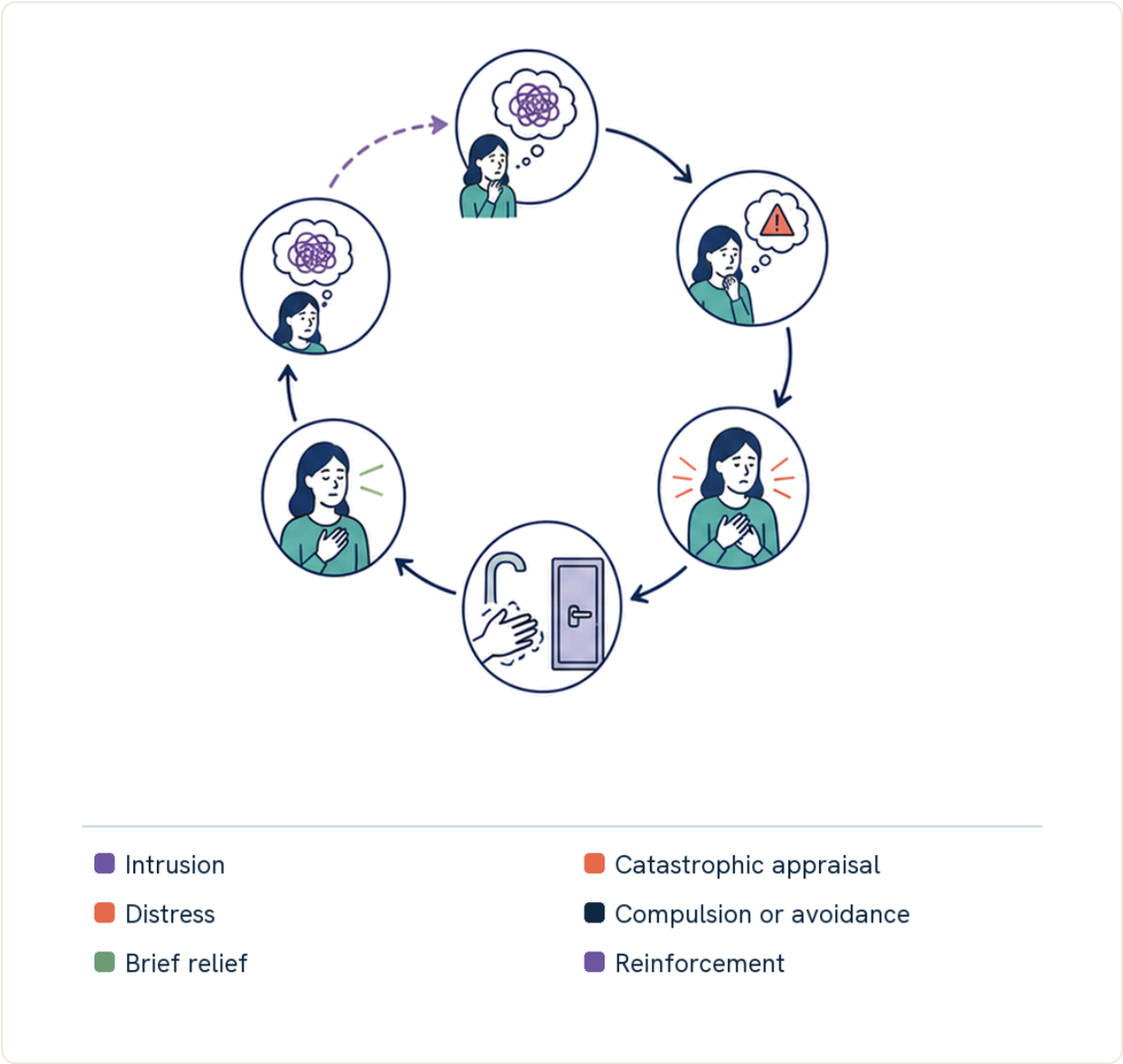



**Fig 14.8** The OCD maintenance cycle: an intrusion is appraised as threatening, producing distress and a compulsion or avoidance response; brief relief negatively reinforces the response and strengthens the cycle.

**Why it matters, and what this note owns.** Examiners test three things on this topic and this note owns all three: the exact definitions of obsession and compulsion (the ego-dystonic, intrusive, resisted quality is the whole discriminator), the four **symptom dimensions** and how each obsession pairs to its compulsion, and the **insight specifier** that ranges from good insight to frankly delusional conviction, alongside the **tic-related ocd** specifier. The management story (a high-dose SSRI for a long trial, then clomipramine, then antipsychotic augmentation, with exposure and response prevention as the psychological spine) is developed in its own management note; the deep antidepressant pharmacology of the SSRIs and clomipramine lives in the Mood disorders: pharmacotherapy notes, and neurosurgery and deep brain stimulation for the refractory patient in the ECT and neurostimulation notes. What follows is the phenomenology, the dimensions, the insight and tic specifiers, and the epidemiology and course.

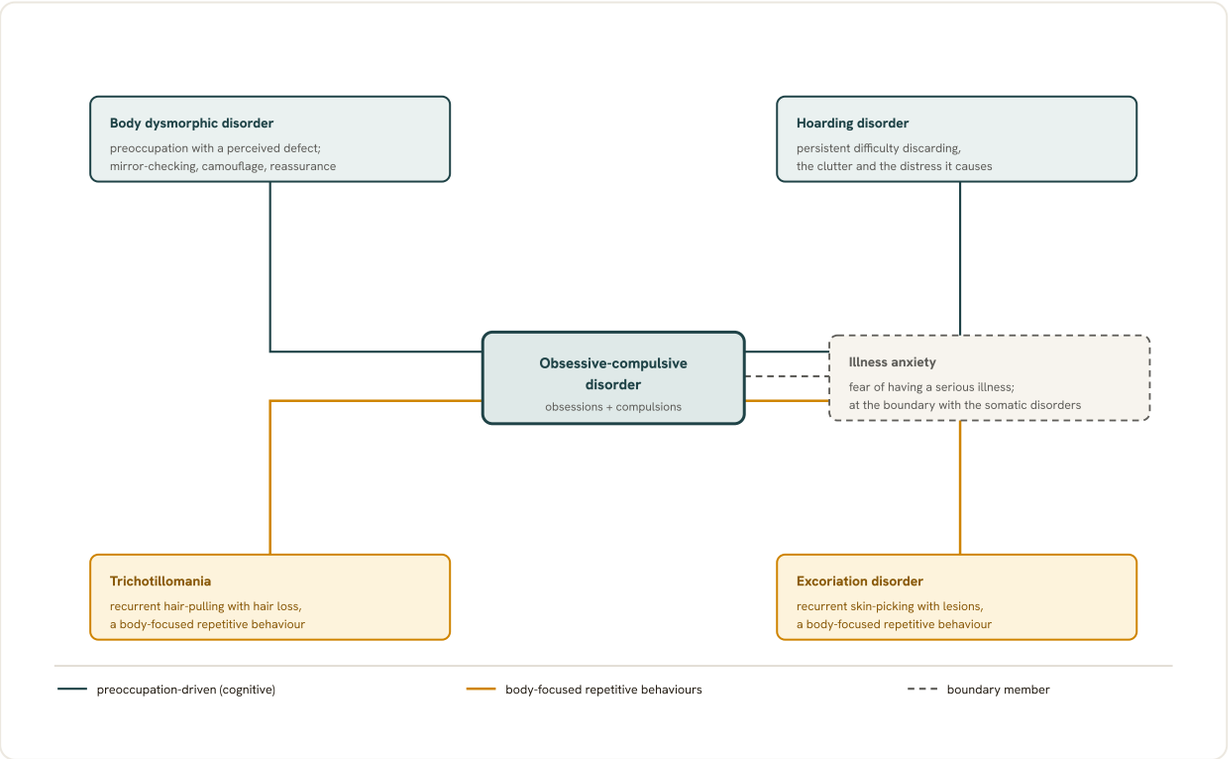



**Fig 14.9** The obsessive-compulsive and related disorders spectrum. OCD sits at the centre; the related disorders share either a driving preoccupation or a repetitive behaviour. Body dysmorphic disorder (a preoccupation with a perceived defect), hoarding disorder (the difficulty discarding) and, at the boundary with the somatic-symptom disorders, illness anxiety are the preoccupation-driven members; trichotillomania (hair-pulling) and excoriation (skin-picking) are the body-focused repetitive behaviours. Grouping them as a spectrum is the pedagogic point: each is recognised by the shared thread of a repetitive act or a fixed preoccupation, and each responds, in part, to the serotonin reuptake inhibitors and to the behavioural therapies. The somatic-symptom boundary is taught in the Somatic-symptom, sexual, impulse-control disorders and suicide notes.

## 11.1 Obsessions: intrusive, unwanted, ego-dystonic mental events

**The definition to state cold.** Obsessions are recurrent and persistent thoughts (e.g. of contamination), images (e.g. of a violent or horrific scene) or urges/impulses (e.g. to stab someone) that are experienced as *intrusive and unwanted* and that, in most people, cause marked anxiety or distress (DSM-5-TR 2022; ICD-11 CDDR). Three qualities carry the diagnosis. They are **intrusive** (they force their way into awareness against the person's will), **unwanted** (not pleasurable, not experienced as voluntary), and **ego-dystonic** (felt as alien to the self and as senseless or repugnant, which is why the person tries to resist them). The individual attempts to ignore or suppress the obsession, or to neutralise it with another thought or action, that is, with a compulsion.

**The ego-dystonic hinge.** The ego-dystonic quality is the single most useful discriminator. A worry in generalised anxiety disorder is ego-syntonic (the person endorses it as a reasonable, if excessive, concern); a delusion is held as true; a ruminative depressive thought is congruent with low mood. An obsession, by contrast, is experienced as intrusive, senseless and one's own but unwanted, which is exactly why the patient fights it. That fight, and the distress it generates, is what makes it pathological rather than merely an odd thought.

**GOOD TO REMEMBER**

- **Obsession:** a recurrent, persistent thought, image or urge experienced as intrusive, unwanted and ego-dystonic, causing marked anxiety or distress; the person resists or tries to neutralise it (usually with a compulsion); the ego-dystonic, resisted quality is the defining discriminator from a GAD worry (ego-syntonic) and from a delusion (held as true).

### 11.2 Compulsions: repetitive acts that neutralise, not connected or clearly excessive

**The definition to state cold.** Compulsions are repetitive behaviours (e.g. hand washing, ordering, checking) or mental acts (e.g. praying, counting, repeating words silently) that the person feels *driven to perform* in response to an obsession or according to rules that must be applied rigidly (DSM-5-TR 2022). Their aim is to prevent or reduce anxiety or distress, or to prevent some dreaded event or situation, or to reach a sense of **completeness** or "just right" (ICD-11 CDDR). Two qualifiers are essential: the compulsion is either *not connected in a realistic way* to what it is meant to neutralise (arranging items symmetrically to stop harm coming to a relative), or is *clearly excessive* (showering for hours to prevent illness).

**Mental compulsions are still compulsions.** A high-yield trap is to equate compulsions with visible behaviours only. Mental acts, silent counting, praying to a formula, mentally reviewing a memory to be sure no harm was done, are compulsions too, and a patient with only intrusive thoughts and covert mental rituals ("pure O") can be mislabelled as having obsessions alone. Compulsions are not done for pleasure, though they may bring transient relief; that relief is what negatively reinforces and perpetuates the cycle.

**WORKED EXAMPLE**

A young man is plagued by intrusive images of harm coming to his mother whenever he leaves the house (obsession: intrusive, unwanted, ego-dystonic). To neutralise the image he touches the doorframe in sets of four and mentally recites a fixed phrase until it feels "just right" (compulsions: one overt, one mental, driven, rigidly ruled). The ritual is not realistically connected to his mother's safety and consumes over an hour a day. Obsession and compulsion are thematically paired (harm and checking/undoing), which is the typical structure.

**GOOD TO REMEMBER**

- **Compulsion:** a repetitive behaviour or mental act the person feels driven to perform in response to an obsession or by rigid rules, aimed at reducing distress or preventing a dreaded event or reaching a "just right" sense; it is either not realistically connected to what it neutralises or is clearly excessive; mental rituals (counting, praying, reviewing) count as compulsions.

### 11.3 Obsessions and compulsions are thematically paired

**The pairing rule.** Most people with OCD have both obsessions and compulsions, and the two are typically related in content or temporal sequence: contamination obsessions drive washing rituals, harm obsessions drive checking, symmetry obsessions drive ordering and repeating. Compulsions are usually performed *in response to* the obsession, but ICD-11 notes that early in the illness a compulsion may precede a clear obsession (a person checks the gas knob repeatedly

and only then concludes they must be afraid of fire). Understanding the obsession-compulsion link matters for treatment, because exposure and response prevention targets exactly that link.

**Associated features that examiners like.** Sensory phenomena, physical "just-right" sensations or a feeling of incompleteness that precede compulsions, are reported by up to **60%** of patients (DSM-5-TR 2022; Tasman). Avoidance of triggers is common, and the characteristic dysfunctional beliefs are an inflated sense of responsibility, overestimation of threat, perfectionism, intolerance of uncertainty and an overvaluation of the power of thoughts (believing that having a forbidden thought is as bad as acting on it). Family involvement in rituals, termed *accommodation*, maintains symptoms and is a treatment target, especially in children.

RULE **Obsession and compulsion are a matched pair.** The obsession is the intrusive, ego-dystonic thought; the compulsion is the driven, neutralising act; they are thematically linked (contamination and washing, harm and checking), and it is that link, not the behaviour alone, that defines and is treated in OCD.

### 11.4 The four symptom dimensions

**The dimensional map.** The content of obsessions and compulsions varies between individuals but clusters into recognised symptom dimensions that are consistent over time in adults, occur across cultures and may map onto partly different neural substrates (DSM-5-TR 2022; ICD-11 CDDR). DSM-5-TR names four: **contamination and washing** (contamination obsessions with cleaning/washing compulsions); **symmetry and ordering** (symmetry obsessions with repeating, ordering and counting compulsions, often with "just-right" sensory phenomena); **forbidden or taboo thoughts** (aggressive, sexual or religious/scrupulosity obsessions and related compulsions, often mental); and **harm and checking** (fears of harm to self or others with checking compulsions). ICD-11 CDDR groups the first three and treats **hoarding**-type accumulation arising from typical obsessions (e.g. fear of harming others) as a consequence rather than a dimension. Most patients have symptoms in more than one dimension.

**The hoarding caveat.** Accumulation driven by an obsession (keeping items for fear that discarding them will cause harm) sits within OCD, but it must be distinguished from primary *hoarding disorder*, now a separate diagnosis in the obsessive-compulsive and related grouping, in which the difficulty discarding is the primary problem and is driven by a perceived need to save and distress at discarding, not by a classic obsession. Naming that boundary is the examiner's expected nuance.

| DIMENSION | TYPICAL OBSESSION | TYPICAL COMPULSION |
|---|---|---|
| **Contamination and washing** | **Fear of germs, dirt, illness, contamination** | Washing, cleaning, decontamination rituals, avoidance |
| **Symmetry and ordering** | Need for symmetry, exactness, "just right" | **Ordering, arranging, repeating, counting** |
| **Forbidden or taboo thoughts** | **Aggressive, sexual or religious (scrupulosity) intrusions** | Mental rituals, praying, confessing, reassurance-seeking |
| **Harm and checking** | Fear of harm to self or others | Checking locks, taps and gas; rechecking; reassurance-seeking |

The four DSM-5-TR symptom dimensions of OCD; each obsession pairs to a characteristic compulsion, and most patients span more than one dimension (DSM-5-TR 2022; ICD-11 CDDR groups the first three).

**GOOD TO REMEMBER**

- **Symptom dimensions:** four DSM-5-TR clusters, contamination and washing; symmetry and ordering; forbidden or taboo thoughts (aggressive/sexual/religious); and harm with checking, ICD-11 grouping the first three; each obsession pairs to its compulsion, most patients span more than one dimension, and obsession-driven accumulation must be separated from primary hoarding disorder.

### 11.5 The insight specifier: good/fair, poor, absent/delusional

**Insight is a spectrum, and it is specified.** People with OCD vary in how far they recognise that their obsessive-compulsive beliefs are untrue or excessive, and DSM-5-TR captures this with a three-level insight specifier (DSM-5-TR 2022). *With good or fair insight*: the person recognises the OCD beliefs are definitely or probably not true, or may or may not be true. *With poor insight*: the person thinks the beliefs are probably true. *With absent insight/delusional beliefs*: the person is completely convinced the beliefs are true. Poorer insight is linked to worse long-term outcome, and insight can fluctuate within a person over the course of illness (and with the current anxiety level), which is why it should be judged over a period of a few days to a week rather than in a single anxious moment.

**The ICD-11 two-level version.** ICD-11 CDDR uses a two-level specifier, *with fair to good insight* and *with poor to absent insight*, collapsing the DSM's poor and absent categories; it applies the same insight logic across the cognitive obsessive-compulsive and related disorders (OCD, body dysmorphic disorder, olfactory reference disorder, hypochondriasis and hoarding disorder). The clinically vital point on both systems is that the delusional-conviction end of OCD is *not* reclassified as a psychotic disorder: an absent-insight/delusional OCD belief stays within OCD (with the specifier) and is treated as OCD, not as schizophrenia.

| INSIGHT LEVEL (DSM-5-TR) | THE PERSON'S STANCE ON THE OCD BELIEF |
|---|---|
| **Good or fair insight** | Recognises beliefs are definitely/probably not true, or may or may not be true |
| **Poor insight** | **Thinks the beliefs are probably true** |
| **Absent insight / delusional** | **Completely convinced the beliefs are true (about 4% or fewer; still coded as OCD, not psychosis)** |

The DSM-5-TR insight specifier; ICD-11 collapses the lower two into a single "poor to absent" level, and the delusional end stays within OCD rather than becoming a psychotic diagnosis (DSM-5-TR 2022; ICD-11 CDDR).

**COMMON TRAP**

Do not reclassify an absent-insight, delusionally-held OCD belief as a psychotic disorder. A small minority (around 4% or fewer) are completely convinced their obsessive belief is true; this is coded as OCD "with absent insight/delusional beliefs", not as schizophrenia or delusional disorder, and it is still treated with the OCD algorithm (SSRI, then augment), not primarily with an antipsychotic. Missing this leads to the wrong diagnosis and the wrong first-line drug.

**GOOD TO REMEMBER**

- **Insight specifier:** DSM-5-TR grades insight as good/fair, poor, or absent/delusional (about 4% or fewer are delusional); ICD-11 uses two levels (fair-to-good vs poor-to-absent); poorer insight predicts worse outcome; the delusional end stays coded as OCD, not a psychotic disorder, and keeps the OCD treatment algorithm.

### 11.6 The tic-related specifier

**What it flags.** DSM-5-TR adds a tic-related specifier: **tic-related ocd** is diagnosed when the individual has a current or past history of a tic disorder (DSM-5-TR 2022). It matters because up to **30%** of people with OCD have a lifetime tic disorder, most commonly males with childhood-onset OCD, and this subgroup differs in symptom themes (more symmetry, "just-right" and ordering), comorbidity (Tourette syndrome, ADHD), course and pattern of familial transmission. Tourette syndrome is cross-listed alongside OCD in ICD-11 precisely because of this high co-occurrence, the shared premonitory-urge phenomenology and the familial link.

**The treatment relevance.** The tic-related subtype is the one classic exception to the "antipsychotics do nothing on their own in OCD" rule: patients with comorbid tics tend to respond somewhat better to antipsychotic augmentation of an SSRI. So the specifier is not merely descriptive, it nudges the augmentation choice, which is why it is worth naming in an answer.

**GOOD TO REMEMBER**

- **Tic-related OCD:** the DSM-5-TR specifier for a current or past tic disorder; up to 30% of OCD patients have a lifetime tic disorder (mostly males, childhood onset); the subtype skews to symmetry/"just-right"/ordering symptoms with Tourette and ADHD comorbidity, and responds relatively better to antipsychotic augmentation of an SSRI.

### 11.7 Criteria in brief: the time-consuming threshold and the boundary with normality

**The threshold.** Beyond the presence of obsessions and/or compulsions, both systems require that they are *time-consuming* (a benchmark of more than **1 hour per day** is given) *or* cause clinically significant distress or functional impairment (DSM-5-TR 2022; ICD-11 CDDR). The symptoms must not be attributable to a substance or another medical condition, and must not be better explained by another mental disorder. ICD-11 lists a set of exclusions to work through, so a checking obsession is not GAD worry, a preoccupation with appearance is body dysmorphic disorder, difficulty discarding is hoarding disorder, hair-pulling is trichotillomania, and so on.

**The boundary with normality.** Intrusive thoughts and repetitive behaviours are common in the general population (a fleeting thought of harming a loved one, double-checking the front door). What converts them into OCD is the time-consuming, distressing, impairing quality; developmentally normative childhood rituals (avoiding pavement cracks) are transient, age-appropriate and not time-consuming, and should not be over-diagnosed. Sudden or unusually late onset should prompt a search for an organic cause, for example a basal ganglia lesion or, in children with abrupt onset, paediatric autoimmune neuropsychiatric disorders associated with streptococcal infection (PANDAS).

TRAP **Do not diagnose OCD from the content alone.** Intrusive thoughts and double-checking are near-universal; OCD needs the symptoms to be time-consuming (over an hour a day) or to cause significant distress or impairment, and a sudden or late-onset picture should trigger a hunt for an organic mimic (basal ganglia lesion, PANDAS) before the label is fixed.

### 11.8 The Yale-Brown Obsessive Compulsive Scale (Y-BOCS): the severity instrument

**What it is.** The Yale-Brown Obsessive Compulsive Scale (Y-BOCS) is the standard clinician-rated severity measure in OCD and the outcome instrument in virtually every drug trial (Kaplan and Sadock 2024). It has **10 items** rated from a semi-structured interview: the first five rate obsessions and the second five rate compulsions, across the same five parameters, time occupied, interference with functioning, distress caused, resistance, and degree of control. Each item scores **0 to 4**, so obsessions and compulsions each total 0 to 20 and the overall score runs **0 to 40**. It is preceded by a symptom checklist that captures the specific content across the dimensions, and it is reproduced on the accompanying scale plate rather than pasted here. The paediatric version is the CY-BOCS.

**Reading the number.** Higher scores mean greater severity, and typical scores for patients presenting with OCD fall in the **16 to 30** range; a threshold of **16** is the usual cut-off for entry into drug trials (Kaplan and Sadock 2024). The Y-BOCS is also how treatment "response" is defined in the literature, most often as a reduction of at least 25 to 35% from the baseline Y-BOCS score, which is why the scale, not just a clinical impression, anchors the decision on whether an adequate trial has failed.

**GOOD TO REMEMBER**

- **Y-BOCS:** clinician-rated, 10 items (5 obsessions + 5 compulsions across time, interference, distress, resistance, control), each 0 to 4, obsessions and compulsions each 0 to 20, total 0 to 40; typical OCD scores 16 to 30, with 16 the usual drug-trial entry threshold; it is the standard severity and outcome measure (response commonly defined as a 25 to 35% reduction). Paediatric form: CY-BOCS.

| | | | |
|---|---|---|---|
| **0 to 40**<br>Y-BOCS TOTAL (10 ITEMS, EACH 0 TO 4) | **16**<br>Y-BOCS DRUG-TRIAL ENTRY THRESHOLD; TYPICAL OCD 16 TO 30 | **1 hour**<br>TIME-CONSUMING BENCHMARK (PER DAY) IN THE CRITERIA | **30%**<br>LIFETIME TIC DISORDER IN OCD (TIC-RELATED SUBTYPE) |

## 11.9 Epidemiology and course

**The numbers.** OCD has a lifetime prevalence of roughly **2 to 3%**, one of the more common serious psychiatric disorders; the classic Epidemiologic Catchment Area figures were about 2.9% in women and 2% in men, a modest female excess in adults (Tasman). The sex balance flips by age of onset: childhood-onset OCD, particularly before the age of 10, is commoner in males and carries greater genetic loading and poorer outcome, whereas adolescent-onset skews female. Around 30% of patients have a lifetime tic disorder, and only about 4% or fewer sit at the absent-insight/delusional end.

**The course.** Onset is typically in the late teens and early twenties, usually gradual; onset after 35 is uncommon and, like sudden onset, should prompt a search for an organic cause. Many adults (30 to 50%) report childhood onset. In adults OCD is generally a chronic condition with waxing and waning of symptoms; a minority run an episodic or a progressively worsening course. There is a more hopeful paediatric signal: of those with childhood or adolescent onset, roughly **40%** achieve full remission by early adulthood (ICD-11 CDDR). Depression is the commonest comorbidity, and untreated OCD is characteristically slow to reach care.

**GOOD TO REMEMBER**

- **OCD epidemiology and course:** lifetime prevalence about 2 to 3% (slight adult female excess; childhood onset before age 10 is male-predominant with worse prognosis); onset usually late teens to early twenties and gradual, with onset after 35 or sudden onset flagging an organic cause; adult course is chronic and waxing-waning, but around 40% of childhood-onset cases remit fully by early adulthood.

## 11.10 The India lens: a wide OCD treatment gap and a benzodiazepine reflex

**Under-recognised and under-treated.** OCD in India carries a wide treatment gap. Symptoms are hidden by shame (contamination, taboo sexual or religious/scrupulosity obsessions are rarely volunteered), long delays precede a first consultation, and the disorder is under-recognised in general practice. Culturally shaped content is genuine: scrupulosity and religious obsessions may be especially distressing where ritual exactitude matters, and contamination themes (concepts of ritual purity and pollution) are common; distinguishing a religious compulsion from devout normative practice sometimes needs a knowledgeable religious informant (ICD-11 CDDR). The

Indian Psychiatric Society CPGs frame the correct approach, an adequate high-dose SSRI trial with exposure and response prevention, developed in the management note.

**The benzodiazepine problem.** Into this gap flows the same reflex that dogs the anxiety disorders in Indian practice: a benzodiazepine (often alprazolam) prescribed as if it were treatment. It is not. Benzodiazepines do nothing for the core obsessions and compulsions, at best they blunt the accompanying anxiety, and long-term use brings tolerance and dependence. The first-line agents are cheap and familiar as Indian brands, escitalopram, sertraline, fluoxetine, fluvoxamine and paroxetine among the SSRIs, and clomipramine (Anafranil) as the tricyclic option, used at high dose for a long trial. Naming the ego-dystonic obsession and the neutralising compulsion, and reaching for an SSRI rather than an anxiolytic, is the practical correction.

INDIA

OCD is common (lifetime prevalence around 2 to 3%) but reaches Indian care late and often disguised, contamination and religious/scrupulosity themes are frequent and culturally shaped, and the treatment gap is wide. The recurring error is treating it with a benzodiazepine (typically alprazolam), which touches none of the obsessions or compulsions; the Indian Psychiatric Society line, and the correct answer, is a high-dose SSRI (escitalopram, sertraline, fluvoxamine, fluoxetine or paroxetine) or clomipramine (Anafranil) with exposure and response prevention, the benzodiazepine having no role in core OCD treatment.

MNEMONIC

### CObRA-DIT

The features to reproduce. **CObRA** holds the definition and the four dimensions: **C**ontamination/washing, **O**rder/symmetry, **b**ad/forbidden taboo thoughts, **R**isk of harm with checking, all built from an **A**lien (ego-dystonic) obsession neutralised by a compulsion. **DIT** holds the specifiers and cut: **D**istress/time-consuming (over 1 hour a day), **I**nsight graded good-fair to absent/delusional, **T**ic-related subtype.

HOLD THIS

OCD is intrusive, unwanted, ego-dystonic obsessions neutralised by driven, rigidly-ruled compulsions (behavioural or mental) that are not realistically connected or clearly excessive and are time-consuming (over an hour a day) or impairing; it runs across four DSM-5-TR dimensions (contamination/washing, symmetry/ordering, forbidden thoughts, harm/checking), carries an insight specifier from good-or-fair through poor to absent/delusional (delusional OCD stays OCD, about 4% or fewer) and a tic-related specifier (up to 30% lifetime tics); severity is scored on the Y-BOCS (10 items, 0 to 40), lifetime prevalence is about 2 to 3%, and onset is usually the late teens to early twenties.

## 12 · The OCD management algorithm

OCD is one of the two topics that examiners use to test whether you actually understand psychiatric prescribing rather than reciting a first-line drug. The disorder responds to the same serotonergic agents as depression, but the *rules of engagement are different*: the dose runs higher,

the trial runs longer, and the response is judged on a scale rather than a gestalt. **ocd management** is therefore a small, tight algorithm with four rungs, and the whole thing turns on one idea you must be able to state in a single breath, a **high-dose ssri** given for a **long trial** alongside **erp**, then clomipramine, then antipsychotic augmentation, then the refractory options.

**Why it matters, and what this note owns.** This note owns the ladder and the numbers, the target doses, the trial durations, the Y-BOCS response and remission thresholds, and where each guideline sits. The deep pharmacology of the SSRIs and of clomipramine (kinetics, adverse effects, interactions) is developed in the Mood disorders: pharmacotherapy notes and is not re-derived here; the general CBT model and the wider exposure principle behind ERP live in the Psychotherapies, clinical assessment, MSE and nosology notes; and deep brain stimulation and ablative neurosurgery for the truly refractory patient are cross-referenced to the ECT and neurostimulation notes. The management algorithm itself is set out in the accompanying figure, and the Y-BOCS form on the accompanying scale plate.

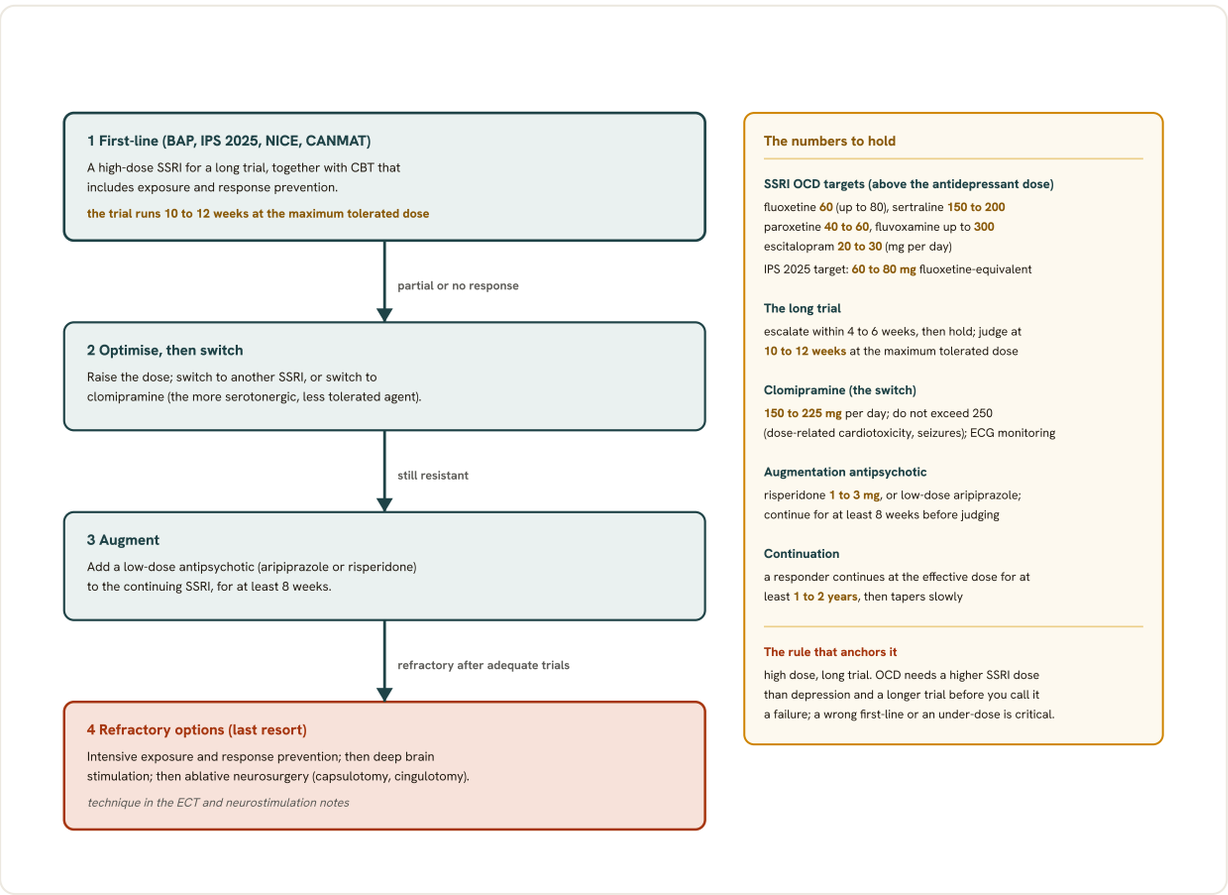



**Fig 14.10** The OCD management algorithm. First-line, anchored on BAP, the IPS 2025 CPG, NICE and CANMAT, is a high-dose serotonin reuptake inhibitor given for a long trial of 10 to 12 weeks together with cognitive behaviour therapy that incorporates exposure and response prevention. On a partial or absent response the dose is optimised and the drug switched to another SSRI or to clomipramine (150 to 225 mg with ECG monitoring); a still-resistant illness is augmented with a low-dose antipsychotic (risperidone 1 to 3 mg or aripiprazole) for at least eight weeks; and the refractory case moves to intensive exposure and response prevention, then deep brain stimulation, then ablative neurosurgery. The anchoring rule is high dose, long trial: OCD needs a higher SSRI dose and a longer trial than depression, so under-dosing or stopping the trial early manufactures a false treatment failure. The deep antidepressant pharmacology is developed in the Mood disorders: pharmacotherapy notes, and the neurosurgical technique in the ECT and neurostimulation notes.

### 12.1 The shape of the ladder: four rungs and one governing principle

**The four rungs.** Every guideline collapses to the same staircase. *First-line* is a high-dose SSRI for a long trial *plus* CBT incorporating exposure and response prevention. *Partial or no response* means optimise the dose, then switch to another SSRI or to clomipramine. *Still resistant* means antipsychotic augmentation, low-dose aripiprazole or risperidone. *Refractory* means intensive ERP and, at a specialist centre, deep brain stimulation or ablative neurosurgery. Learn the staircase as a unit; the examiner wants the order, not a list.

**The governing principle.** The one line that runs through the whole ladder is that OCD is *under-treated by default*, because the doses are timid and the trials are cut short. Hold the principle first, then hang the drugs on it.

> **HOLD THIS**
> First-line OCD is a high-dose SSRI (target roughly fluoxetine 60 mg equivalent) for a long trial of about 10 to 12 weeks at the maximum tolerated dose, together with CBT incorporating exposure and response prevention; only then clomipramine, then aripiprazole or risperidone augmentation, then the refractory options.

### 12.2 First-line: a high-dose SSRI plus ERP, across every guideline

**The BAP position.** Lead with the British Association for Psychopharmacology: offer an SSRI as first-line pharmacological treatment for OCD, and consider higher daily SSRI doses, sometimes beyond standard formulary limits, because meta-analysis shows greater efficacy (though poorer tolerability) at higher dosages; offer exposure therapy or CBT as the evidence-based acute psychological treatment (BAP 2014). This is the verified **first-line ocd** anchor and must not be contradicted.

**What the other guidelines say, and the IPS 2025 line to quote.** The convergence is total. The Indian Psychiatric Society is explicit: offer an SSRI as the first-line pharmacological treatment and CBT with exposure and response prevention as first-line psychotherapy, treating OCD with *higher SSRI doses than those used for depression* and reducing to a low-to-medium dose only if the higher dose is not tolerated (the Indian Psychiatric Society 2025 / IPS 2025). NICE has adults choose an SSRI (fluoxetine, fluvoxamine, paroxetine, sertraline or citalopram) or more intensive CBT with ERP by degree of functional impairment, combining an SSRI with CBT for severe impairment (NICE 2005). CANMAT names an SSRI (escitalopram, fluoxetine, fluvoxamine, paroxetine or sertraline) and CBT with ERP as co-equal first-line, adding CBT to medication to enhance response and relapse prevention (CANMAT 2014). The WFSBP likewise puts an SSRI and CBT first-line across children, adolescents and adults (WFSBP 2023). Every source says the same thing: *SSRI plus ERP, and push the dose*.

| GUIDELINE | FIRST-LINE PHARMACOLOGY | FIRST-LINE PSYCHOLOGY / KEY NOTE |
|---|---|---|
| **BAP 2014** | **SSRI, high dose (may exceed formulary limits)** | ERP / CBT; clomipramine equally efficacious but less tolerated |
| **the Indian Psychiatric Society 2025** | **SSRI, higher than depression doses** | CBT with ERP; monotherapy for mild-moderate, SSRI-combined for severe |
| **NICE 2005** | SSRI (fluoxetine, fluvoxamine, paroxetine, sertraline, citalopram) | CBT with ERP matched to impairment; combined if severe |
| **CANMAT 2014** | SSRI (escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline) | CBT with ERP co-equal first-line |

First-line OCD across the major guidelines; the shared message is a high-dose SSRI plus CBT incorporating exposure and response prevention, with the IPS 2025 CPG and BAP both stressing doses above the antidepressant range.

RULE **High dose, long trial, with ERP.** First-line OCD is an SSRI pushed to the maximum tolerated dose (above the depression range) for about 10 to 12 weeks, combined with CBT incorporating exposure and response prevention; this is unanimous across BAP, the Indian Psychiatric Society, NICE, CANMAT and WFSBP.

### 12.3 The high-dose rule: the SSRI target doses to quote

**Higher than the antidepressant dose.** The doses of SSRIs used in OCD run above those used in depression: fluoxetine to **60 mg** (sometimes 80), paroxetine **40 to 60 mg**, sertraline typically **150 to 200 mg** (the Maudsley notes sertraline has been pushed to 250 to 400 mg and even 650 mg without major safety problems), fluvoxamine to 300 mg, and escitalopram 20 to 30 mg (Maudsley Prescribing Guidelines; the Indian Psychiatric Society 2017/2025). The IPS 2025 target is pragmatic and worth quoting verbatim: aim for roughly the **60 to 80 mg** fluoxetine-equivalent, reducing to a tolerated dose only if the higher dose is not tolerated (IPS 2025). Escalate to the effective dose within about 4 to 6 weeks, then hold.

**The one citalopram caution.** The FDA cautions against citalopram above 40 mg/day because of dose-dependent QTc prolongation, so citalopram is the one SSRI whose OCD dose does *not* get pushed indefinitely (BAP 2014).

| SSRI | INDIAN BRANDS (EXAMPLES) | OCD TARGET DOSE (MG/DAY) |
|---|---|---|
| **Fluoxetine** | Fludac, Prodep | **60 (up to 80)** |
| **Sertraline** | Daxid, Serta, Zosert | 150 to 200 (higher tried) |
| **Fluvoxamine** | Fluvoxin | up to 300 |
| **Paroxetine** | Paxidep, Xet | 40 to 60 |
| **Escitalopram** | Nexito, Stalopam | 20 to 30 |

OCD SSRI target doses run above the antidepressant range; the exam anchor is fluoxetine 60 mg and the IPS 2025 target of 60 to 80 mg fluoxetine-equivalent (Maudsley; IPS 2025). Doses in cells are bare numbers; the unit is in the header.

**GOOD TO REMEMBER**

- **SSRI (first-line for OCD):** serotonin reuptake inhibitor; the OCD principle is a dose *higher* than the depression dose (fluoxetine 60 mg, target 60 to 80 mg fluoxetine-equivalent) held for a long trial; the signature caution is dose-dependent QTc prolongation limiting citalopram to 40 mg/day; the one first-line indication to state is high-dose SSRI plus ERP for all severities of OCD.

### 12.4 The long trial: judging response on the Y-BOCS at 10 to 12 weeks

**Why the trial is long.** Initial response in OCD emerges more slowly than in depression: it can take **10 to 12 weeks** at the maximum tolerated dose before you can call a trial failed (Maudsley; the Indian Psychiatric Society 2025). The IPS 2025 rule is precise: continue a maximally tolerated effective SSRI dose for at least **12 weeks** before judging efficacy, having escalated to the effective dose within 4 to 6 weeks (IPS 2025). The New Oxford Textbook frames the failure point the same way, under 25% reduction of the baseline Y-BOCS after 12 weeks of SRI treatment, of which at least 6 weeks should be at the maximal tolerated dose, counts as non-response.

**The scale that governs the algorithm.** The Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) is the clinician-rated, semi-structured instrument used at baseline and every 4 weeks to objectify response. It rates obsessions and compulsions separately on five aspects each (time, interference, distress, resistance, control), scored 0 to 4, for a total of **0 to 40**. The Goodman severity bands are worth memorising cold: 0 to 7 subclinical, 8 to 15 mild, 16 to 23 moderate, 24 to 31 severe, 32 to 40 extreme. A **25 to 35%** reduction is considered response; remission is a Y-BOCS of **12 or below** (the Indian Psychiatric Society 2017). In children the equivalent is the CY-BOCS (mild 16 to 19, moderate 20 to 29, severe 30 to 40) (the Indian Psychiatric Society 2019). The form itself is reproduced on the accompanying scale plate.

| Y-BOCS TOTAL | SEVERITY BAND |
|---|---|
| **0 to 7** | Subclinical |
| **8 to 15** | Mild |
| **16 to 23** | **Moderate** |
| **24 to 31** | Severe |
| **32 to 40** | Extreme |

Y-BOCS severity bands (Goodman et al 1989): 10 items each 0 to 4, total 0 to 40; response is a 25 to 35% reduction, remission a score of 12 or below.

**GOOD TO REMEMBER**

- **Y-BOCS:** clinician-rated 10-item scale, each 0 to 4, total 0 to 40; bands 0-7 subclinical, 8-15 mild, 16-23 moderate, 24-31 severe, 32-40 extreme; response = 25 to 35% reduction, remission = 12 or below; it is the instrument on which every rung of the OCD algorithm is judged, rated at baseline and every 4 weeks.

**COMMON TRAP**

The classic error is declaring an SSRI a failure too early or at too low a dose. An OCD trial is not adequate until the patient has had roughly 10 to 12 weeks at the maximum tolerated dose, with at least 6 of those weeks at the ceiling. Switching or augmenting before that, or judging on the depression-range dose, mislabels a slow-but-real responder as treatment-resistant and sends the ladder off prematurely.

### 12.5 CBT incorporating exposure and response prevention: the first-line psychotherapy

**What it is.** **cbt for ocd** (CBT tailored to obsessions and compulsions) is built around exposure and response prevention (erp): the patient is exposed, in a graded hierarchy, to the feared stimulus (the contaminated object, the intrusive thought) and then *prevented* from performing the neutralising ritual, so that anxiety habituates and the belief that the ritual averts catastrophe is disconfirmed. Foa's work established that exposure and ritual prevention must be delivered *together*, exposure alone or response prevention alone yields inferior outcomes. It is first-line psychotherapy in every guideline, monotherapy for mild-to-moderate illness and combined with an SSRI for severe illness (the Indian Psychiatric Society 2025; CANMAT 2014).

**Where it sits on the ladder.** ERP is not only a first-line treatment but also a first-line *augmentation* for partial or non-responders to an SSRI, where feasible (IPS 2025). Therapist-guided exposure is preferred to self-exposure (CANMAT 2014), and family accommodation should be assessed and reduced, since high accommodation predicts poor outcome (the Indian Psychiatric Society 2017).

**GOOD TO REMEMBER**

- **CBT with ERP (first-line psychotherapy for OCD):** graded exposure to the feared stimulus with prevention of the neutralising ritual, driving habituation and belief disconfirmation; the principle is that exposure and response prevention must be delivered together (either alone is inferior); the first-line indication is mild-to-moderate OCD as monotherapy and severe OCD combined with a high-dose SSRI.

### 12.6 Partial or no response: optimise, then switch SSRI or to clomipramine

**Optimise first, then switch.** When a first adequate SSRI trial fails, the sequence is optimise the dose (push to the maximum tolerated), then switch, either to a different SSRI or to clomipramine (BAP 2014; NICE 2005). Switching between treatments of proven efficacy helps some patients (BAP 2014). The IPS 2025 places clomipramine as a *second-line* agent, used when SSRIs are ineffective, given its poorer tolerability (IPS 2025).

**The clomipramine-versus-SSRI point.** This is the classic exam contrast. Clomipramine is the one tricyclic with genuine anti-obsessional efficacy (it is the most serotonergic TCA), and historically the first drug shown to work in OCD (1967). Its efficacy is at the margins of superiority over the SSRIs, but it is *less well tolerated* (anticholinergic burden, cardiac conduction effects, seizure risk in overdose), which is exactly why an SSRI is tried first and clomipramine held in reserve (BAP 2014). The OCD dose is **150 to 225 mg/day** (range 75 to 250), and ECG

monitoring is required (the Indian Psychiatric Society 2025 / 2017). Intravenous clomipramine is a specialist option after oral non-response.

TRAP **Clomipramine is not more effective enough to use first.** All SSRIs and clomipramine are efficacious in OCD; clomipramine sits at the margins of superiority but is less tolerated, so an adequate SSRI trial (high dose, long) comes first, with clomipramine as the switch on failure, under ECG monitoring.

GOOD TO REMEMBER

- **Clomipramine (Indian brand Anafranil):** the most serotonergic tricyclic and the original anti-obsessional drug; mechanism is potent serotonin (and noradrenaline) reuptake inhibition; second-line in OCD at 150 to 225 mg/day because it is less tolerated than an SSRI (anticholinergic, cardiac conduction, seizure in overdose), so it needs ECG monitoring and is used as the switch after an adequate SSRI trial fails.

### 12.7 Augmentation: low-dose aripiprazole or risperidone added to the SSRI

**The third rung.** For the patient who is a partial responder after an adequate SSRI or clomipramine trial, the next step is **augmentation** with a low-dose antipsychotic added to the continuing serotonergic drug. The IPS 2025 names low-dose aripiprazole or risperidone as the first-line pharmacological augmentation in treatment-resistant OCD, continued for at least 8 weeks; the doses are risperidone **1 to 3 mg/day** and aripiprazole in the low range (IPS 2025). BAP has the strongest augmentation evidence for risperidone, with aripiprazole also supported (BAP 2014), and CANMAT lists adjunctive aripiprazole or risperidone as the first-line adjuncts for treatment-resistant OCD (CANMAT 2014). The Maudsley agrees the usual second-line step is the addition of risperidone or aripiprazole.

**The monotherapy caveat.** Antipsychotics are for *augmentation only*: NICE is explicit that antipsychotics should not normally be used as monotherapy to treat OCD (NICE 2005). The augmentation target is often the patient with comorbid tic disorder or poorer insight.

GOOD TO REMEMBER

- **Antipsychotic augmentation (aripiprazole / risperidone):** low-dose dopamine blockade added to a continuing SSRI or clomipramine for treatment-resistant OCD (risperidone 1 to 3 mg/day, low-dose aripiprazole), for at least 8 weeks; strongest evidence is for risperidone; the signature caution is that antipsychotics are augmentation only and never monotherapy for OCD.

### 12.8 Refractory OCD: intensive ERP, then DBS and ablative neurosurgery

**Define refractory first.** **refractory ocd** means failure of at least two adequate SSRI trials, clomipramine, and CBT with ERP (with antipsychotic augmentation), each delivered at full dose and duration (the Indian Psychiatric Society 2017). Before escalating, the honest question is always whether the earlier trials were genuinely adequate, high dose, long enough, and with real ERP.

**The refractory rung.** For the genuinely refractory patient, intensify the psychological treatment (intensive, therapist-led ERP) and, at a specialist centre only, consider the somatic options:

intravenous clomipramine, then *deep brain stimulation*, and ablative neurosurgery (anterior capsulotomy or cingulotomy), reserved for treatment-refractory OCD after multidisciplinary review (BAP 2014; the Indian Psychiatric Society 2017; WFSBP 2023). In India this pathway runs through specialist centres such as NIMHANS. The neurostimulation and neurosurgical detail (DBS targets, capsulotomy) is developed in the ECT and neurostimulation notes. Neuromodulation such as rTMS or tDCS is an evolving, preliminary-evidence augmentation option (IPS 2025).

**WORKED EXAMPLE**

A 26-year-old man with contamination obsessions and washing rituals has a Y-BOCS of 28 (severe). He is started on fluoxetine, escalated to 60 mg over 5 weeks, and continued at 60 mg with weekly ERP. At 12 weeks his Y-BOCS is 25, under a 25% reduction, so this is non-response despite an adequate high-dose, long trial. The dose is optimised to 80 mg for a further period; still a partial responder, he is switched to a second SSRI, and on continued partial response low-dose risperidone (1 to 2 mg/day) is added for 8 weeks, with intensive ERP. Only after this whole sequence would clomipramine and, ultimately, specialist referral be considered.

### 12.9 Continuation and maintenance: at least 1 to 2 years at the effective dose

**Do not stop early.** OCD is chronic and relapsing, so a responder is continued on the effective SSRI (or clomipramine) at the *dose that produced improvement*. BAP and NICE say continue for at least 12 months; the IPS 2025 goes further, continue an effective SSRI or clomipramine for at least **1 to 2 years** after remission, at the dose that produced improvement (IPS 2025); CANMAT says at least 12 to 24 months (CANMAT 2014). The principle that motivates this: continuing an effective serotonin reuptake inhibitor markedly lowers the relapse rate compared with stopping after the initial response, so the effective dose is held rather than reduced. Taper slowly when eventually stopping, since anxiety-spectrum patients are sensitive to discontinuation.

| 60 | 10 to 12 weeks | 32 to 40 | 1 to 2 years |
|---|---|---|---|
| FLUOXETINE OCD TARGET, MG (HIGHER THAN DEPRESSION) | ADEQUATE TRIAL AT MAXIMUM TOLERATED DOSE | Y-BOCS EXTREME BAND (RESPONSE = 25 TO 35% DROP) | IPS 2025 CONTINUATION AFTER REMISSION |

**INDIA**

OCD sits inside India's wider anxiety-spectrum treatment gap: the disorder is common yet under-recognised, and the reflex of a benzodiazepine (still over-prescribed across Indian outpatient practice) does nothing for obsessions and risks dependence. The Indian Psychiatric Society CPGs, updated for OCD to 2025, anchor the same high-dose-SSRI-plus-ERP algorithm to Indian practice, and the tertiary pathway for refractory OCD (IV clomipramine, DBS, capsulotomy) runs through specialist centres such as NIMHANS. Indian brands are readily available, fluoxetine (Fludac, Prodep), sertraline (Daxid, Zosert), escitalopram (Nexito), fluvoxamine (Fluvoxin) and clomipramine (Anafranil), so the limiting factor is recognition and adequate dosing, not access.

MNEMONIC

**SSRI → SWITCH → ADD → REFER**

The four rungs in order: **SSRI** (high dose, long trial, with ERP) → **SWITCH** (optimise, then another SSRI or clomipramine) → **ADD** (aripiprazole or risperidone augmentation) → **REFER** (intensive ERP, then DBS / neurosurgery at a specialist centre).

PEARL

The single commonest reason "OCD does not respond to SSRIs" in the exam vignette is not true resistance but an inadequate trial, a depression-range dose stopped at 6 weeks. State the high-dose, long-trial rule and you have answered most OCD management questions before you reach the second rung.

## 13 · The Yale-Brown Obsessive Compulsive Scale

If a single instrument owns obsessive-compulsive disorder the way the HAM-D owns depression, it is this one. The **yale-brown obsessive compulsive scale** (**y-bocs**) is the standard clinician-rated severity measure for OCD and the outcome instrument in virtually every drug trial (Kaplan and Sadock 2024). Its design is deliberate and testable: it rates severity independently of symptom content, so a person whose only compulsion is silent counting and a person who washes for hours can be scored on the same scale and tracked over time. That content-blind design is exactly why examiners like it, and why it is the number that decides whether an adequate SSRI trial has actually failed.

**Why it matters, and what this note owns.** This is a scale note, so it teaches what the Y-BOCS is, how it is built, and, above all, its **severity bands**, the total-score ranges that convert a bare number into "mild", "moderate" or "extreme". The clinical phenomenology of obsessions and compulsions, the four symptom dimensions and the insight specifier live in the OCD clinical-features note; the high-dose SSRI, then clomipramine, then antipsychotic-augmentation algorithm lives in the OCD management note, and the deep antidepressant pharmacology in the Mood disorders: pharmacotherapy notes. What follows is the structure (a **ten-item** severity scale sitting on top of a symptom checklist), the scoring, the verified bands, and how the number is used to define response. The instrument is copyright Goodman and Rasmussen, so the accompanying scale plate typesets the structure and bands in our own words with a citation rather than reproducing the form; refer to that plate and do not expect the questionnaire pasted here.

| Y-BOCS (TYPESET, NOT REPRODUCED) | VERIFIED STRUCTURE AND SEVERITY BANDS |
|---|---|
| **Instrument** | Yale-Brown Obsessive Compulsive Scale, clinician-rated, the reference severity measure for OCD (Goodman, Price, Rasmussen and colleagues, 1989) |
| **Items** | 10 items: 5 rate the obsessions and 5 rate the compulsions, each across the same five parameters (time occupied, interference, distress, resistance and control); each item scored 0 to 4; total range 0 to 40 |
| **Companion** | a symptom checklist precedes the severity scale, so the clinician first maps the symptom dimensions and then rates their severity |
| **Severity bands** | **0 to 7 subclinical, 8 to 15 mild, 16 to 23 moderate, 24 to 31 severe, 32 to 40 extreme** |

Y-BOCS: typeset structure and verified bands only; the form is copyright Goodman and Rasmussen and its distribution is permission-required, so it is not reproduced (Goodman WK, Price LH, Rasmussen SA, et al. Arch Gen Psychiatry. 1989;46:1006-1011).

**Fig 14.11** The Yale-Brown Obsessive Compulsive Scale (Y-BOCS). The reference severity measure for OCD: ten clinician-rated items, five for the obsessions and five for the compulsions, each rating time occupied, interference, distress, resistance and control on a 0 to 4 scale, for a total of 0 to 40, preceded by a symptom checklist. The severity bands run 0 to 7 subclinical, 8 to 15 mild, 16 to 23 moderate, 24 to 31 severe and 32 to 40 extreme. The form is copyright-restricted, so its structure and verified bands are given here in typeset form rather than reproduced.

### 13.1 What the Y-BOCS is, and who built it

**The one-line definition.** The Yale-Brown Obsessive Compulsive Scale is a semi-structured, clinician-administered interview that measures the *severity* of obsessive-compulsive symptoms, developed by Goodman, Rasmussen and colleagues in the late 1980s and published in 1989 (Kaplan and Sadock 2024; Goodman et al. 1989). It has become the standard instrument for assessing OCD severity and is used in virtually every drug trial, and it may also be used clinically to monitor treatment response. The paediatric adaptation is the Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS), and a dimensional version (the DY-BOCS) also exists.

**Severity, not diagnosis.** The crucial framing point is that the Y-BOCS does not diagnose OCD; the diagnosis rests on the DSM-5-TR or ICD-11 criteria. The Y-BOCS presumes OCD is present and then quantifies how bad it is, which is why it is a severity and outcome measure rather than a screening or case-finding tool. It rates severity independently of the content or type of obsessions and compulsions, so the same score can be reached by very different symptom pictures.

**GOOD TO REMEMBER**

- **Y-BOCS:** a semi-structured, clinician-rated interview (Goodman, Rasmussen et al. 1989) that measures the *severity* of OCD, not the diagnosis; it is the standard OCD severity and outcome instrument used in virtually every drug trial; it rates severity independently of symptom content; paediatric version CY-BOCS.

### 13.2 Two parts: the symptom checklist, then the severity scale

**The checklist comes first.** The Y-BOCS is administered in two parts. Before the first use of the severity ratings, an associated symptom **checklist** is administered, a long menu (commonly cited as a 64-item checklist) covering the range of obsessions (aggressive, contamination, sexual, hoarding, religious/scrupulosity, symmetry, somatic) and compulsions (washing/cleaning, checking, repeating, counting, ordering/arranging, hoarding, mental rituals) organised by category (Kaplan and Sadock 2024; Foa et al.). Its job is to map *which* symptoms the patient has, which is invaluable for treatment planning and for anchoring the severity questions that follow.

**The severity scale comes second.** Once the clinician knows the range of symptoms the patient endorses, the **severity scale** proper is applied. This is the ten-item core that yields the 0 to 40 score, and its ratings are based on the range of symptoms the patient endorsed on the checklist. The distinction is a favourite exam point: the checklist tells you *what* the symptoms are; the severity scale tells you *how severe* they are and how they change with treatment. The severity scale is the more accurate tool for determining level of severity and tracking progress; the checklist is the better tool for understanding the range of a patient's symptoms.

RULE **Checklist maps content, severity scale measures severity.** Administer the symptom checklist first to establish *which* obsessions and compulsions are present; then apply the ten-item severity scale to those endorsed symptoms to produce the 0 to 40 severity score that is tracked over treatment.

### 13.3 The ten items: five obsessions, five compulsions, five parameters each

**The architecture to state cold.** The severity scale has **10 items**. The first five rate *obsessions* and the second five rate *compulsions*, and the two sets ask parallel questions across the same five parameters (Kaplan and Sadock 2024). The five parameters are: the **time** occupied by the symptoms, the **interference** they cause with functioning, the **distress** they cause, the patient's attempts to **resist** them, and the degree of **control** the patient has over them. Because the obsessions block and the compulsions block use the identical five parameters, learning the five parameters once gives you all ten items.

**Why the parallel structure matters.** The parallel obsessions-versus-compulsions design lets a clinician read an obsessions subtotal and a compulsions subtotal separately, which is useful when, for example, a "pure O" patient scores heavily on obsessions and lightly on compulsions. It also means treatment can be watched dimensionally: exposure and response prevention often moves the compulsions subscore first. The semi-structured interview and ratings can be completed in 15 minutes or less; a self-administered version takes about 10 to 15 minutes.

| ITEM BLOCK | ITEMS | THE FIVE PARAMETERS RATED (EACH 0 TO 4) |
|---|---|---|
| **Obsessions** | **Items 1 to 5** | Time occupied; interference; distress; resistance; control |
| **Compulsions** | **Items 6 to 10** | Time occupied; interference; distress; resistance; control (parallel) |

The ten-item severity scale: obsessions (items 1 to 5) and compulsions (items 6 to 10) rated across the identical five parameters, each item scored 0 to 4 (Kaplan and Sadock 2024).

**COMMON TRAP**

Do not confuse resistance with control, or read "high resistance" as a good sign. On the Y-BOCS a higher score is worse throughout, but the *resistance* item is scored counter-intuitively: making little or no effort to resist scores high (worse), while active resistance scores low. A patient who has "given up" fighting the compulsion scores worse on resistance, not better. Misreading the resistance item flips the interpretation of that item.

### 13.4 The scoring: 0 to 4 per item, subtotals 0 to 20, total 0 to 40

**The arithmetic to memorise.** Each of the ten items has item-specific anchors scored from **0 to 4** (Kaplan and Sadock 2024). The five obsessions items therefore sum to an obsessions subtotal of 0 to 20, the five compulsions items sum to a compulsions subtotal of 0 to 20, and the overall Y-BOCS total runs **0 to 40**. Higher scores mean greater severity. Typical scores for patients presenting with OCD fall in the **16 to 30** range, and a threshold of **16** is the value typically used for inclusion in drug trials, a number worth carrying because it recurs across the OCD trial literature.

**The anchors, in words.** Each 0 to 4 anchor is behaviourally defined, for example on the "time occupied by obsessions" item: 0 = none, 1 = mild (less than an hour a day), 2 = moderate (1 to 3 hours), 3 = severe (3 to 8 hours), 4 = extreme (more than 8 hours). That behavioural anchoring is what gives the scale its good inter-rater reliability. Reliability studies show good internal consistency, inter-rater reliability, and test-retest reliability over a one-week interval.

**WORKED EXAMPLE**

A patient rates 3 on obsession-time (3 to 8 hours of intrusive contamination thoughts), 3 on interference, 3 on distress, 2 on resistance and 2 on control: obsessions subtotal 13. On the compulsion side she rates 3, 3, 2, 2, 2 for washing rituals: compulsions subtotal 12. Y-BOCS total = 25, which places her in the *severe* band (24 to 31) and comfortably above the 16 trial-entry threshold. If a maximally tolerated, adequately long SSRI trial later brought her to 16, that is a 36% reduction, meeting the usual response definition.

### 13.5 The severity bands: subclinical, mild, moderate, severe, extreme

**The five bands to know.** The total score is conventionally read into five **severity bands** (Goodman et al. 1989; conventional Y-BOCS severity ranges). They are: **subclinical** 0 to 7, **mild** 8 to 15, **moderate** 16 to 23, **severe** 24 to 31, and **extreme** 32 to 40. The boundaries are worth over-learning because they are the exact numbers examiners ask for, and because they explain the trial-entry cut-off: a Y-BOCS of 16 is the floor of the *moderate* band, so trials enrolling at 16 and above are, by design, enrolling at least moderately ill patients. The bands are typeset (not reproduced) on the accompanying scale plate.

**How the bands map to decisions.** The bands are not merely descriptive. Mild-to-moderate OCD can reasonably be offered CBT with exposure and response prevention as monotherapy first-line; moderate-to-severe OCD warrants an SSRI, and severe illness warrants a combined SSRI plus

CBT/ERP from the outset (IPS 2025). At the extreme end, surgical candidates for neurosurgery or deep brain stimulation for refractory OCD typically carry Y-BOCS scores of a minimum of 26 to 30 of a possible 40 (Kaplan and Sadock 2024), which sits at the top of the severe band and into the extreme range.

| BAND | Y-BOCS TOTAL (RANGE) | CLINICAL READING |
|---|---|---|
| **Subclinical** | 0 to 7 | Symptoms present but below clinical threshold |
| **Mild** | 8 to 15 | **Below the usual 16 trial-entry cut-off; CBT/ERP monotherapy reasonable** |
| **Moderate** | **16 to 23** | Floor is the 16 drug-trial threshold; SSRI indicated |
| **Severe** | 24 to 31 | Combined SSRI plus CBT/ERP from the outset |
| **Extreme** | 32 to 40 | **Top of scale; refractory-OCD neurosurgery/DBS candidates typically 26 to 30 minimum** |

The five Y-BOCS severity bands and the total-score ranges that define them; note that 16, the usual drug-trial entry threshold, is the floor of the moderate band (Goodman et al. 1989; Kaplan and Sadock 2024).

**GOOD TO REMEMBER**

- **Severity bands:** subclinical 0 to 7; mild 8 to 15; moderate 16 to 23; severe 24 to 31; extreme 32 to 40. The 16 drug-trial cut-off is the floor of the moderate band; typical presenting OCD scores 16 to 30; neurosurgery/DBS candidates for refractory OCD usually score at least 26 to 30 out of a possible 40.

### 13.6 Using the number: defining response and an adequate trial

**Response is a percentage fall on the Y-BOCS.** The Y-BOCS is not only a snapshot; it is how "response" is operationalised in OCD. Across the trial literature, treatment response is most often defined as a reduction of at least **25 to 35%** from the baseline Y-BOCS total (New Oxford Textbook; Kaplan and Sadock 2024), and remission is often set at a low absolute score (for example a CY-BOCS below 11 in paediatric OCD). This is why the scale, rather than a clinical impression, is what tells you whether a trial has genuinely failed. A patient who "feels no better" but has moved from 28 to 19 has in fact responded (a 32% fall) and the SSRI should be continued, not abandoned.

**The adequate-trial anchor.** The single commonest error in OCD is calling a drug a failure too early. OCD needs a *high* SSRI dose held for a *long* trial: continue a maximally tolerated effective SSRI dose for at least **12 weeks** before judging efficacy, having escalated to the effective dose within the first 4 to 6 weeks (IPS 2025), and continue an effective agent for at least 1 to 2 years after remission. Only when the Y-BOCS has failed to fall by the response threshold after that full high-dose, long-duration trial is the patient a true non-responder who moves down the algorithm to clomipramine and then antipsychotic augmentation.

**TRAP** **Do not declare an SSRI failure before a full high-dose, long trial.** OCD demands a higher SSRI dose than depression, held for at least 12 weeks; judge failure by a less-than-25-to-35% fall on the Y-BOCS, not by the patient's subjective sense, before moving to clomipramine or augmentation.

**INDIA**

The highest-yield Indian point is that the Y-BOCS is the objective anchor the system most lacks. The OCD treatment gap in India is wide, OCD is frequently mislabelled (contamination fears dismissed as "habit", scrupulosity read as religiosity), and into that gap flows the same benzodiazepine over-prescription problem that dogs the anxiety disorders, with long-term alprazolam handed out where a high-dose SSRI and exposure and response prevention are indicated. A benzodiazepine has no place in OCD treatment (it does not treat obsessions or compulsions), and the Indian Psychiatric Society CPG (IPS 2025) is explicit: an SSRI at high dose first-line, escitalopram (Nexito, Rexipra), fluoxetine, sertraline or fluvoxamine, with CBT/ERP, then clomipramine (Anafranil) second-line, with the Y-BOCS tracked at baseline and through the trial so the "12 weeks at a high dose" rule is actually enforced rather than assumed.

| | | | |
|---|---|---|---|
| **0 to 40** | **16** | **32 to 40** | **12 weeks** |
| Y-BOCS TOTAL, TEN-ITEM SCALE (EACH ITEM 0 TO 4) | DRUG-TRIAL ENTRY CUT-OFF; FLOOR OF THE MODERATE BAND | EXTREME SEVERITY BAND (TOP OF SCALE) | MINIMUM HIGH-DOSE SSRI TRIAL BEFORE JUDGING FAILURE |

**MNEMONIC**

### TIDRC

The five parameters rated for both obsessions and compulsions: **T**ime occupied, **I**nterference with functioning, **D**istress, **R**esistance, and **C**ontrol. Rate these five for the obsessions (items 1 to 5) and the same five for the compulsions (items 6 to 10); each is 0 to 4, and the ten sum to the 0 to 40 total.

**HOLD THIS**

Ten items, each 0 to 4, total 0 to 40: subclinical 0 to 7, mild 8 to 15, moderate 16 to 23, severe 24 to 31, extreme 32 to 40, with 16 the drug-trial entry cut-off and the floor of the moderate band.

## 14 · The neurobiology of OCD: the CSTC circuit

Obsessive-compulsive disorder has the cleanest circuit story in psychiatry, and examiners love it precisely because it can be drawn as a loop. Where the anxiety disorders are diseases of an amygdala-centred fear circuit, OCD is a disease of a **cortico-striato-thalamic loop** that has lost its normal capacity to switch a thought or action off. The obsession is a message that will not stop repeating; the compulsion is the ritual the loop drives to try to discharge it. The anatomy to name is the **cortico-striato-thalamo-cortical** circuit, the **cstc** loop: from the **orbitofrontal** cortex and the anterior cingulate down to the striatum (the **caudate**), through the globus pallidus and substantia nigra, up to the **thalamus** and back to the cortex. Learn it as a loop with a brake, an

accelerator and a gate, and the imaging findings, the drug rationale and even the neurosurgical targets all fall into place.

**Why it matters clinically.** The imaging signature of OCD is a hyperactive orbitofronto-caudate-thalamic loop, and the two treatments that work, the high-dose serotonin reuptake inhibitor and exposure with response prevention, both turn that hyperactivity down; the neurobiology is therefore the rationale for the treatment, not decoration. Two ideas anchor the whole answer. First, the loop is normally self-limiting because the striatum gates and quietens cortical activity, so a healthy loop lets a thought arise, be checked, and be released. Second, OCD is modelled as an imbalance between a hyperactive *direct* (excitatory, "go") pathway and a relatively weak *indirect* (inhibitory, "stop") pathway through the **basal ganglia ocd**, so the loop reverberates and the thalamus over-drives the orbitofrontal cortex, and the patient cannot let the thought go. The direct and indirect pathway machinery and the receptor systems are developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; this topic applies that basal-ganglia anatomy to OCD and refers to the accompanying figure for the map.

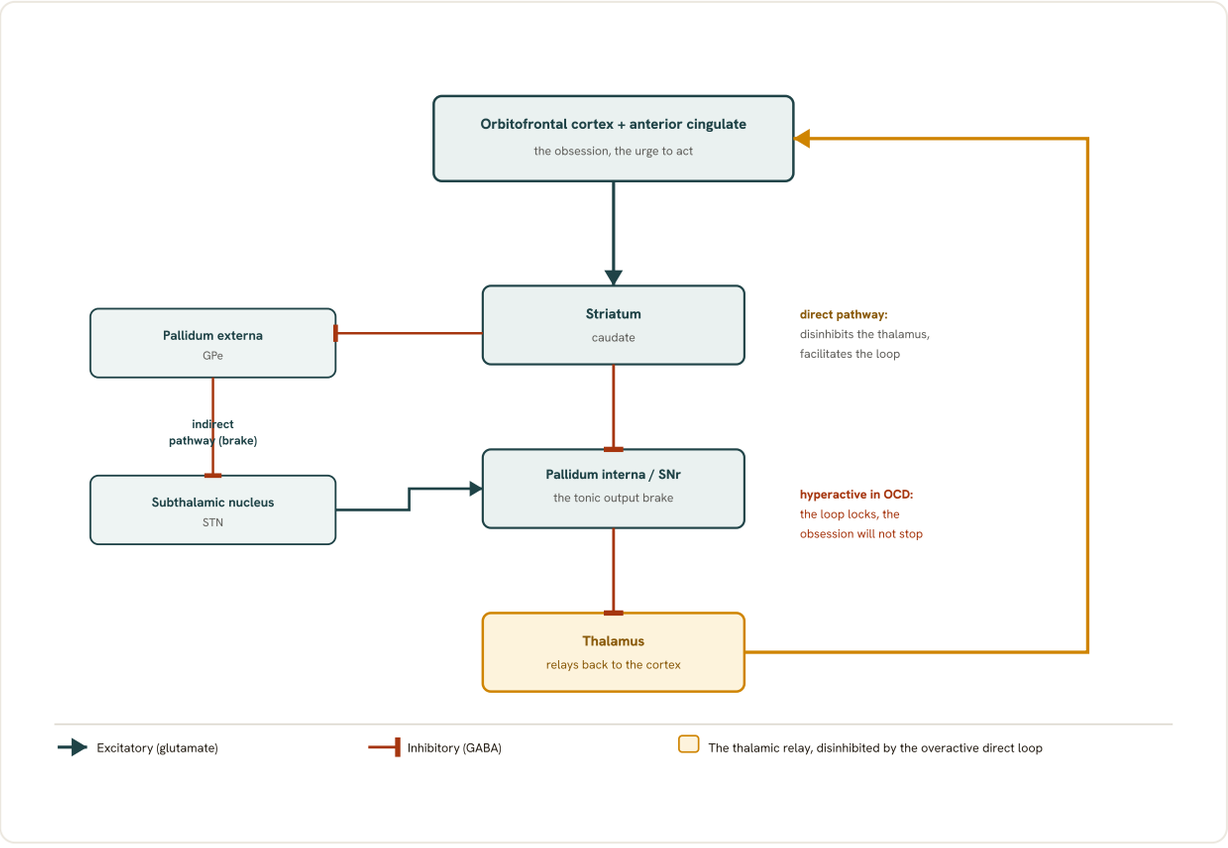



**Fig 14.12** The cortico-striato-thalamo-cortical (CSTC) circuit in OCD. The orbitofrontal cortex and anterior cingulate drive the striatum (caudate); the direct pathway then runs striatum to pallidum interna and substantia nigra reticulata to thalamus, each step GABAergic and inhibitory, so the net effect is to lift the tonic brake on the thalamus and let the cortico-thalamic loop run; the indirect pathway, through the external pallidum and the subthalamic nucleus, is the opposing brake that normally damps it. In OCD the direct loop is overactive relative to the indirect brake, so the loop becomes hyperactive and self-sustaining, the neural correlate of an obsession and a compulsion that will not switch off, and the target that a serotonin reuptake inhibitor, exposure and response prevention, and in the last resort deep brain stimulation all act to quieten. The receptor systems and the wider pathway anatomy are taught in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes.

### 14.1 The loop in one sentence: cortex to striatum to pallidum to thalamus to cortex

**The anatomy to draw first.** The cortico-striato-thalamo-cortical circuit begins in the cortex, projects to the striatum (in OCD, chiefly the head of the caudate nucleus), which projects to the output nuclei of the basal ganglia (the globus pallidus interna and the substantia nigra pars reticulata), which project to the thalamus, which projects back up to the same region of cortex that started the loop (Kaplan & Sadock CTP 2024; Stahl's Essential Psychopharmacology). It is a closed re-entrant loop, and its job is to select and gate behaviour: to let a wanted action or thought proceed and to suppress the unwanted competitors.

**Why the caudate is the pivot.** The striatum is the entry gate of the loop and the caudate is the associative-territory striatum that receives the **orbitofrontal** and cingulate projections; it is the structure whose glutamatergic tone is raised in OCD and whose activity normalises when the patient responds to treatment (Kaplan & Sadock CTP 2024). Naming the caudate as the striatal node of the OCD loop is the single most examined anatomical fact of the topic.

### 14.2 The cortical origin: the orbitofrontal cortex and the anterior cingulate

**The two cortical nodes to name.** The OCD loop originates in two adjacent cortical regions. The **orbitofrontal** cortex (OFC) evaluates the affective and reward significance of stimuli and signals "something is wrong here, act on it"; the anterior cingulate cortex (ACC) is the error- and conflict-monitor that generates the nagging sense that an action is incomplete or a threat unaddressed (Kaplan & Sadock CTP 2024). In OCD both are over-active, so the loop is fed a continuous, false "error, not yet safe, do it again" signal, which is felt as the obsession and drives the checking or washing compulsion.

**The clinical face of orbitofrontal over-activity.** An over-active OFC is why the obsession carries a compelling sense of wrongness or danger that the patient cannot dismiss even when they know it is irrational; the ACC error-signal is why the compulsion never quite satisfies and must be repeated. The heightened error-monitoring is measurable as an exaggerated error-related negativity on EEG, a candidate endophenotype of OCD.

**WORKED EXAMPLE**

A patient leaves the house, then is gripped by the thought that the gas is on and the house will burn. The *orbitofrontal* cortex tags the situation as dangerous and salient; the anterior cingulate fires a persistent "this is not resolved" error-signal; the caudate fails to gate the signal, so it reverberates round the thalamus and back to the cortex. The patient returns and checks the stove. The check briefly quietens the loop, which negatively reinforces it, so the loop fires harder next time. That reverberating orbitofronto-caudate-thalamic circuit, not a real gas leak, is the disease.

### 14.3 The direct pathway: the excitatory "go" loop

**How the accelerator works.** Within the striatum two output pathways compete. The *direct pathway* runs from the striatum straight to the globus pallidus interna and substantia nigra pars reticulata (the basal-ganglia output nuclei); it is populated by excitatory D1 receptors and, by inhibiting those inhibitory output nuclei, it *releases* the thalamus, which then excites the cortex

(Stahl's Essential Psychopharmacology). The net effect is "go": the direct pathway is the accelerator that lets a cortical program, a thought or an action, proceed.

**Why it is the villain in OCD.** A relatively over-active direct pathway keeps the thalamus disinhibited, so it keeps driving the orbitofrontal cortex, and the loop cannot be switched off; the obsessional program runs on and on. The proposed core lesion of OCD is exactly this: an imbalance in which the excitatory direct loop dominates and the inhibitory indirect loop is too weak to counter it.

### 14.4 The indirect pathway: the inhibitory "stop" loop

**How the brake works.** The *indirect pathway* runs from the striatum to the globus pallidus *externa*, then to the subthalamic nucleus, then to the output nuclei; it is populated by inhibitory D2 receptors and its net effect is to *increase* inhibition of the thalamus, which reduces cortical excitation (Stahl's Essential Psychopharmacology). The indirect pathway is the brake that normally suppresses competing or unwanted programs, "stop".

**The balance is the whole model.** In the healthy loop the direct "go" and indirect "stop" pathways are balanced, so the striatum can select the wanted program and cancel the rest, a clean gate. In OCD the balance tips toward the direct pathway: too much "go", too little "stop", so unwanted intrusive programs are not cancelled and the thalamus is left over-driving the cortex. This direct-over-indirect imbalance is the mechanistic heart of the topic.

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RULE **OCD is a hyperactive direct loop with a failing brake.** The imaging and modelling both point one way: an over-active excitatory direct pathway keeps the thalamus disinhibited and the orbitofronto-caudate-thalamic loop reverberating, while the inhibitory indirect pathway is too weak to gate the intrusive program, so the obsession cannot be switched off.

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### 14.5 The output gate and the return to cortex: globus pallidus, substantia nigra and thalamus

**The gate.** The globus pallidus interna and the substantia nigra pars reticulata are the tonically active, inhibitory output gate of the basal ganglia; they normally clamp the thalamus down. The direct pathway *opens* the gate (disinhibits the thalamus) and the indirect pathway *closes* it (Stahl's Essential Psychopharmacology). Reduced globus pallidus volume is among the more replicated structural findings in OCD, consistent with a dysfunctional gate (Kaplan & Sadock CTP 2024).

**The return limb.** The **thalamus** is the relay that closes the loop, projecting back up to the orbitofrontal cortex and cingulate that started it. When the direct pathway holds the gate open, the thalamus over-excites the cortex, the cortex re-drives the striatum, and the circuit becomes a self-sustaining reverberation, which is the circuit-level description of an obsession that will not stop. Treatment-naive children with OCD show enlarged thalamic volumes, fitting an over-active return limb (Kaplan & Sadock CTP 2024).

MNEMONIC

**CSTC = Cortex, Striatum (caudate), pallidum/nigra, Thalamus, back to Cortex**

Trace the loop in order: **C**ortex (orbitofrontal + cingulate) to **S**triatum (the caudate gate) to the pallidum and substantia nigra (the output gate) to the **T**halamus (the relay) and back to **C**ortex. Then overlay the two pathways: *direct* = D1, "go", opens the gate; *indirect* = D2, "stop", closes the gate. OCD tips the balance toward "go".

### 14.6 The imaging signature: a hyperactive orbitofronto-caudate-thalamic loop

**The finding to quote.** Functional imaging in OCD shows increased resting glucose metabolism in the **orbitofrontal** cortex, the ventral striatum, the (right) **caudate** nucleus and the anterior cingulate, at rest and increasing further on symptom provocation (Kaplan & Sadock CTP 2024). Crucially, orbitofrontal and caudate metabolic rates are highly correlated, which is read as the interconnecting loops being "freed from their usual inhibitory influences and reverberating", the imaging translation of the failing brake.

**The treatment proof.** The same imaging validates the circuit as the target: both medication (a serotonin reuptake inhibitor) and behavioural therapy reduce orbitofrontal and caudate activity, and in children the raised caudate glutamate (Glx) falls to control levels after about **12 weeks** of an SSRI, tracking clinical response (Kaplan & Sadock CTP 2024). The circuit is not just correlated with OCD, it is where the treatments act, which is why the neurobiology carries the management rationale.

TRAP **Calling OCD an "amygdala" or "fear-circuit" disorder.** The anxiety disorders sit on the amygdala-centred fear circuit; OCD sits on the cortico-striato-thalamo-cortical loop through the *basal ganglia*, with the caudate, not the amygdala, as the pivot. Mixing the two circuits is the classic error, and it is why OCD is now classed apart from the anxiety disorders in DSM-5-TR and ICD-11.

### 14.7 The serotonin-glutamate chemistry that tunes the loop

**Why serotonin, and why a high dose.** The loop is tuned by serotonin: serotonergic projections from the raphe modulate the orbitofrontal-striatal circuit, and only the serotonergic antidepressants (the SSRIs and the serotonergic tricyclic clomipramine) work in OCD, which is the pharmacological argument for a serotonergic abnormality (Kaplan & Sadock CTP 2024). The clinical corollary is the OCD dosing rule: a *higher* SSRI dose than in depression, sustained for a *longer* trial of about **10 to 12 weeks** before judging response, because the circuit re-tunes slowly.

**The glutamate layer.** The excitatory transmitter of the cortico-striatal projection is glutamate, and raised caudate glutamate (Glx) in OCD is the neurochemical face of the over-active loop; it falls with successful SSRI treatment (Kaplan & Sadock CTP 2024). This glutamatergic model is why refractory OCD is a target for glutamate-modulating agents and, at the extreme, for neurosurgical or stimulation interruption of the loop. Deep brain stimulation and neurosurgery

for refractory OCD, which physically interrupt this circuit at the ventral capsule or cingulate, are developed in the ECT and neurostimulation notes.

**GOOD TO REMEMBER**

- **Serotonin reuptake inhibitor (mechanism and OCD rule):** only the serotonergic agents work in OCD (the SSRIs and clomipramine), the mechanism being a slow re-tuning of the hyperactive orbitofronto-caudate-thalamic loop; the signature principle is a *high* dose for a *long* trial of about **10 to 12 weeks** before declaring failure, and both drug and exposure therapy normalise the caudate hyperactivity on imaging.

### 14.8 Clomipramine, the serotonergic tricyclic that proved the loop

**The historical anchor.** Clomipramine (Anafranil), a non-selective serotonin reuptake-inhibiting tricyclic, was the first drug shown to work in OCD (used by Fernandez and Lopez-Ibor in the late 1960s) and remains the yardstick against which the SSRIs are compared. Its efficacy, tied to its serotonergic action, was the pharmacological evidence that fixed serotonin at the centre of the OCD circuit model.

**Where it sits now.** Clomipramine (75 to 300 mg/day) is at least as effective as the SSRIs and by some meta-analyses numerically more so, but its anticholinergic, cardiac and seizure burden means the better-tolerated SSRI is first-line and clomipramine is held for SSRI non-response; the response is better when depression coexists. The detailed antidepressant pharmacology, the SSRIs, SNRIs and clomipramine, their doses, adverse effects and interactions, is developed in the Mood disorders: pharmacotherapy notes.

**GOOD TO REMEMBER**

- **Clomipramine (mechanism and role):** a non-selective serotonin reuptake-inhibiting tricyclic, the *first* effective OCD drug and the proof that OCD is a serotonergic circuit disorder; the signature caution is its anticholinergic, cardiotoxic and seizure-lowering load, so despite efficacy equal to or better than the SSRIs it is second-line, reserved for SSRI non-response (typical range 75 to 300 mg/day).

### 14.9 Measuring the loop clinically: the Y-BOCS

**What the scale is.** The Y-BOCS (Yale-Brown Obsessive-Compulsive Scale), developed by Goodman and Charney in 1989, is the clinician-administered gold-standard severity measure for OCD; it rates obsessions and compulsions across five dimensions each (time, interference, distress, resistance and control), on a 0 to 4 anchor, for a total of **0 to 40** (Clinical Methods in Psychiatry, AIIMS; Tasman Psychiatry). It is the instrument used to define response (conventionally a 25 to 35 percent fall) and remission (a score of about **10 or less**) in the OCD trials. The instrument form itself is reproduced on the accompanying scale plate.

**The severity bands to hold.** The Goodman severity anchors are: 0 to 7 subclinical, 8 to 15 mild, 16 to 23 moderate, 24 to 31 severe, and 32 to 40 extreme; a total of 16 and above is the usual threshold for a clinical trial and for committing to pharmacotherapy. Anchoring "moderate OCD" to a Y-BOCS of 16 to 23 is the number examiners most often want.

| 0 to 40 | 16 to 23 | 10 to 12 | 75 to 300 |
|---|---|---|---|
| Y-BOCS TOTAL RANGE (10 ITEMS) | Y-BOCS MODERATE BAND | WEEKS: ADEQUATE SSRI TRIAL IN OCD | CLOMIPRAMINE (MG/DAY) |

**INDIA**

OCD in India carries a large treatment gap: the disorder is common, chronically under-recognised and, when it does present, often mislabelled and mistreated. A recurring Indian pattern is the reflex prescription of a *benzodiazepine* for the anxiety and distress of OCD, which sedates the patient but does nothing to the serotonergic loop and courts dependence; the benzodiazepine is not a treatment for OCD. The Indian Psychiatric Society Clinical Practice Guidelines for OCD frame the correct sequence, a high-dose serotonin reuptake inhibitor for an adequate trial, then clomipramine, then antipsychotic augmentation, with exposure and response prevention throughout, and the SSRIs and clomipramine (Anafranil) are all widely available as inexpensive Indian generics. The gap is not a drug-availability problem, it is a recognition-and-rational-prescribing problem.

**HOLD THIS**

OCD is a hyperactive cortico-striato-thalamo-cortical loop: an over-active orbitofrontal cortex and anterior cingulate drive a caudate that cannot gate them, so an excess of the excitatory direct ("go") pathway over the inhibitory indirect ("stop") pathway leaves the thalamus disinhibited and the loop reverberating, and the high-dose serotonin reuptake inhibitor and exposure therapy both work by turning that orbitofronto-caudate hyperactivity back down.

## 15 · Clomipramine versus SSRIs in OCD

This is the single most reliably examined drug comparison in the whole anxiety-spectrum syllabus, because it hides a trap in plain sight. The naive candidate reasons that if clomipramine is *marginally more efficacious* on meta-analysis it ought to be first-line; the examiner is testing whether you understand that a first-line recommendation is a decision about the *whole* risk-benefit balance, not about the point estimate of efficacy alone. The **clomipramine versus ssri** question turns on one sentence you should be able to deliver cold: the two are comparably effective (clomipramine perhaps a whisker ahead), but clomipramine is far less tolerable and more dangerous, so the SSRIs win first-line on tolerability and safety.

**Why it matters, and what this note owns.** This note owns the head-to-head: the serotonergic-specificity argument that explains why clomipramine (alone among tricyclics) works in OCD at all, the meta-analytic evidence on relative **efficacy in ocd**, the **tolerability ocd** reasons clomipramine loses first-line, and exactly where it sits on the ladder. The wider four-rung OCD algorithm (high-dose SSRI, long trial, then augmentation, then refractory options) is set out in the OCD management algorithm note and only referenced here; the deep antidepressant pharmacology of the SSRIs and clomipramine, their kinetics, adverse effects and interactions, is developed in the Mood disorders: pharmacotherapy notes. The comparison itself is laid out in the accompanying figure.

### 15.1 The serotonergic-specificity argument: why clomipramine works when other TCAs do not

**The founding observation.** Clomipramine was the *first* drug shown to work in OCD, in studies from the late 1960s onward, and this is not incidental trivia: it is the pharmacological clue that shaped the whole serotonin hypothesis of OCD. Clomipramine is the one tricyclic that is a *potent serotonin reuptake inhibitor*; the Companion notes it was the first drug to inhibit serotonergic uptake without primarily inhibiting noradrenergic reuptake (its active metabolite desmethylclomipramine is the noradrenergic one). Conventional tricyclics, with the exception of clomipramine, are *not* effective in OCD (Shorter Oxford). That single dissociation is the argument.

**The desipramine experiment that clinches it.** The clean demonstration is the head-to-head against a noradrenergic TCA: clomipramine (serotonergic) beats desipramine (noradrenergic) in OCD, and the same pattern of serotonergic-reuptake inhibitors outperforming non-serotonergic agents recurs across the obsessive-compulsive spectrum (New Oxford Textbook). The take-home is a mechanism rule, only drugs that potently block the serotonin transporter, whether the SSRIs or clomipramine, are anti-obsessional. The amygdala and cortico-striatal fear-circuit substrate behind this is in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes.

RULE **Serotonergic potency, not the tricyclic class, is what treats OCD.** Clomipramine works because it potently blocks the serotonin transporter; noradrenergic tricyclics such as desipramine do not, which is why clomipramine and the SSRIs are the only anti-obsessional agents.

### 15.2 The efficacy evidence: clomipramine marginally ahead, but not significantly

**What the meta-analyses actually show.** On placebo-referenced **meta-analysis ocd** data, both clomipramine and all SSRIs are efficacious; the SSRIs are roughly twice as likely as placebo to produce a clinical response (the Soomro meta-analysis, 17 adult studies, 3097 participants; New Oxford Textbook). The nuance the examiner wants is the *relative* comparison. The Companion states it plainly: in meta-analyses clomipramine is *marginally more efficacious* than the SSRIs, but the SSRIs are somewhat better tolerated. BAP frames the same finding as clomipramine sitting *at the margins of superiority* over SSRIs while being less well tolerated (BAP 2014).

**The statistical caveat, and the confound.** The apparent clomipramine edge is fragile. The Skapinakis network meta-analysis, combining direct and indirect evidence, found clomipramine had a larger effect size against placebo than the SSRIs, but *this difference was not statistically significant*, and individual SSRIs produced similar effect sizes with no single compound superior (New Oxford Textbook). The classic explanation for the apparent gap is a confound of era: the older clomipramine trials enrolled more severe, more treatment-naive samples and used indirect (cross-study) rather than head-to-head comparison, inflating its relative effect. When the evidence is pooled properly the two are, for practical purposes, equally effective.

| DIMENSION | CLOMIPRAMINE | SSRIS (CLASS) |
|---|---|---|
| **Serotonergic mechanism** | Potent SERT block (most serotonergic TCA) | Selective, potent SERT block |
| **Placebo-referenced efficacy** | **Marginally higher effect size, not statistically significant** | Robustly efficacious, no single SSRI superior |
| **Tolerability** | **Poorer (anticholinergic, sedation, weight)** | Better tolerated |
| **Safety in overdose** | Cardiotoxic, seizure risk, dangerous | Relatively safe |
| **Line of treatment** | Second-line / switch after SSRI | **First-line** |

The clomipramine-versus-SSRI head-to-head: comparable efficacy (clomipramine perhaps marginally ahead but not significantly), decisively worse tolerability and overdose safety, hence SSRIs first-line and clomipramine reserved (Companion; New Oxford; BAP 2014).

### 15.3 The tolerability and safety cost: why the marginal edge does not buy first-line

**The three dangers.** Clomipramine carries the full tricyclic liability that the SSRIs largely shed. First, the *anticholinergic* burden, dry mouth, constipation, urinary retention, blurred vision, sedation and weight gain, which drives dropout. Second, *cardiac* effects, QTc prolongation and slowed intracardiac conduction, so an ECG is checked before and during treatment and it is avoided after recent myocardial infarction or with uncorrected hypokalaemia (Stahl; NICE 2005). Third, the *seizure* risk, which is dose-dependent and the reason the ceiling is fixed: at 300 mg/day the seizure incidence approaches **7 per 1000**, a generally unacceptable risk, so the maximum is held at **250 mg/day** (Stahl; New Oxford Textbook). Clomipramine is also markedly more dangerous in overdose than any SSRI.

**Why this decides the line.** When two drugs are near-identical in efficacy, the tie is broken by tolerability and safety, and here the gap is not marginal at all. That is the entire logic of first-line: the SSRIs deliver essentially the same benefit at a fraction of the harm, so they are tried first and clomipramine is held in reserve for when an SSRI has failed or cannot be tolerated (BAP 2014; NICE 2005).

TRAP **"More efficacious, therefore first-line" is the wrong inference.** Clomipramine's meta-analytic efficacy edge is marginal and non-significant, and it is bought at a heavy tolerability and overdose-safety price, so an SSRI is first-line and clomipramine is the switch, never the opening move.

### 15.4 Where clomipramine sits on the ladder: the switch after an adequate SSRI trial

**The BAP position, first.** Lead with the British Association for Psychopharmacology: offer an SSRI as first-line pharmacological treatment for OCD, since all SSRIs and clomipramine have efficacy but SSRIs are generally better tolerated; clomipramine is an evidence-based acute treatment whose efficacy is at the margins of superiority over SSRIs but which is less well tolerated (BAP 2014). Clomipramine is therefore the drug you switch to, not the drug you start with, and the switch is made after an adequate first SSRI trial fails or is not tolerated.

**What the other guidelines recommend, at guideline level.** The convergence is total. The Indian Psychiatric Society places clomipramine explicitly as a *second-line* agent, used at 150 to 225 mg/day when SSRIs are ineffective, given its poorer tolerability (the Indian Psychiatric Society 2025). NICE recommends offering clomipramine after an adequate SSRI trial has been ineffective or poorly tolerated, or if the patient prefers it or previously responded, checking an ECG and blood pressure first where there is significant cardiovascular risk (NICE 2005, the CG31 OCD guideline). CANMAT lists clomipramine (with citalopram, mirtazapine or venlafaxine XR) as *second-line* pharmacotherapy for OCD (CANMAT 2014). The WFSBP likewise keeps an SSRI first-line and reserves clomipramine as second-line when first-line treatment is inadequate (WFSBP 2022). Every source says the same thing, SSRI first, clomipramine as the reserve.

| GUIDELINE | CLOMIPRAMINE'S PLACE | DOSE (MG/DAY) / NOTE |
|---|---|---|
| **BAP 2014** | **Evidence-based, but second-line (less tolerated)** | marginal efficacy edge, offered as switch |
| **the Indian Psychiatric Society 2025** | **Second-line when SSRIs ineffective** | 150 to 225 |
| **NICE 2005 (CG31)** | After adequate SSRI ineffective / not tolerated | ECG + BP first if cardiovascular risk |
| **CANMAT 2014** | Second-line pharmacotherapy | with citalopram, mirtazapine, venlafaxine XR |

Clomipramine is uniformly second-line for OCD across BAP, the Indian Psychiatric Society, NICE, CANMAT and WFSBP, reached as the switch after an adequate first-line SSRI trial fails; the OCD dose is 150 to 225 mg/day with ECG monitoring.

### 15.5 The dose and monitoring: 150 to 225 mg/day, ECG, and the 250 mg ceiling

**The numbers to quote.** The OCD dose of clomipramine is **150 to 225 mg/day** (starting from 75 mg and titrating), and the maximum recommended dose of **250 mg/day** should not usually be exceeded because of dose-related cardiotoxicity and seizures (the Indian Psychiatric Society 2025; New Oxford Textbook). Patients treated for OCD often need the high end of the wider dosing range, 200 to 250 mg/day, still under the 250 mg ceiling (Stahl). ECG monitoring is required before and during treatment, and blood pressure is checked (the Indian Psychiatric Society 2017; NICE 2005).

**The interaction that catches people out.** An SSRI switch to clomipramine is not always clean, because fluvoxamine (a CYP1A2 inhibitor) and fluoxetine/paroxetine (CYP2D6 inhibitors) raise clomipramine levels, so an unwary cross-taper can push a "standard" clomipramine dose into toxic, pro-convulsant, cardiotoxic territory (Stahl; the Indian Psychiatric Society 2019). Combining an SSRI with clomipramine also risks serotonin syndrome. The full interaction map is in the Mood disorders: pharmacotherapy notes.

| | | |
|---|---|---|
| **150 to 225** | **250** | **7 per 1000** |
| CLOMIPRAMINE OCD DOSE (MG PER DAY) | MAXIMUM DOSE CEILING, MG PER DAY (SEIZURE / CARDIAC RISK) | SEIZURE INCIDENCE AT 300 MG PER DAY (UNACCEPTABLE) |

**WORKED EXAMPLE**

A 34-year-old woman with checking compulsions has failed an adequate trial of sertraline pushed to 200 mg/day for 12 weeks and a second adequate trial of fluoxetine 80 mg/day, both with real ERP, without response. Rather than reach for clomipramine first (its efficacy edge is marginal and non-significant), the switch is now justified precisely because two first-line SSRI trials have genuinely failed. She is started on clomipramine 75 mg, titrated toward 150 to 225 mg/day, with a baseline ECG and blood pressure and repeat ECG on dose increases. Because her prior fluoxetine is a CYP2D6 inhibitor with a long half-life, the clomipramine is introduced cautiously to avoid a levels spike into cardiotoxic or pro-convulsant range.

### 15.6 The whole comparison in one line, and the India lens

**The one-line answer.** If asked to compare in a sentence: clomipramine and the SSRIs are comparably effective anti-obsessional agents (clomipramine perhaps marginally more efficacious on meta-analysis, but not significantly so), and because clomipramine is less well tolerated and more dangerous in overdose, the SSRIs are first-line on the tolerability-and-safety balance, with clomipramine reserved as second-line for the patient in whom an adequate SSRI trial has failed. Everything above is scaffolding for that sentence.

**INDIA**

The comparison is not academic in India, where the anxiety and OCD treatment gap is wide, recognition is patchy, and the reflex prescription is too often a benzodiazepine, which does nothing for obsessions and risks dependence (the benzodiazepine over-prescription problem in Indian outpatient practice is discussed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes). Both drug classes are cheap and available: the SSRIs as fluoxetine (Indian brands Fludac, Prodep), sertraline (Daxid, Zosert) and escitalopram (Nexito), and clomipramine as Anafranil. The Indian Psychiatric Society CPGs, updated for OCD to 2025, anchor exactly the SSRI-first, clomipramine-second-line logic to Indian practice, so the practical barrier is under-recognition and timid dosing, not access.

**HOLD THIS**

Clomipramine (most serotonergic TCA) and the SSRIs are comparably effective in OCD, with clomipramine only marginally and non-significantly ahead on meta-analysis; because clomipramine is less tolerable (anticholinergic, cardiac, seizure-risk) and dangerous in overdose, the SSRIs are first-line and clomipramine is the second-line switch after an adequate SSRI trial fails, dosed 150 to 225 mg/day with ECG monitoring.

**MNEMONIC**

**SAME PUNCH, WORSE SIDES**

Clomipramine and the SSRIs land the **same anti-obsessional punch** (equal-ish efficacy, clomipramine a marginal, non-significant nudge ahead), but clomipramine has the **worse side effects and worse overdose safety**, so tolerability, not efficacy, decides the line and the SSRIs go first.

**GOOD TO REMEMBER**

- **Clomipramine (Indian brand Anafranil):** the most serotonergic tricyclic and the original anti-obsessional drug (its serotonergic potency, not its tricyclic class, is why it works); mechanism is potent serotonin reuptake inhibition; marginally, non-significantly more efficacious than SSRIs on meta-analysis yet second-line in OCD at 150 to 225 mg/day because it is less tolerable (anticholinergic, cardiac conduction, seizure above 250 mg/day) and dangerous in overdose, so it is the switch after an adequate SSRI trial fails, under ECG monitoring.
- **SSRIs (first-line for OCD):** selective potent serotonin reuptake inhibitors; robustly efficacious with no single SSRI superior; the signature reason they beat clomipramine to first-line is superior tolerability and overdose safety at comparable efficacy; the one first-line principle is a high-dose SSRI plus ERP before any switch to clomipramine.

**PEARL**

The whole topic is a worked example of a general prescribing principle: when two drugs are equally effective, the first-line choice is decided by tolerability and safety, not by a marginal, non-significant efficacy point estimate. State that principle and the clomipramine-versus-SSRI answer writes itself.

## 16 · Exposure and response prevention

**Exposure and response prevention** (**erp**) is the specific behavioural treatment for obsessive-compulsive disorder, and it is one of the highest-yield psychotherapy answers in the whole anxiety block. Where the wider exposure principle and the general CBT model belong to the Psychotherapies, clinical assessment, MSE and nosology notes, this note owns the disorder-specific machinery: how the graded **exposure hierarchy** is built, what the **response prevention** component actually forbids, why anxiety falls on repeated exposure without the ritual, and the evidence base and delivery. Every major guideline, BAP 2014, NICE 2005 and the Indian Psychiatric Society CPG updated to 2025, names CBT incorporating ERP as first-line psychotherapy for OCD, co-equal with a high-dose SSRI and superior at holding gains.

**Why it matters, and how to answer it.** ERP is not "exposure with some talking"; it is a precise procedure with two named halves and a named mechanism. The candidate who scores states the mechanism (**habituation** of anxiety on prolonged, repeated exposure to a feared cue while the neutralising ritual is blocked), names both components (graded exposure plus **ritual prevention**), and knows the line placement (first-line, roughly as effective as an SSRI acutely and better at preventing relapse). Hold one sentence above all others: ERP works because staying with the anxiety, and refusing the compulsion that would abort it, lets the patient learn by direct experience that the feared catastrophe does not occur and that the anxiety subsides on its own.

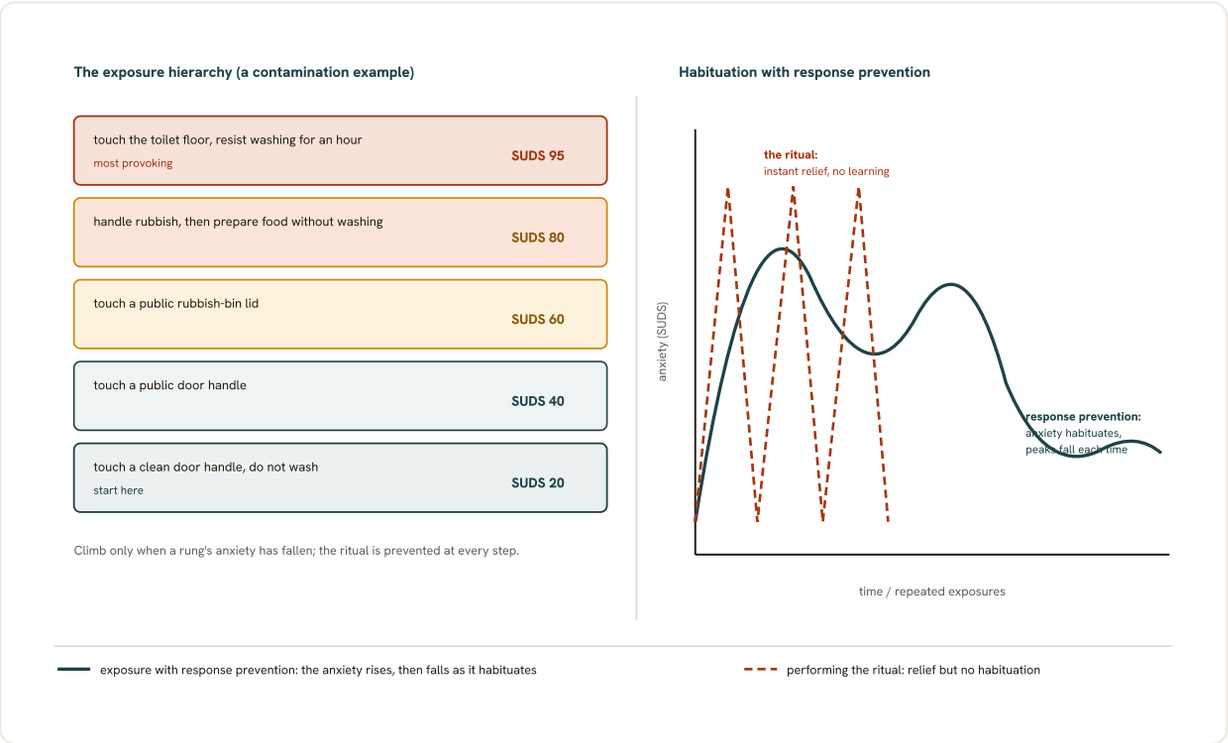



**Fig 14.13** Exposure and response prevention (ERP). The patient and therapist build a hierarchy of feared situations, each rated for anxiety on a 0 to 100 subjective-units-of-distress scale, and work up it from the least to the most provoking. At each rung the patient stays in the situation and the compulsion is prevented; the anxiety rises, plateaus and then falls of its own accord as the body habituates, and the peak is lower on each repetition. Performing the ritual instead gives instant relief but teaches nothing, so the fear re-spikes to the same height every time. Preventing the response is therefore the active ingredient, and the wider exposure technique is taught in the Psychotherapies, clinical assessment, MSE and nosology notes.

## 16.1 What ERP is: two components with one mechanism

**The definition.** Exposure and response prevention is a manualised cognitive-behavioural treatment in which the patient is deliberately and repeatedly brought into contact with the situations, objects, thoughts or images that trigger obsessional distress (*exposure*), while refraining from the compulsion or avoidance that would normally relieve that distress (*response prevention*, also called ritual prevention) (Kaplan and Sadock 2024; Foa, Yadin and Lichner). Foa's canonical protocol, marketed as EX/RP, has five working parts: in-vivo exposure, imaginal exposure, ritual prevention, processing of the experience, and home practice. The exposure and the response prevention are not optional extras of each other; exposure without ritual prevention lets the patient neutralise and learn nothing, and ritual prevention without exposure gives the anxiety nothing to habituate to.

### Exposure

Deliberate, planned contact with a feared cue (a situation, object, thought or image) sufficient to raise obsessional anxiety.

### Response prevention

Blocking the compulsive ritual and the covert neutralising or avoidance that would otherwise discharge the anxiety.

### Habituation

The automatic decline of an emotional response on repeated, prolonged exposure to the same stimulus once the ritual no longer aborts it.

### 16.2 The mechanism: habituation of anxiety when the ritual is blocked

**The vicious cycle ERP breaks.** In OCD an obsession provokes anxiety, the compulsion (or avoidance) discharges that anxiety, and the relief negatively reinforces the ritual, so the loop tightens with every repetition. Because the ritual is performed *before* the anxiety could fall by itself, the patient never learns that it would have fallen anyway, and never learns that the dreaded outcome would not have happened. Habituation is the principle that irrational fear and its behaviours fade on repeated and prolonged exposure to the source of the fear (Reddy, NIMHANS patient guide). ERP engineers exactly this: by exposing the patient to the trigger and forbidding the ritual, the anxiety is allowed to rise and then, if the patient stays with it, to subside on its own; with repetition the trigger progressively loses its power to provoke distress.

**What the patient must not do.** The habituation curve is fragile. Substituting one compulsion for another, using a distraction or relaxation trick to blunt the anxiety, or escaping the situation early all abort the exposure before habituation can occur, so the belief survives intact (Reddy). The instruction is to stay with the anxiety until it comes down substantially. Rituals and compulsions typically settle first; the obsessions themselves take longer to "wear out".

RULE **Block the ritual, or nothing changes.** Exposure only works if the neutralising response is prevented, because habituation and the disconfirmation of the feared outcome both require the patient to stay in the anxiety without the compulsion aborting it.

### 16.3 The exposure hierarchy: least to most anxiety-provoking

**How it is built.** Treatment begins by listing every situation, object, thought and image that triggers the patient's obsessions and rituals, plus everything the patient avoids to keep rituals at bay, and then arranging them in a graded hierarchy from the least to the most anxiety-provoking (Reddy; Kaplan and Sadock 2024). Each item is rated by the patient for the distress it provokes, conventionally on a subjective-units-of-distress scale, so the ladder is calibrated to that individual. The accompanying figure sets out this exposure-hierarchy ladder: low-distress rungs at the bottom, the most feared cue at the top, with the patient climbing only when the current rung has habituated. Exposure proceeds up the ladder, starting where anxiety is tolerable and moving to a harder item only once the easier one has stopped provoking much distress.

**Graded, not flooding.** The "graded" quality is deliberate and is what makes ERP tolerable enough to complete. Both in-vivo exposure (real contact, for example touching a "contaminated" surface and not washing) and imaginal exposure (vividly confronting the feared consequence, for example that harm will befall a loved one) are worked up the same hierarchy. This is the sense in which ERP is disorder-specific exposure: the ladder is composed of the patient's own obsessional cues and the response-prevention rule is attached to every rung.

### 16.4 Response (ritual) prevention: the discipline that makes exposure count

**What is prevented.** Ritual prevention bans the overt compulsion (the washing, checking, counting, reassurance-seeking) and, just as importantly, the covert neutralising and the mental rituals that patients substitute when the overt act is blocked. It also targets the avoidance that keeps triggers out of reach in the first place, for instance avoiding public toilets so as never to

have to wash extensively (Reddy). Where obsessions and compulsions occur only at home, treatment must become home-based, with the family briefed so they neither perform the ritual for the patient nor supply reassurance.

**Family accommodation.** A family that carries out rituals for the patient or endlessly reassures is accommodating the OCD and directly undermining response prevention; high family accommodation predicts poorer outcome, and reducing it through structured psychoeducation is part of the work (IPS 2025). A willing family member can be trained as a co-therapist to support graded exposure and ritual prevention at home.

TRAP **The hidden ritual.** A patient who stops washing but silently counts, prays or seeks reassurance instead has substituted a covert ritual for an overt one; habituation will not occur, and the therapist who misses it wrongly concludes ERP has failed.

### 16.5 Evidence base and effectiveness

**How well it works.** ERP is the most effective and most widely used behaviour therapy for OCD, effective in roughly **60 to 70%** of patients who engage with it (Reddy, NIMHANS). Guideline-level evidence places CBT with ERP as first-line psychotherapy, of broadly similar acute efficacy to an SSRI for mild-to-moderate OCD and superior in maintaining gains over time (BAP 2014; IPS 2025). A rigorous adult trial found that ERP augmentation of an ongoing SSRI outperformed risperidone augmentation, which is a favourite exam point: when an SSRI response is partial, adding ERP is a first-line augmentation strategy, ahead of an antipsychotic (Kaplan and Sadock 2024; IPS 2025). Combined ERP plus a high-dose SSRI is the recommended package for moderate-to-severe OCD, while ERP monotherapy suffices for milder illness.

**Its limits.** ERP demands motivation and the willingness to tolerate deliberate distress, and there can be significant resistance to undertaking it; a skilled therapist is needed where self-help does not suffice (Kaplan and Sadock 2024). Poor insight, entrenched family accommodation and comorbid depression all blunt response. Adequate "dose and duration" of exposure matter exactly as they do for a drug: too little exposure, like too low an SSRI dose, is a common reason for apparent failure.

| 60 to 70 | 10 to 12 | 1 to 2 |
|---|---|---|
| PERCENT RESPOND TO ERP (NIMHANS) | HOURS OF ERP FOR MOST TO RESPOND | HOURS A SINGLE PROLONGED EXPOSURE MAY NEED |

### 16.6 Delivery: dose, duration and format

**How much is enough.** Habituation takes time within a session and across the course. A single exposure may need to run long enough for anxiety to fall, sometimes only **30 minutes** but often a prolonged **1 to 2 hours** in more resistant fears; most patients show response between **10 to 12 hours** of exposure and response prevention overall (Reddy). Foa's protocol delivers this across roughly 17 to 20 sessions combining therapist-supervised and self-directed home practice. Daily home practice is the engine of change: ERP done only in the clinic, without between-session exposure, tends to stall.

**Formats and access.** ERP can be delivered face-to-face, in low-intensity or guided-self-help form for milder OCD (up to about 10 therapist hours per NICE 2005), by group or telephone CBT, and increasingly through internet-delivered programmes, though therapist-led ERP outperforms computerised CBT. Response is objectified by tracking OCD symptom severity on the Yale-Brown Obsessive-Compulsive Scale, whose severity bands and trial-entry threshold are taught, and the instrument reproduced, on the accompanying scale plate in the Y-BOCS note.

**WORKED EXAMPLE**

A young man with contamination obsessions washes for hours and avoids all public surfaces. Therapist and patient list his triggers and rank them from least to most distressing: touching his own front door (low), a shared office keyboard (mid), a hospital door handle (high), a public-toilet flush (top). Starting at the bottom, he touches the door and, under response prevention, does not wash; his anxiety climbs, then falls over the next hour as he stays with it. After several repetitions the door provokes almost nothing, and he climbs to the next rung. The covert urge to "wash later" is named early and blocked, so habituation, not a delayed ritual, is doing the work.

**COMMON TRAP**

Calling ERP off too soon. Like an under-dosed SSRI trial, an ERP course that is too short, too shallow up the hierarchy, or done only in sessions without daily home practice will look like non-response. Before concluding ERP has failed, confirm the exposures were adequate in intensity and duration, that no covert ritual or reassurance was smuggled in, and that family accommodation was addressed.

**INDIA**

Some of the most usable ERP material for Indian patients is Indian: the NIMHANS OCD patient guide (Reddy) lays out the hierarchy, the habituation rationale and the 60-to-70% figure in plain language, and the IPS CPG (updated for OCD to 2025) anchors CBT-with-ERP as first-line psychotherapy for Indian practice. This matters because OCD sits inside India's wide anxiety-spectrum treatment gap, and the reflex prescription of a benzodiazepine, still over-prescribed across Indian outpatient care, does nothing for obsessions: it neither provides exposure nor allows habituation, and it can blunt the very anxiety ERP needs the patient to feel. Where medication is combined with ERP the Indian-brand SSRIs are freely available, fluoxetine (Fludac, Prodep), sertraline (Daxid, Zosert), fluvoxamine (Fluvoxin) and escitalopram (Nexito), so the limiting factor is trained delivery of ERP, not drug access.

**MNEMONIC**

### EXPOSE → PREVENT → STAY → HABITUATE

**EXPOSE** to the feared cue, up a graded hierarchy → **PREVENT** the ritual and the covert neutralising → **STAY** with the anxiety instead of escaping → **HABITUATE**, as the anxiety falls on its own and the trigger loses its power.

**HOLD THIS**

ERP works by blocking the ritual so the patient stays in the anxiety long enough to habituate and to learn the feared catastrophe never comes: two components (graded exposure plus response prevention), one mechanism (habituation), first-line for OCD.

**GOOD TO REMEMBER**

- **Exposure and response prevention:** the disorder-specific behaviour therapy for OCD, combining graded exposure up a patient-built hierarchy (in-vivo and imaginal) with response (ritual) prevention that blocks both overt and covert neutralising; the mechanism is habituation of anxiety once the ritual can no longer abort it, with disconfirmation of the feared outcome; first-line psychotherapy, roughly as effective as a high-dose SSRI acutely and better at maintaining gains, effective in about 60 to 70% of engaged patients, and the preferred first-line augmentation (ahead of an antipsychotic) for a partial SSRI response; the signature caution is that a hidden covert ritual, too-short exposure, or family accommodation will abort habituation and mimic non-response.

## 17 · Treatment-resistant OCD

Roughly a third to a half of OCD patients fail to respond adequately to a first serotonergic drug, and the examiner uses this fact to test one thing: can you tell a genuinely refractory patient from an *under-treated* one. The commonest cause of apparent non-response is not biology but an inadequate first trial, a depression-range dose stopped too soon and delivered without exposure and response prevention. **treatment-resistant ocd** is therefore a diagnosis of exclusion that you earn only after confirming an adequate trial, and its ladder then runs through **antipsychotic augmentation**, combined ERP, and finally the somatic options of neuromodulation and **neurosurgery ocd**.

**Why it matters, and what this note owns.** This note owns the resistance ladder above first-line: what counts as an adequate trial, how augmentation is sequenced, and where the neuromodulation and ablative options sit. The full first-line algorithm (the high-dose SSRI, the long trial, the Y-BOCS thresholds, clomipramine as the switch) is developed in the OCD management notes and is not re-derived here; the SSRI and clomipramine pharmacology lives in the Mood disorders: pharmacotherapy notes; the general exposure principle behind ERP lives in the Psychotherapies, clinical assessment, MSE and nosology notes; and the technical detail of **deep brain stimulation** and ablative neurosurgery (electrode targets, capsulotomy, cingulotomy technique) is cross-referenced to the ECT and neurostimulation notes. The refractory ladder is set out in the accompanying figure.

### 17.1 Confirm an adequate trial before you call it resistant

**Adequate means high dose, long duration, real adherence.** Before anything is added or switched, confirm the failed trial was genuinely adequate on three axes. A *high dose*, pushed to the maximum tolerated (SSRI target roughly **60 to 80 mg** fluoxetine-equivalent, above the depression range). A *long duration*, at least **10 to 12 weeks** at that ceiling before the trial is judged failed, with escalation to the effective dose within about 4 to 6 weeks (IPS 2025). And *adherence*, that the patient actually took the drug and, critically, received exposure and response prevention rather than a drug alone. The New Oxford Textbook frames the failure point as under

a 25% reduction of baseline Y-BOCS after 12 weeks of SRI treatment, of which at least 6 weeks are at the maximal tolerated dose.

**The formal definition.** True resistance is failure of at least two adequate SSRI trials, an adequate clomipramine trial, and CBT with ERP, each at full dose and duration (the Indian Psychiatric Society 2017). The honest first question at every rung is whether the earlier steps were real.

**HOLD THIS**

Do not call OCD treatment-resistant until you have confirmed an adequate trial on all three axes: a high dose (60 to 80 mg fluoxetine-equivalent), a long duration (10 to 12 weeks at the maximum tolerated dose), and genuine adherence including ERP; most apparent non-response is an under-treated first trial, not true resistance.

**RULE** **Adequate trial first, ladder second.** The first move in a "non-responder" is to re-audit the failed trial for dose, duration and adherence, not to reach for the next drug.

### 17.2 Optimise and switch: the steps that precede augmentation

**Optimise, then switch.** Once the trial is confirmed adequate but the response is partial or absent, the sequence before augmentation is to optimise the dose to the maximum tolerated, then switch, either to a different SSRI or to clomipramine (BAP 2014; NICE 2005). Switching between treatments of proven efficacy helps some patients (BAP 2014). Clomipramine is the second-line agent, at **150 to 225 mg/day** under ECG monitoring, used because it is the most serotonergic tricyclic but less well tolerated than an SSRI (the Indian Psychiatric Society 2025). The clomipramine-versus-SSRI contrast is developed in the clomipramine notes; here it is simply the switch on the way up the ladder.

**COMMON TRAP**

Jumping to augmentation or neurosurgery while the earlier rungs were sub-therapeutic. A patient who "failed" fluoxetine 20 mg for 6 weeks without ERP has not failed OCD treatment at all; augmenting that with an antipsychotic exposes them to metabolic risk for no reason and mislabels a slow-but-real responder as refractory.

### 17.3 Antipsychotic augmentation: the best-evidenced next rung

**What it is and the doses.** For the confirmed partial responder to an adequate SSRI or clomipramine trial, **antipsychotic augmentation** means adding a low-dose antipsychotic to the continuing serotonergic drug. BAP has the strongest augmentation evidence for risperidone, with aripiprazole also supported (BAP 2014). The Indian Psychiatric Society names low-dose aripiprazole or risperidone as the first-line pharmacological augmentation in treatment-resistant OCD, continued for at least **8 weeks**, at risperidone **1 to 3 mg/day** and aripiprazole in the low range (the Indian Psychiatric Society 2025). CANMAT lists adjunctive aripiprazole or risperidone as the first-line adjuncts, reserved for inadequate SSRI response given tolerability concerns (CANMAT 2014). WFSBP likewise puts antipsychotic augmentation first among the pharmacological options for resistant OCD (WFSBP 2023).

**The monotherapy caveat and the serotonergic alternatives.** Antipsychotics are for augmentation only: NICE is explicit that they should not normally be used as monotherapy for OCD, and reserves augmentation for after failed SSRI, combined SSRI-plus-CBT and clomipramine trials, under multidisciplinary review (NICE 2005). Beyond dopamine blockade, the guidelines note serotonergic augmentation with the 5-HT3 antagonists ondansetron or granisetron, and glutamatergic options (topiramate, lamotrigine, riluzole, memantine) that BAP regards as weaker or unproven (BAP 2014). The augmentation target is often the patient with comorbid tics or poorer insight.

| AUGMENTATION OPTION | GUIDELINE STANDING | DOSE / NOTE |
|---|---|---|
| **Risperidone (Sizodon, Risdone)** | **First-line augmentation; strongest evidence (BAP, IPS, CANMAT)** | 1 to 3 mg/day, at least 8 weeks |
| **Aripiprazole (Arpizol, Asprito)** | First-line augmentation, co-equal with risperidone | low range, at least 8 weeks |
| **Ondansetron / granisetron** | Serotonergic (5-HT3) augmentation option | added to fluoxetine or fluvoxamine |
| **Topiramate / lamotrigine** | Weaker glutamatergic augmentation | SSRI add-on, limited evidence |

Augmentation in treatment-resistant OCD; low-dose risperidone or aripiprazole added to a continuing SSRI or clomipramine is the best-evidenced next rung, never monotherapy (BAP 2014; IPS 2025; CANMAT 2014). Doses are bare numbers in the cells; the unit is in the header line.

**GOOD TO REMEMBER**

- **Antipsychotic augmentation (risperidone / aripiprazole):** low-dose dopamine blockade *added* to a continuing SSRI or clomipramine for the confirmed partial responder in treatment-resistant OCD (risperidone 1 to 3 mg/day, low-dose aripiprazole) for at least 8 weeks; strongest evidence is for risperidone; the signature caution is that antipsychotics are augmentation only and never monotherapy for OCD.

### 17.4 Combining and intensifying ERP

**ERP is a first-line augmentation, not just a first-line treatment.** Where the failed pharmacological trial did not include structured exposure, adding or intensifying exposure and response prevention is itself a first-line augmentation strategy for the partial or non-responder to an SSRI, wherever feasible (the Indian Psychiatric Society 2025). BAP frames combining an SSRI or clomipramine with ERP as the step when efficacy needs to be maximised (BAP 2014), and for the refractory patient the psychological arm is intensified to therapist-led, high-frequency ERP. Family accommodation should be assessed and reduced, since high accommodation predicts poor outcome (the Indian Psychiatric Society 2017). The general exposure principle sits in the Psychotherapies, clinical assessment, MSE and nosology notes.

**GOOD TO REMEMBER**

- **Combining and intensifying ERP:** graded exposure with prevention of the neutralising ritual, added to pharmacotherapy as a first-line augmentation for partial responders; the principle is that a drug trial without ERP is an incomplete trial, so intensifying therapist-led exposure is a legitimate rung before escalating to somatic options; the first-line indication is the SSRI partial responder in whom real exposure has not yet been delivered.

### 17.5 Neuromodulation and the neurosurgical threshold

**Non-invasive neuromodulation.** Before the invasive options, rTMS and tDCS augmenting an SSRI are an evolving, preliminary-evidence option in partial or treatment-resistant OCD (the Indian Psychiatric Society 2025), and WFSBP lists rTMS among the non-pharmacological choices for resistant illness (WFSBP 2023). These sit below the ablative and stimulation options in invasiveness and are detailed in the ECT and neurostimulation notes.

**Who crosses the neurosurgical threshold.** The invasive options are reserved for severe, incapacitating OCD that has exhausted conventional drug and behavioural therapy. The practical selection criteria worth carrying: a chronic, severe, disabling illness (typically over 5 years), a surgical-candidate Y-BOCS of at least **26 to 30** out of a possible 40, and documented failure of an exhaustive array of adequate trials (each medication trial no less than 2 months), all confirmed by a multidisciplinary committee with formal informed consent (Kaplan and Sadock 2024). Fewer than 1% of treatment-seeking OCD patients meet current surgical criteria. In India this pathway runs through specialist tertiary centres such as NIMHANS; intravenous clomipramine is a further specialist option before device or lesion procedures (the Indian Psychiatric Society 2017).

**TRAP** **Neurosurgery is for the exhausted ladder, not the difficult patient.** The bar is a Y-BOCS of 26 to 40, years of failed adequate trials, and multidisciplinary consent; a distressing but incompletely treated case does not qualify.

### 17.6 Deep brain stimulation: reversible modulation of the CSTC circuit

**What DBS is.** **dbs** (deep brain stimulation) delivers high-frequency electrical stimulation through bilateral electrodes implanted stereotactically, connected to a subcutaneous pulse generator in the chest wall. Unlike a lesion it is *reversible and adjustable*: it can be switched off or reprogrammed if it causes adverse effects or is ineffective, though it commits the patient to indefinite specialist follow-up (Kaplan and Sadock 2024). It received FDA humanitarian device exemption for intractable OCD in 2009 and is an approved treatment in the European Union; other psychiatric uses remain investigational.

**The targets and the numbers.** The most frequent DBS target for OCD is the ventral capsule/ventral striatum (VC/VS), evolved from the anterior limb of the internal capsule and now extending posteriorly to include the ventral internal capsule, caudate and nucleus accumbens; other reported OCD targets are the nucleus accumbens directly, the subthalamic nucleus, the inferior thalamic peduncle and the medial forebrain bundle (New Oxford Textbook). A cohort of 34 patients at the VC/VS target on high-frequency stimulation for 36 months showed a **62%**

response rate, response being at least a **35%** Y-BOCS reduction; full effects typically take about 3 months to evolve. The rationale is modulation of the hyperactive cortico-striato-thalamo-cortical (CSTC) circuit; the fear-circuit anatomy sits in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes. Transient stimulation-related hypomania affects a substantial minority and resolves with parameter changes.

**GOOD TO REMEMBER**

- **Deep brain stimulation (DBS) for OCD:** reversible, adjustable high-frequency stimulation via bilateral implanted electrodes; the mechanism is modulation of the hyperactive CSTC circuit; the commonest target is the ventral capsule/ventral striatum (VC/VS), with roughly a 62% response rate (at least 35% Y-BOCS reduction) at 36 months; the signature point is FDA humanitarian device exemption for intractable OCD (2009), reserved for the fully exhausted ladder with a surgical Y-BOCS of 26 to 40.

### 17.7 Ablative neurosurgery: capsulotomy and cingulotomy

**The two lesion procedures in use.** Where DBS modulates, ablative **neurosurgery ocd** makes a permanent stereotactic lesion. Four bilateral lesion procedures were historically used; two remain current, anterior capsulotomy and anterior cingulotomy (Kaplan and Sadock 2024). *Anterior capsulotomy* (Leksell, Sweden) lesions the ventral part of the anterior limb of the internal capsule, impinging on the adjacent ventral striatum (the modern "ventral capsulotomy"), interrupting fibres between ventral prefrontal cortex and subcortical nuclei; a large review found about **64%** of cases substantially benefited. *Anterior cingulotomy* (Ballantine, Massachusetts General Hospital) places small thermocoagulation lesions in the anterior cingulate cortex (Brodmann areas 24 and 32) at the margin of the cingulum bundle; recent reviews show about 38 to 47% of OCD patients are full responders, with non-responders sometimes responding to a repeat procedure.

**The trade-off with DBS.** Ablation is a permanent, non-adjustable lesion and its outcome cannot be fairly judged for up to 2 years, but once done it needs no device team; DBS is reversible and acts within months but commits the patient to indefinite specialist programming. Both are last-rung options at specialist centres after the whole ladder has genuinely failed. The technical detail lives in the ECT and neurostimulation notes.

**WORKED EXAMPLE**

A 34-year-old woman has had incapacitating contamination OCD for 9 years, Y-BOCS 34 (extreme). Records confirm two adequate high-dose SSRI trials (fluoxetine 80 mg and sertraline 250 mg, each over 12 weeks with therapist-led ERP), an adequate clomipramine trial at 225 mg/day, and 8-week augmentation trials of risperidone then aripiprazole, all failed. After multidisciplinary review and formal consent at a tertiary centre, she is offered a reversible option first: VC/VS deep brain stimulation, with intensive ERP resumed within the first month post-implant. Only if that fails would an ablative capsulotomy be weighed. The point the examiner wants is that the whole ladder was audited as adequate before the somatic threshold was crossed.

| 10 to 12 weeks | 1 to 3 | 26 to 30 | 62 |
|---|---|---|---|
| ADEQUATE SSRI TRIAL AT MAXIMUM TOLERATED DOSE | RISPERIDONE AUGMENTATION, MG/DAY, AT LEAST 8 WEEKS | MINIMUM SURGICAL-CANDIDATE Y-BOCS (0 TO 40 SCALE) | VC/VS DBS RESPONSE RATE, PER CENT, AT 36 MONTHS |

**INDIA**

Treatment-resistant OCD sits inside India's wider anxiety-spectrum treatment gap: the disorder is common yet under-recognised, and much "resistance" in Indian outpatient practice is really under-dosing plus the reflex of a benzodiazepine, still over-prescribed, that does nothing for obsessions and risks dependence. The Indian Psychiatric Society CPGs, updated for OCD to 2025, anchor the same audit-the-trial-then-augment ladder, and reserve the tertiary pathway (intravenous clomipramine, DBS, capsulotomy) for the genuinely refractory patient managed through specialist centres such as NIMHANS. Indian brands for the augmentation and switch drugs are readily available, risperidone (Sizodon, Risdone), aripiprazole (Arpizol, Asprito) and clomipramine (Anafranil), so the true limiting factor is confirming an adequate trial, not access.

**MNEMONIC**

**AUDIT → SWITCH → AUGMENT → MODULATE → LESION**

The resistance ladder in order: **AUDIT** the failed trial (high dose, long duration, adherence, ERP) → **SWITCH** (optimise, then another SSRI or clomipramine) → **AUGMENT** (low-dose risperidone or aripiprazole, plus intensified ERP) → **MODULATE** (rTMS/tDCS, then reversible DBS at the VC/VS) → **LESION** (ablative capsulotomy or cingulotomy at a specialist centre).

**PEARL**

In the resistant-OCD vignette the marks are almost never in the neurosurgery; they are in proving the earlier ladder was adequate. Name the three axes of an adequate trial (high dose, 10 to 12 weeks, adherence with ERP) and place DBS and capsulotomy only after two SSRIs, clomipramine, augmentation and ERP have genuinely failed, and you have answered the question before you reach the operating theatre.

## 18 · Body dysmorphic disorder

The exam sells body dysmorphic disorder (**bdd**) as the "ugliness" disorder, but that framing hides the point: the defect is not there, or is so slight that no one else can see it, and the whole clinical picture is built out of an intrusive preoccupation plus repetitive behaviours done to check or fix it. That architecture, a distressing appearance-related thought that will not leave and a compulsive ritual done in response, is why the DSM-5-TR and ICD-11 moved **bdd** out of the somatoform group and into the **obsessive-compulsive and related disorders**, sitting alongside OCD, hoarding and the body-focused repetitive behaviours (Kaplan and Sadock 2024; ICD-11 CDDR). Read it as OCD wearing a mirror: the same obsession-then-compulsion loop, but the content is fixed on how the person looks.

**Why it matters, and what this note owns.** Two things make BDD disproportionately examinable and clinically dangerous. First, it is *under-recognised*, patients present to dermatology and

cosmetic surgery, not psychiatry, and hide the concern from shame, so the diagnosis is missed unless you ask. Second, it carries one of the highest suicide burdens in psychiatry, higher rates of suicidal ideation and attempts than most disorders it is confused with. This note teaches the diagnosis and its discriminations (the **imagined defect**, the repetitive behaviours, the muscle-dysmorphia variant, the poor-insight and delusional forms), then the guideline-anchored treatment: a serotonin reuptake inhibitor at high dose and BDD-tailored CBT, first-line for every insight level including the delusional one. The general OCD algorithm and deep antidepressant dosing live elsewhere and are cross-referenced.

### 18.1 The core: preoccupation with an imagined or slight defect

**The defining criterion.** BDD is a **preoccupation** with one or more perceived defects or flaws in physical appearance that are *not observable or appear only slight to others* (DSM-5-TR 2022; ICD-11 CDDR). That last clause is the whole diagnosis: the concern is out of all proportion to any objective finding. The preoccupation is time-consuming (typically occupying at least an hour a day and often far more), intrusive, distressing and hard to resist, exactly the phenomenology of an obsession. Concerns most often focus on the skin, hair and nose but can involve any body area, and most patients are preoccupied with several areas over the course of the illness.

**Not a normal appearance concern.** Everyone dislikes something about how they look; BDD is separated from that by the distress, the time consumed, the functional wreckage and the repetitive behaviours. Insight is characteristically poor, the belief that one is disfigured feels true, which is why simple reassurance and even a mirror showing an ordinary face do not shift it.

**GOOD TO REMEMBER**

- **Body dysmorphic disorder (defining criterion):** preoccupation with one or more perceived defects in appearance that are *not observable or appear only slight to others*, plus repetitive behaviours or mental acts performed in response, causing clinically significant distress or impairment; classified with the obsessive-compulsive and related disorders (DSM-5-TR 2022; ICD-11 CDDR).

### 18.2 The second pillar: repetitive behaviours and mental acts

**The compulsive half is required.** A DSM-5-TR diagnosis needs the person, at some point, to have performed *repetitive behaviours or mental acts* in response to the appearance concern (DSM-5-TR 2022). These are the BDD compulsions: mirror checking (repeated checking of mirrors and other reflective surfaces such as windows), excessive grooming (hair styling, make-up), camouflaging (a hat over "balding", sunglasses over "asymmetrical" eyes, heavy make-up), reassurance seeking, skin picking to "fix" the skin, and repeatedly comparing one's appearance with other people's (New Oxford Textbook; Kaplan and Sadock 2024). They average a striking **3 to 8 hours a day**, are distressing and hard to resist, and, like OCD compulsions, do not relieve the distress and often worsen it.

**Medical risks live here.** Some behaviours are physically dangerous: compulsive skin picking (done by roughly 27 to 45% of BDD patients) can cause serious skin damage occasionally needing emergency surgery, and compulsive tanning raises cancer risk. Camouflaging doubles as a *safety*

*behaviour*, motivated by avoiding a feared outcome, which is why it is a target in CBT rather than something to accommodate.

RULE **Obsession plus compulsion, aimed at appearance.** BDD is diagnosed only when the appearance preoccupation is accompanied by repetitive behaviours or mental acts (mirror checking, grooming, camouflaging, reassurance seeking, comparing) done in response to it, the OCD loop with appearance as its content.

### 18.3 Muscle dysmorphia: the specifier that runs the other way

**Too small, not too flawed.** Muscle dysmorphia is a DSM-5-TR specifier of BDD in which the person is preoccupied with the idea that their body build is *too small or insufficiently muscular* (DSM-5-TR 2022). It occurs nearly always in men, who describe themselves as "puny", "tiny" or "too small" while in reality looking normal or unusually muscular from excessive weightlifting or steroid use (Kaplan and Sadock 2024; ICD-11 CDDR). The specifier applies even if the person is also preoccupied with other body areas.

**Why it is the severe variant.** Muscle dysmorphia carries *poorer* quality of life and *higher* rates of suicidality and substance use than other forms of BDD, in particular anabolic-androgenic steroid abuse, with the attendant medical harms (New Oxford Textbook; Kaplan and Sadock 2024). Practical trap: it is easily missed because gym culture normalises the behaviour, and it must be separated from an eating disorder, if the preoccupation is with body fat or weight in someone meeting eating-disorder criteria, that diagnosis takes precedence and BDD is not coded.

GOOD TO REMEMBER

- **Muscle dysmorphia:** a DSM-5-TR specifier of BDD, preoccupation that one's body build is too small or insufficiently muscular; nearly always in men; associated with anabolic-androgenic steroid abuse and higher suicidality; excluded when the focus is body fat/weight in an eating disorder (DSM-5-TR 2022; Kaplan and Sadock 2024).

### 18.4 Insight, referential thinking and the delusional variant

**Insight is a specifier, not a separate disorder.** Both DSM-5-TR and ICD-11 grade insight rather than splitting off a "delusional" diagnosis. DSM-5-TR specifies *with good or fair insight*, *with poor insight*, and *with absent insight / delusional beliefs*; ICD-11 collapses these to **fair to good insight** versus **poor to absent insight** (DSM-5-TR 2022; ICD-11 CDDR). Insight is characteristically poor, most patients hold their belief with over-valued-idea or delusional conviction.

**The delusional form is still BDD.** The old label "delusional dysmorphophobia" is gone: when the appearance belief reaches delusional intensity it is coded as BDD with absent insight, *not* as a delusional disorder, provided the belief stays within the appearance theme (ICD-11 CDDR; Companion to Psychiatric Studies). This matters for treatment (18.6): delusional BDD responds to the SRI, not to an antipsychotic alone. Add to this a strong streak of *referential thinking*, most patients believe others take special notice of the "defect", stare or mock, and an interpretive bias that reads neutral faces as contemptuous, which fuels social withdrawal.

TRAP **Do not rediagnose delusional BDD as a psychotic disorder.** A delusional-intensity appearance belief confined to the appearance theme is BDD with absent insight; treat it with a serotonin reuptake inhibitor, because dopamine-antagonist monotherapy is usually ineffective for delusional BDD.

## 18.5 The discriminations that get tested

**Four boundaries own the marks.** BDD sits at the crossroads of several disorders, and the exam lives on the seams. Hold the discriminators as a table rather than prose.

| CONFUSED WITH | SHARED SURFACE | THE DISCRIMINATOR |
|---|---|---|
| **Social anxiety disorder** | Fear of being looked at, social avoidance | **In BDD the fear is specifically about others judging a *perceived appearance defect*; in social anxiety the fear spans behaviour and evaluation across social contexts, not one defect (ICD-11 CDDR)** |
| **Eating disorder (anorexia/bulimia)** | Distorted body image, appearance preoccupation | **If the preoccupation is with body fat or weight in someone meeting eating-disorder criteria, diagnose the eating disorder; BDD covers non-weight defects (DSM-5-TR 2022)** |
| **Obsessive-compulsive disorder** | Obsessions plus compulsions, poor-insight spectrum | **Content: BDD obsessions/compulsions are confined to *appearance*; if wider obsessional themes are present code OCD too (Kaplan and Sadock 2024)** |
| **Normal appearance concern** | Disliking a feature | **BDD adds an hour-plus daily preoccupation, repetitive behaviours, marked distress and functional impairment (DSM-5-TR 2022)** |
| **Delusional disorder (somatic)** | Fixed false belief about the body | **If the belief stays within the appearance theme it is BDD with absent insight, not delusional disorder (ICD-11 CDDR)** |

The five boundary calls for body dysmorphic disorder; in each, the discriminator is the *content and breadth* of the concern, not its intensity (DSM-5-TR 2022; ICD-11 CDDR).

**GOOD TO REMEMBER**

- **BDD discriminators:** vs social anxiety, the fear is about a *specific perceived defect*; vs eating disorders, the focus is *not* body fat/weight; vs OCD, the obsessions/compulsions are confined to *appearance*; a delusional-intensity appearance belief is still BDD (absent insight), not delusional disorder (DSM-5-TR 2022; ICD-11 CDDR).

### 18.6 The high suicide risk, and the cosmetic-surgery trap

**This is the number that matters most.** BDD carries one of the highest suicide burdens in psychiatry. In clinical samples, lifetime rates of suicidal ideation run **78 to 81%** and suicide attempts **24 to 28%** (New Oxford Textbook). Population studies show appearance-specific suicidal ideation and attempts many-fold higher than in non-BDD groups, and the completed-suicide rate, though preliminary, appears markedly elevated. Mental-health quality-of-life scores in BDD are *poorer* than in depression, OCD, schizophrenia or bipolar disorder, and hospitalisation and unemployment rates are high. Muscle dysmorphia and adolescent BDD carry even higher suicidality.

**Cosmetic treatment does not work and can harm.** A majority of BDD patients seek and receive costly cosmetic, dermatologic or dental treatment; it is essentially *ineffective* for BDD and can worsen the picture, with some patients performing dangerous self-surgery (New Oxford Textbook). The first clinical move on suspecting BDD is therefore to *redirect away* from the surgeon and toward psychiatric treatment, screening suicide risk from the outset.

> **PEARL**
>
> The patient in front of the dermatologist or cosmetic surgeon who returns again and again dissatisfied, or who fixates on a barely visible "flaw", is the classic undetected BDD. Because they hide the concern from shame and rarely volunteer it, you must ask directly, and once you suspect it, screen suicide risk before anything else. Cosmetic intervention rarely satisfies and can precipitate crisis.

### 18.7 First-line treatment: a high-dose SRI

**SRIs are first-line for every insight level.** Serotonin reuptake inhibitors (SSRIs, and clomipramine as the SRI-class TCA) are, with BDD-tailored CBT, the recommended first-line pharmacotherapy, endorsed by the NICE OCD/BDD guideline (New Oxford Textbook; NICE 2005). The evidence base includes a placebo-controlled fluoxetine trial and a blinded crossover showing the SRI clomipramine (Anafranil) beating the non-SRI desipramine, the direct parallel to the clomipramine-versus-SSRI point in OCD: it is the *serotonergic* action that matters, not the tricyclic structure. Crucially, delusional (absent-insight) patients respond to the SRI alone; antipsychotic monotherapy is usually ineffective for delusional BDD, so the SRI is given regardless of insight.

**Dose high, choose escitalopram or fluoxetine.** BDD, like OCD, needs *higher SRI doses than depression* (New Oxford Textbook). Escitalopram (Nexito, Rexipra) and fluoxetine are the best-studied and usual first choices, escitalopram having few drug interactions; a relapse-prevention trial used escitalopram at a mean of about **26 mg/day**. Citalopram is least preferred because its dosing ceiling sits below the dose usually needed. The brand names to lead with in India are the escitalopram, fluoxetine, sertraline and fluvoxamine antidepressant brands; clomipramine is Anafranil.

| AGENT | ROLE IN BDD | ANCHOR / DOSE NOTE (MG/DAY WHERE GIVEN) |
|---|---|---|
| **Escitalopram (Nexito, Rexipra)** | **Preferred first-line SRI** | Best-studied with fluoxetine; few interactions; relapse-prevention trial mean about 26 |
| **Fluoxetine** | First-line SRI | Placebo-controlled trial positive; dose higher than for depression |
| **Sertraline / fluvoxamine** | Alternative first-line SRIs | Open-label support; high-dose principle applies |
| **Clomipramine (Anafranil)** | **Reserved: SRI-class TCA after SSRI non-response** | Beat desipramine in a crossover; TCA cautions; kept behind SSRIs on tolerability |
| **Antipsychotic alone** | Not for delusional BDD as monotherapy | Dopamine-antagonist monotherapy usually ineffective; give the SRI |

Pharmacotherapy of BDD: a high-dose SRI first-line for every insight level, with clomipramine reserved for SSRI non-response (New Oxford Textbook; NICE 2005). Doses in the header; bare figures in the cells.

**WORKED EXAMPLE**

A man is fixated that his nose is grossly deformed; it looks normal, he checks mirrors and windows for hours, cancels social plans and has begun asking about rhinoplasty. Screen suicide risk (elevated in BDD), redirect from the surgeon, and start escitalopram, titrating toward a *high* dose and holding it for a full 12 to 16 weeks, at the highest tolerated dose for at least 3 to 4 of those weeks, before judging response. Add BDD-tailored CBT. Even if his conviction is delusional, the SRI, not an antipsychotic alone, is the treatment.

**GOOD TO REMEMBER**

- **SRI in BDD:** first-line pharmacotherapy at *high dose* (higher than for depression), escitalopram or fluoxetine preferred, clomipramine reserved for SSRI non-response; given for *all* insight levels because delusional BDD responds to the SRI while antipsychotic monotherapy usually does not; adequate trial 12 to 16 weeks at maximal tolerated dose (New Oxford Textbook; NICE 2005).

### 18.8 First-line treatment: BDD-tailored CBT

**Generic CBT is not enough.** The recommended psychotherapy is *cognitive behavioural therapy specifically tailored to BDD*, not off-the-shelf CBT (New Oxford Textbook; NICE 2005). It targets the appearance-related core beliefs ("I am defective", "I am unlovable"), uses **exposure and response prevention** against the rituals and safety/camouflaging behaviours, perceptual and mirror-retraining to break the zoomed-in scrutiny, and cognitive restructuring of the referential and threat-interpretation biases. The wider CBT model and the exposure principle are developed in the Psychotherapies, clinical assessment, MSE and nosology notes.

**How the two combine.** A high-dose SRI and BDD-tailored CBT are the two first-line pillars; combining them is standard for more severe or treatment-resistant cases, and psychiatric treatment (not cosmetic intervention) is what actually improves BDD symptoms and functioning.

**GOOD TO REMEMBER**

- **BDD-tailored CBT:** first-line psychotherapy; core = exposure and response prevention against checking/camouflaging plus cognitive restructuring of appearance beliefs and mirror-retraining; must be BDD-specific, not generic CBT; combined with a high-dose SRI in severe or resistant cases (New Oxford Textbook; NICE 2005).

## 18.9 Definitions to hold cold

### Imagined defect

The appearance flaw central to BDD is not observable, or appears only slight, to others; the concern is disproportionate to any objective finding.

### Mirror checking

The signature BDD compulsion: repeated checking of mirrors and reflective surfaces (also windows) to inspect the perceived defect; averages hours a day and does not relieve distress.

### Camouflaging

Hiding the disliked area (hats, sunglasses, make-up, posture); both a compulsion and a safety behaviour, a CBT target.

### Muscle dysmorphia

BDD specifier: preoccupation that the body is too small/insufficiently muscular; nearly always male; linked to anabolic steroid use and higher suicidality.

### Absent insight / delusional

Highest-conviction insight specifier; the appearance belief is held with delusional certainty yet remains BDD, responsive to the SRI.

**INDIA**

BDD is a textbook casualty of the Indian treatment gap for the anxiety, OCD and related disorders: it presents to dermatology, cosmetic and "fairness"-focused services, not psychiatry, and is rarely named, so a high-suicide-risk disorder goes untreated while the cosmetic industry profits from an intervention that does not work. The same benzodiazepine over-prescription reflex that plagues Indian anxiety practice is misplaced here too, an alprazolam script does nothing for the appearance preoccupation and delays the treatment that helps. The Indian Psychiatric Society CPGs anchor the obsessive-compulsive-spectrum standard: a high-dose SSRI first-line, escitalopram (Nexito, Rexipra) or fluoxetine, then clomipramine (Anafranil), with BDD-tailored CBT, and no place for a benzodiazepine as treatment. The genuinely high-yield Indian move is recognition and suicide screening: ask about hidden appearance preoccupation, and refer away from the surgeon.

| 78 to 81% | 24 to 28% | 3 to 8 | 12 to 16 |
|---|---|---|---|
| LIFETIME SUICIDAL IDEATION IN CLINICAL BDD SAMPLES | LIFETIME SUICIDE ATTEMPTS IN CLINICAL BDD SAMPLES | HOURS A DAY OCCUPIED BY BDD COMPULSIVE BEHAVIOURS | WEEKS: ADEQUATE HIGH-DOSE SRI TRIAL BEFORE JUDGING RESPONSE |

MNEMONIC

**MIRROR**

**M**uscle-dysmorphia specifier (too small, male, steroids); **I**magined or slight defect, not observable to others; **R**epetitive behaviours required (mirror checking, camouflaging, comparing); **R**eferential thinking and poor insight (delusional variant is still BDD); **O**CD-spectrum classification; **R**isk of suicide very high, and cosmetic surgery does not help. Treatment: high-dose SRI plus BDD-tailored CBT for every insight level.

HOLD THIS

BDD = preoccupation with a perceived defect that is unobservable or slight to others, *plus* repetitive behaviours (mirror checking, camouflaging, comparing), in the OCD spectrum; very high suicide risk; treat with a *high-dose* SRI (escitalopram or fluoxetine, clomipramine reserved) and BDD-tailored CBT for all insight levels, because delusional BDD responds to the SRI and cosmetic surgery does not.

## 19 · Hoarding, trichotillomania and excoriation

The exam packs three disorders into one short block, and the reason to learn them together is that all three are new arrivals in the **obsessive-compulsive and related disorders** chapter, sitting beside OCD and body dysmorphic disorder in both DSM-5-TR and ICD-11 (DSM-5-TR 2022; ICD-11 CDDR). Hoarding disorder was a subtype of OCD and is now a separable syndrome; trichotillomania moved out of the old impulse-control chapter; and excoriation disorder is brand new. Trichotillomania and excoriation are the two **body-focused repetitive** behaviour disorders, and the phrase you must be able to say is exactly that: recurrent, self-directed grooming behaviours (hair-pulling, skin-picking) that produce a physical lesion and that the person keeps trying, and failing, to stop.

**Why it matters, and what this note owns.** This is a low-yield block by volume but a high-yield one for keywords: each entity is a one-line viva answer built around a single defining criterion. The traps are all discriminations, hoarding versus normal collecting, trichotillomania versus alopecia, skin-picking versus an OCD compulsion, so the note teaches the criterion first and the discriminator second. On treatment, one behavioural technique carries the whole block: habit reversal training. The deep antidepressant dosing that these SSRIs share with OCD lives in the Mood disorders: pharmacotherapy notes and is cross-referenced, not repeated.

### 19.1 Hoarding disorder: the defining criterion is difficulty discarding

**Persistent difficulty discarding, not the clutter.** The essential feature of hoarding disorder is a *persistent* **difficulty discarding** or parting with possessions, regardless of their actual value (DSM-5-TR Criterion A). The examinable point is that the diagnosis is anchored on the *discarding difficulty*, not on the mess: the clutter is a downstream consequence. The difficulty is driven by a perceived need to save the items and by distress at the thought of discarding them (Criterion B), which is what makes the saving *purposeful* and separates it from passive accumulation.

**Clutter, distress and the third-party clause.** The retained possessions congest and clutter *active living areas* so their intended use is compromised, the person cannot cook in the kitchen or sleep in the bed (Criterion C), and this causes clinically significant distress or impairment, including an unsafe home (Criterion D). A neat examiner's hook sits in Criterion C: if the living areas are uncluttered *only because a third party* (family, cleaners, authorities) cleared them, the person still has the disorder. The *with excessive acquisition* specifier applies to the 80 to 90% who also over-buy or over-collect, and insight is graded good/fair, poor, or absent/delusional.

**GOOD TO REMEMBER**

- **Hoarding disorder (defining criterion):** persistent *difficulty discarding* possessions regardless of value, driven by a perceived need to save and distress at discarding (Criteria A to B), producing clutter that compromises active living areas (C) and significant distress/impairment (D); coded with the obsessive-compulsive and related disorders; specify *with excessive acquisition* (DSM-5-TR 2022; ICD-11 6B24).

### 19.2 Hoarding versus normal collecting, and versus the OCD it left

**Collecting is organised; hoarding is not.** The first discrimination is against **normative collecting**, which is organised, selective and a source of pleasure, and does *not* produce the clutter, distress or functional impairment of the disorder (DSM-5-TR 2022). Even when the sheer quantity is similar, the collector's items are curated and the living space stays usable; the hoarder's are piled in disorganised heaps in spaces meant for other purposes.

**Why it is no longer an OCD subtype.** In OCD, accumulation would be driven by an *obsession* (for example not discarding papers because they might contain harm-preventing information) and the saving feels ego-dystonic and unwanted. In hoarding disorder the saving is *not* triggered by an intrusive obsession; the person genuinely wants to keep the items and feels distress only when made to discard (New Oxford Textbook; DSM-5-TR 2022). Criterion F also forces you to exclude the medical and other-disorder mimics, brain injury and cerebrovascular disease, the low energy of depression, psychotic delusions, and the accumulation seen in major neurocognitive disorder, which is why new-onset hoarding in an older adult demands a cognitive and neurological work-up first.

**TRAP** **New-onset hoarding in an older adult is a red flag, not a diagnosis.** Before coding hoarding disorder, exclude brain injury, cerebrovascular disease and a major neurocognitive disorder (Criterion F); late-onset accumulation is disproportionately organic.

### 19.3 Hoarding: what actually helps

**Disorder-specific CBT is the mainstay; drugs are weak.** Hoarding disorder responds *poorly* to the SSRI-plus-ERP algorithm that works for OCD, part of the evidence that it is a distinct condition (New Oxford Textbook). The best-supported treatment is a *hoarding-specific* cognitive behavioural therapy that targets the discarding difficulty directly, sorting-and-discarding practice, cognitive work on saving beliefs and decision-making, motivational enhancement for the characteristically limited insight, and skills training for the organising and planning deficits. It is

delivered over many months and gains are partial; there is no quick pharmacological fix, so the framing to hold is behavioural-first.

**GOOD TO REMEMBER**

- **Hoarding-specific CBT:** the first-line treatment for hoarding disorder; targets the difficulty discarding, saving beliefs, decision-making, motivation and organising skills; the disorder responds *poorly* to the standard OCD SSRI regimen, so behaviour therapy, not a drug, leads (New Oxford Textbook).

### 19.4 Trichotillomania: recurrent hair-pulling with hair loss

**The five-part criterion.** Trichotillomania (hair-pulling disorder) is recurrent pulling out of one's own **hair-pulling** resulting in *hair loss* (Criterion A), with repeated attempts to decrease or stop (B), clinically significant distress or impairment (C), not attributable to another medical/dermatological condition (D), and not better explained by another mental disorder such as body dysmorphic disorder (E) (DSM-5-TR 2022). The commonest sites are scalp, eyebrows and eyelashes; pulling may be a focused ritual (with mounting tension then relief) or automatic (outside full awareness), and the person may examine, manipulate or swallow the pulled hair.

**Two clinical hooks worth marks.** It is a **body-focused repetitive** behaviour: crucially, it is *not* triggered by an obsession, which is exactly how it separates from OCD. And *trichophagia* (swallowing hair) can build a trichobezoar, a gastric hairball causing obstruction, anaemia or perforation, the classic surgical complication and a favourite viva twist. Onset typically coincides with puberty and the course is chronic with waxing and waning.

**GOOD TO REMEMBER**

- **Trichotillomania (defining criterion):** recurrent *hair-pulling resulting in hair loss* with repeated attempts to stop (Criteria A to B), distress/impairment (C), not due to a dermatological condition (D) and not explained by BDD (E); a body-focused repetitive behaviour *not* driven by an obsession; watch for trichophagia and trichobezoar (DSM-5-TR 2022).

### 19.5 Excoriation (skin-picking) disorder: the parallel entity

**The same architecture, on skin.** Excoriation (skin-picking) disorder is recurrent **skin-picking** resulting in *skin lesions* (Criterion A), with repeated attempts to decrease or stop (B), significant distress or impairment (C), not attributable to a substance (for example cocaine) or another medical condition such as scabies (D), and not better explained by another mental disorder (E) (DSM-5-TR 2022). Read it as the dermatological twin of trichotillomania: the second **body-focused repetitive** behaviour disorder, most often targeting the face, arms and hands, and, like hair-pulling, running from focused to automatic and not obsession-driven.

**The exclusions carry the traps.** Criterion D is the examiner's ground: skin-picking in amphetamine or cocaine intoxication ("meth mites", the delusion of infestation) is coded as a substance-induced obsessive-compulsive and related disorder, not excoriation disorder; and picking to correct a perceived appearance flaw is *body dysmorphic disorder*, not excoriation. Onset is often around puberty and the female preponderance is marked in clinical samples.

**GOOD TO REMEMBER**

- **Excoriation (skin-picking) disorder (defining criterion):** recurrent *skin-picking resulting in skin lesions* with repeated attempts to stop (Criteria A to B), distress/impairment (C), not due to a substance (cocaine) or medical condition (scabies) (D) and not explained by another disorder such as BDD (E); the second body-focused repetitive behaviour disorder (DSM-5-TR 2022).

RULE **Body-focused repetitive behaviours are not compulsions.** Hair-pulling and skin-picking produce a lesion and are *not* triggered by an obsession; that missing obsession is what separates trichotillomania and excoriation from OCD.

## 19.6 The four entities at a glance

**One table fixes the criterion and the discriminator.** The block is easiest to hold as a grid of "defining act" against "the thing it is *not*".

| DISORDER (ICD-11) | DEFINING CRITERION | KEY DISCRIMINATOR |
|---|---|---|
| **Hoarding disorder (6B24)** | Persistent difficulty discarding possessions, driven by need to save + distress | **Not normal collecting (that is organised, no distress); not obsession-driven as in OCD; exclude organic/neurocognitive causes** |
| **Trichotillomania (6B25.0)** | Recurrent hair-pulling with hair loss, repeated attempts to stop | **A body-focused repetitive behaviour, *no* obsession; not dermatological alopecia; not BDD hair removal; beware trichobezoar** |
| **Excoriation / skin-picking (6B25.1)** | Recurrent skin-picking with skin lesions, repeated attempts to stop | **Body-focused repetitive behaviour, *no* obsession; not stimulant-induced picking; not BDD skin-picking to fix a flaw** |
| **OCD (6B20, for contrast)** | Obsessions and/or compulsions; compulsions relieve obsession-driven distress | **The presence of an *obsession* driving the behaviour is what the three OCRDs above lack** |

The compact OCRD block: each entity is anchored on its defining act, and every discrimination turns on whether an *obsession* drives the behaviour (DSM-5-TR 2022; ICD-11 CDDR).

| 2.5 | 1 to 2 | 10:1 | 1200 to 2400 |
|---|---|---|---|
| HOARDING DISORDER POINT PREVALENCE, % | TRICHOTILLOMANIA 12-MONTH PREVALENCE, % | FEMALE-TO-MALE RATIO, TRICHOTILLOMANIA CLINICAL SAMPLES | N-ACETYLCYSTEINE (MG/DAY) TRIALLED IN TRICHOTILLOMANIA |

## 19.7 Habit reversal training: the shared first-line psychotherapy

**The one behavioural technique for the block.** Habit reversal training (HRT), developed by Azrin, is the first-line treatment for the body-focused repetitive behaviours (Kaplan and Sadock 2024). Its three core elements are the answer to any viva: *awareness training* (self-monitoring, to catch the urge and the moments that precede pulling or picking); *stimulus control* (changing the environment so the behaviour is less likely to start, for example gloves, plasters or a barrier); and *competing response training*, the core, engaging at the first sign of the urge in a motor behaviour

physically *incompatible* with the habit (classically clenching a fist for a minute) until the urge passes. Relaxation and social/contingency reinforcement support the package.

**Where drugs sit.** Pharmacotherapy is adjunctive and the evidence is thin. SSRIs are the usual first-line drug for the body-focused repetitive behaviours on tolerability grounds, and clomipramine (Anafranil) has the best trichotillomania trial data among the antidepressants; the glutamate modulator N-acetylcysteine reduced hair-pulling in a placebo-controlled trial and reads as comparable to behaviour therapy (Kaplan and Sadock 2024). Combining HRT with medication may beat either alone.

**GOOD TO REMEMBER**

- **Habit reversal training (principle and structure):** Azrin's first-line behavioural treatment for trichotillomania and excoriation; three elements, *awareness training*, *stimulus control*, and *competing response training* (a motor act incompatible with the habit); drugs (SSRIs, clomipramine, N-acetylcysteine) are adjunctive with a thin evidence base (Kaplan and Sadock 2024).

**INDIA**

This block magnifies the Indian treatment gap for the anxiety, OCD and related disorders, because these are the disorders patients hide hardest. Trichotillomania and excoriation present to dermatology and cosmetic services as "alopecia" or "acne", and hoarding is dismissed as untidiness or eccentricity, so the psychiatric diagnosis is rarely named. The reflex prescription of a benzodiazepine, still over-prescribed across Indian outpatient practice, does nothing for a body-focused repetitive behaviour and delays the behaviour therapy that helps. The Indian Psychiatric Society CPGs anchor the obsessive-compulsive-spectrum standard for the SSRI half, and Indian brands are readily available, fluoxetine (Fludac, Prodep), sertraline (Daxid, Zosert), escitalopram (Nexito), fluvoxamine (Fluvoxin) and clomipramine (Anafranil), so the limiting factor is recognition, not access. The genuinely high-yield Indian move is to ask directly about hair-pulling, skin-picking and discarding difficulty, and to refer to habit reversal training rather than reach for an anxiolytic.

**PEARL**

All four OCRDs in this block separate on one axis. OCD has an *obsession* driving a compulsion that relieves it. Hoarding, trichotillomania and excoriation have no obsession: the person *wants* to keep the objects, or the pulling/picking simply discharges tension. If you can find the obsession, you are in OCD; if you cannot, you are in one of the three new neighbours.

**HOLD THIS**

Hoarding is anchored on *difficulty discarding*; trichotillomania and excoriation are the two *body-focused repetitive* behaviours (hair-pulling with hair loss, skin-picking with skin lesions), none of them obsession-driven, and habit reversal training is the shared first-line treatment for the two body-focused ones.

## 20 · Illness anxiety and the OCRD spectrum

This is the short topic that fixes a favourite exam trap: where does illness anxiety actually live, and what is the **ocrd spectrum** that keeps trying to claim it? The clinical picture is old, the patient preoccupied with the fear of having a serious disease, checking the body and seeking or avoiding doctors, on a scale out of all proportion to any finding. The classical name was hypochondriasis; the stigma attached to that word is why DSM-5 renamed it (Kaplan and Sadock 2024). The examinable point is that the two manuals split it in opposite directions, and understanding why turns on the **obsessive-compulsive spectrum** idea.

**Why it matters, and what this note owns.** The whole disorder is built from an intrusive preoccupation plus repetitive behaviours (checking, reassurance-seeking) done in response, the same obsession-then-compulsion architecture as OCD, which is exactly why ICD-11 files **health anxiety** disorder inside the obsessive-compulsive and related disorders while DSM-5-TR files illness anxiety among the somatic-symptom disorders. This note teaches that placement, the criterion-anchored diagnosis of illness anxiety, the concept of the OCRD spectrum that binds OCD, BDD, hoarding, the body-focused behaviours and health anxiety by their shared repetitive-behaviour feature, and the first-line treatment. The illness-anxiety-versus-somatic-symptom-disorder boundary (how much bodily symptom is present) is worked out in the Somatic-symptom, sexual, impulse-control disorders and suicide notes; here we own the OCRD-facing side.

### 20.1 Illness anxiety disorder: the criterion and the two subtypes

**The defining criterion.** Illness anxiety disorder is a **preoccupation** with having or acquiring a serious illness, in which somatic symptoms are absent or, if present, only mild, and the preoccupation persists despite appropriate medical evaluation and reassurance (DSM-5-TR 2022; Kaplan and Sadock 2024). It is not held with delusional intensity and is not restricted to a concern about appearance (that would be body dysmorphic disorder). The person has a high level of health anxiety and performs excessive health-related behaviours, or shows maladaptive avoidance, and the duration must be at least **6 months** (though the specific feared illness may change over that time).

**Two subtypes, opposite behaviours.** DSM-5-TR specifies a *care-seeking type*, in which medical care including physician visits, tests and procedures is used frequently, and a *care-avoidant type*, in which medical care is rarely used (DSM-5-TR 2022). The care-avoidant patient is easy to miss precisely because they stay away from clinics; the fear is so intense that they avoid the very reassurance they crave. Core phenomenology, in the older language, is disease conviction, disease fear and bodily preoccupation.

**GOOD TO REMEMBER**

- **Illness anxiety disorder (defining criterion):** preoccupation with having or acquiring a serious illness with somatic symptoms absent or only mild, persisting *despite* appropriate evaluation and reassurance, for at least **6 months**; not delusional, not confined to appearance; specify *care-seeking* vs *care-avoidant* type (DSM-5-TR 2022).

### 20.2 The DSM-versus-ICD split: same patient, two chapters

**DSM-5-TR: renamed and split across the somatic group.** DSM-5 removed the term hypochondriasis for its pejorative connotation. About two-thirds to three-quarters of former hypochondriasis cases, those with prominent somatic symptoms, are recoded as *somatic symptom disorder*; the remaining minority (around **20%**), whose symptoms are minor and whose focus is the fear of illness itself, become *illness anxiety disorder* (Kaplan and Sadock 2024; DSM-5-TR 2022). Both sit in the somatic-symptom-and-related-disorders chapter. The dividing line is the amount of somatic symptom present.

**ICD-11: kept the name, moved it into the OCRD grouping.** ICD-11 retained the label *hypochondriasis (health anxiety disorder)* and placed it, as code 6B23, inside the **obsessive-compulsive or related disorders** grouping, not the somatic block (ICD-11 CDDR). Its stated rationale is shared phenomenology, repetitive intrusive thoughts about having an illness and repetitive or excessive health-related behaviours driven by that preoccupation, plus a similar treatment response. In ICD-11 the presence of somatic symptoms is explicitly *not* an essential feature, so the label turns on the anxious preoccupation and the checking, not on bodily complaints.

| FEATURE | DSM-5-TR | ICD-11 CDDR |
|---|---|---|
| **Name** | Illness anxiety disorder | **Hypochondriasis (health anxiety disorder)** |
| **Chapter** | Somatic symptom and related disorders | **Obsessive-compulsive and related disorders (6B23)** |
| **Somatic symptoms** | Absent or mild; if prominent, code somatic symptom disorder instead | **Not an essential feature** |
| **Rationale for placement** | Health-focused somatic preoccupation | **Repetitive intrusive thoughts + checking, like OCD; shared treatment response** |
| **Insight** | Not of delusional intensity | **Insight specifier applies (fair-to-good vs poor-to-absent)** |

The single most tested fact on this topic: the same clinical picture is an OCRD in ICD-11 but a somatic-symptom disorder in DSM-5-TR (ICD-11 CDDR; DSM-5-TR 2022).

RULE **Two manuals, opposite files.** Hypochondriasis / illness anxiety is an *obsessive-compulsive related disorder* in ICD-11 (6B23) but a *somatic-symptom disorder* in DSM-5-TR, split off by how much bodily symptom is present.

### 20.2b The OCRD spectrum: what binds the group

**The unifying feature.** The **ocrd spectrum** (the obsessive-compulsive related, or obsessive-compulsive spectrum, disorders) is the family of conditions linked by a shared core: an intrusive, repetitive *preoccupation* and the repetitive *behaviours* performed in response to it (New Oxford Textbook; ICD-11 CDDR). In ICD-11 the grouping contains obsessive-compulsive disorder (6B20), body dysmorphic disorder (6B21), olfactory reference disorder (6B22), hypochondriasis /

health anxiety disorder (6B23), hoarding disorder (6B24) and the body-focused repetitive behaviour disorders (6B25, trichotillomania and excoriation/skin-picking); Tourette syndrome is cross-listed for its analogous premonitory-urge-and-repetitive-behaviour phenomenology. The historical idea, proposed by Hollander, arranged these on a compulsivity-to-impulsivity dimension, harm-avoidant checking at one pole, reward-driven grooming behaviours at the other.

**What earns a place, and what does not.** Membership is defined by the repetitive-behaviour-or-preoccupation feature and by serotonergic treatment response, which is why illness anxiety, whose sufferers check the body and seek reassurance, fits so naturally that ICD-11 moved it in. The spectrum is a *conceptual* bridge: it explains why these disorders cluster, co-occur and respond to serotonin reuptake inhibitors, but it does not collapse them into one diagnosis. The insight specifier (fair-to-good vs poor-to-absent) applies across OCD, BDD, olfactory reference disorder, hypochondriasis and hoarding, another shared thread (ICD-11 CDDR).

**Obsessive-compulsive spectrum**
disorders linked by intrusive repetitive preoccupation, repetitive behaviours in response, and serotonergic treatment response

**OCD (6B20)**
obsessions and compulsions across varied content

**Body dysmorphic disorder (6B21)**
the loop fixed on a perceived appearance defect

**Hoarding disorder (6B24)**
difficulty discarding, driven by perceived need to save

**Body-focused repetitive behaviours (6B25)**
trichotillomania (hair-pulling), excoriation (skin-picking)

**Hypochondriasis / health anxiety (6B23)**
the loop fixed on the fear of serious illness

GOOD TO REMEMBER

- **OCRD spectrum (unifying feature):** conditions bound by an intrusive repetitive *preoccupation* plus repetitive *behaviours* done in response, and by serotonergic treatment response; ICD-11 grouping = OCD, BDD, olfactory reference disorder, hypochondriasis, hoarding, body-focused repetitive behaviours, with Tourette cross-listed (ICD-11 CDDR).

## 20.3 The discriminations: what illness anxiety is not

**Four boundaries carry the marks.** Illness anxiety sits between the somatic, anxious, obsessional and psychotic groups, and each seam is testable. Hold them as a table.

| CONFUSED WITH | SHARED SURFACE | THE DISCRIMINATOR |
|---|---|---|
| **Somatic symptom disorder** | Health preoccupation, doctor-shopping | **In SSD there are prominent, distressing *somatic symptoms*; in illness anxiety somatic symptoms are absent or mild and the focus is the *fear of illness* (DSM-5-TR 2022)** |
| **Generalised anxiety disorder** | Worry about health | **GAD worry spans many domains (finances, family, work); illness anxiety worry is *specifically and persistently* about having a serious disease (New Oxford Textbook)** |
| **Obsessive-compulsive disorder** | Intrusive illness thoughts, checking | **OCD illness thoughts are usually experienced as intrusive and senseless with wider obsessional content; illness anxiety fear is a sustained, ego-syntonic disease conviction (Kaplan and Sadock 2024)** |
| **Delusional disorder, somatic type** | Fixed belief about having a disease | **Illness anxiety belief is *not* of delusional intensity and yields, at least briefly, to evidence; a truly unshakeable somatic delusion is delusional disorder (Kaplan and Sadock 2024)** |

The four boundary calls for illness anxiety; the discriminator is the *somatic load, the breadth of worry, and the fixity of belief*, not the health theme itself (DSM-5-TR 2022; Kaplan and Sadock 2024).

TRAP **Do not upgrade illness anxiety to a somatic delusion.** The illness-anxiety belief is over-valued, not delusional; it softens with evidence even if it returns. A fixed, evidence-proof conviction of disease is delusional disorder (somatic type), a different diagnosis and a different treatment.

### 20.4 First-line treatment: the SSRI and CBT

**An SSRI, at an adequate trial.** The serotonin reuptake inhibitors are the first-line pharmacotherapy for health anxiety, consistent with its OCRD placement and serotonergic response (Kaplan and Sadock 2024). The pivotal evidence is Fallon and colleagues' placebo-controlled trial of fluoxetine (Fludac, Prodep) at **20 to 80 mg/day**: responders separated from placebo by around 8 weeks and the gain held to 24 weeks, so the trial must run long enough (about **12 weeks**) before it is judged a failure. As across the OCRD group, doses tend to the higher end, and any of the SSRIs, escitalopram (Nexito, Rexipra), sertraline (Daxid, Zosert), fluvoxamine (Fluvoxin) or fluoxetine, is reasonable; deep dosing lives in the Mood disorders: pharmacotherapy notes. Reassurance, though part of every intervention, is not a treatment on its own and, given repeatedly, feeds the checking loop.

**CBT is the prototype psychotherapy.** Cognitive behavioural therapy is the first-line, best-evidenced psychological treatment for health anxiety; controlled trials, including Barsky and

Ahern's brief six-session individual CBT, show durable falls in hypochondriacal beliefs and health anxiety at one-year follow-up (Kaplan and Sadock 2024). CBT targets the misinterpretation of benign bodily sensations, the catastrophic disease beliefs, and the safety behaviours (body-checking, reassurance-seeking, symptom-Googling) that maintain the fear, exactly the exposure-and-response-prevention logic of the wider OCRD group; the general CBT model lives in the Psychotherapies, clinical assessment, MSE and nosology notes.

**GOOD TO REMEMBER**

- **SSRI (in health anxiety):** first-line drug, serotonergic response mirroring the OCRD group; Fallon's RCT used fluoxetine at **20 to 80 mg/day** with response by ~8 weeks, so give an adequate (~**12-week**) trial before calling it a failure (Kaplan and Sadock 2024).
- **CBT (in health anxiety):** first-line, best-evidenced psychotherapy; targets misinterpretation of bodily sensations and the reassurance-seeking / checking safety behaviours, with durable one-year gains (Barsky and Ahern; Kaplan and Sadock 2024).

| | | | |
|---|---|---|---|
| **6 months** | **20 to 80** | **~20%** | **6B23** |
| MINIMUM DURATION FOR ILLNESS ANXIETY DISORDER | FLUOXETINE TRIAL DOSE IN HEALTH ANXIETY (MG/DAY) | OF FORMER HYPOCHONDRIASIS BECOMES ILLNESS ANXIETY (REST = SSD) | ICD-11 CODE, INSIDE THE OCRD GROUPING |

**INDIA**

Health anxiety is common in Indian general practice yet reaches psychiatry late, part of the wider anxiety and OCD treatment gap, and it is a prime setting for the country's benzodiazepine over-prescription problem: a worried, somatising patient is handed alprazolam or clonazepam for the anxiety rather than started on the SSRI and CBT that actually treat the disorder. The Indian Psychiatric Society clinical practice guidelines keep an SSRI (with CBT) as first-line for the anxiety and obsessive-compulsive disorders and reserve benzodiazepines for short-term crisis use only; the anxiolytic dependence story is in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. The SSRIs are widely and cheaply available under Indian brands, escitalopram as Nexito or Rexipra, fluoxetine as Fludac or Prodep, sertraline as Daxid or Zosert, so cost is rarely the barrier; recognition is.

**PEARL**

Read illness anxiety as OCD wearing a stethoscope: the intrusive thought is *"I have a serious disease"*, the compulsions are body-checking, symptom-Googling and reassurance-seeking, and the relief is momentary before the doubt returns. That framing tells you why ICD-11 put it in the OCRD grouping and why the treatment is the OCRD treatment, an SSRI at an adequate dose plus CBT with response prevention, not endless investigation and not a benzodiazepine.

**HOLD THIS**

Illness anxiety (hypochondriasis) is a somatic-symptom disorder in DSM-5-TR but an obsessive-compulsive related disorder (6B23) in ICD-11; first-line treatment is an SSRI plus CBT, never a benzodiazepine.

## Trauma and stressor-related disorders

### 21 · PTSD: clinical features and criteria

**Post-traumatic stress disorder** is the disorder in which a terrifying event does not become a memory. In health, a horrific experience is filed away as something that happened *there* and *then*; in **ptsd** it is relived *here* and *now*, forcing itself back into the present through intrusive images, flashbacks and nightmares, so that the person is driven to avoid every reminder and lives permanently braced for the next catastrophe. Examiners test this on a tight lattice of exact points, the definition of the qualifying trauma, the symptom clusters (four in DSM-5, three in ICD-11), the greater-than-one-month duration, the delayed-onset and dissociative subtypes, the epidemiology, and the **pcl-5** with its provisional cut-off, and the classic trap is to accept a personality-disorder label, or a benzodiazepine, where the answer is trauma.

**Why it matters clinically.** Trauma exposure is near-universal but **ptsd** follows in only a minority, which makes the diagnosis about a *specific* post-exposure syndrome rather than distress alone. Two facts anchor every answer. First, the phenomenological signature is re-experiencing *in the present* plus a persistent sense of current threat, which separates true **post-traumatic stress disorder** from ordinary painful recollection. Second, the two classifications no longer align neatly, so a full answer must hold both the DSM-5 four-cluster and the **icd-11 ptsd** three-cluster architecture side by side, and know that a patient can meet one and miss the other. The amygdala and fear-circuit anatomy on which the alarm is built is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes, and the treatment (trauma-focused psychotherapy first, then the SSRIs/SNRI, prazosin for nightmares, no benzodiazepine) sits in a separate management note; it is not re-derived here.

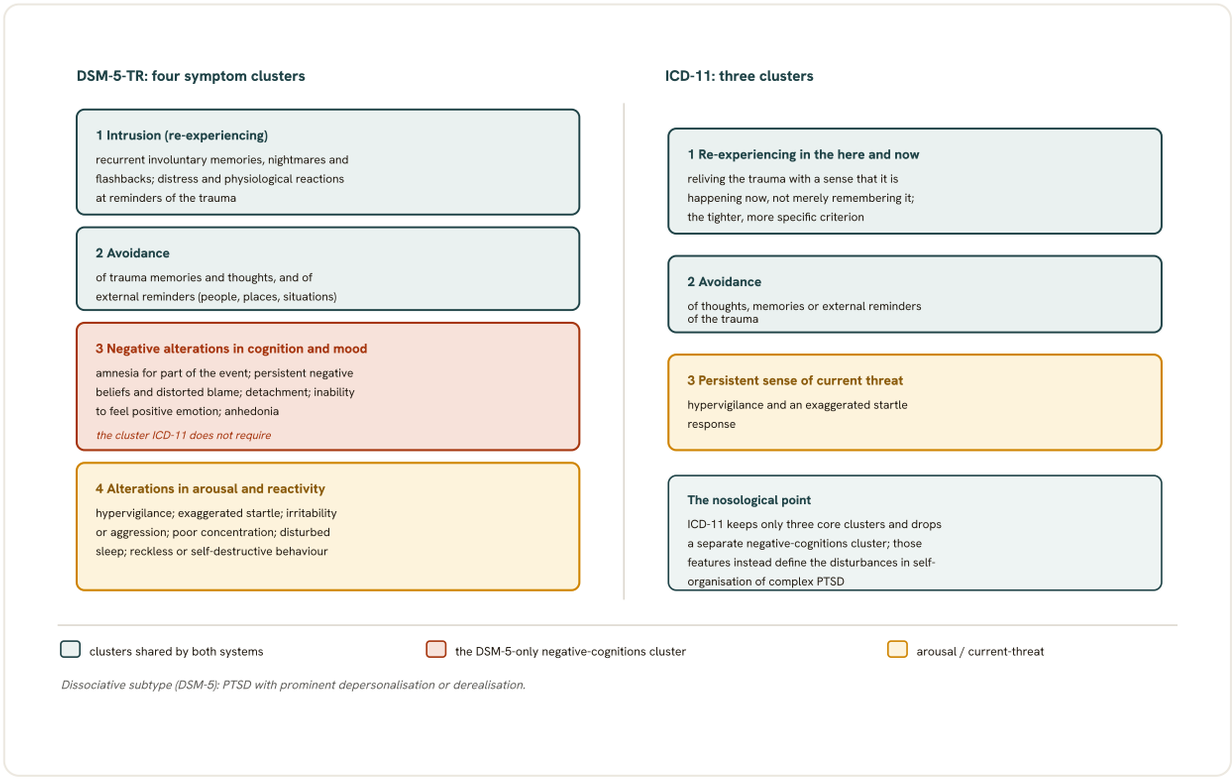



**Fig 14.14** The PTSD symptom clusters, DSM-5-TR against ICD-11. DSM-5-TR requires all four clusters: intrusion, avoidance, negative alterations in cognition and mood, and alterations in arousal and reactivity. ICD-11 keeps a tighter three: re-experiencing in the here and now (a sense that the trauma is happening now, not just being remembered), avoidance, and a persistent sense of current threat; it deliberately drops the separate negative-cognitions cluster, whose features instead define the disturbances in self-organisation of complex PTSD. DSM-5 also recognises a dissociative subtype with prominent depersonalisation or derealisation.

**PCL-5**

**Instructions:** Below is a list of problems that people sometimes have in response to a very stressful experience. Keeping your worst event in mind, please read each problem carefully and then select one of the numbers to the right to indicate how much you have been bothered by that problem in the past month.

Your worst event: _____

| In the past month, how much were you bothered by: | Not at all | A little bit | Moderately | Quite a bit | Extremely |
|---|---|---|---|---|---|
| 1. Repeated, disturbing, and unwanted memories of the stressful experience? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 2. Repeated, disturbing dreams of the stressful experience? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 3. Suddenly feeling or acting as if the stressful experience were actually happening again (as if you were actually back there reliving it)? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 4. Feeling very upset when something reminded you of the stressful experience? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 5. Having strong physical reactions when something reminded you of the stressful experience (for example, heart pounding, trouble breathing, sweating)? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 6. Avoiding memories, thoughts, or feelings related to the stressful experience? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 7. Avoiding external reminders of the stressful experience (for example, people, places, conversations, activities, objects, or situations)? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 8. Trouble remembering important parts of the stressful experience? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 9. Having strong negative beliefs about yourself, other people, or the world (for example, having thoughts such as: I am bad, there is something seriously wrong with me, no one can be trusted, the world is completely dangerous)? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 10. Blaming yourself or someone else for the stressful experience or what happened after it? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 11. Having strong negative feelings such as fear, horror, anger, guilt, or shame? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 12. Loss of interest in activities that you used to enjoy? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 13. Feeling distant or cut off from other people? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 14. Trouble experiencing positive feelings (for example, being unable to feel happiness or have loving feelings for people close to you)? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 15. Irritable behavior, angry outbursts, or acting aggressively? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 16. Taking too many risks or doing things that could cause you harm? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 17. Being "superalert" or watchful or on guard? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 18. Feeling jumpy or easily startled? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 19. Having difficulty concentrating? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |
| 20. Trouble falling or staying asleep? | 0 ○ | 1 ○ | 2 ○ | 3 ○ | 4 ○ |

**PCL-5** (18 August 2023) National Center for PTSD Page 3 of 3

The PCL-5: 20 self-report items mapped to the four DSM-5 clusters, each rated 0 to 4 over the past month (total 0 to 80); a provisional cut-off of about 31 to 33 suggests probable PTSD, but the National Center for PTSD states the cut-off as provisional and under review, so it is used to flag, not to diagnose.

PCL-5 (Weathers et al.); US National Center for PTSD, in the public domain (a work of the United States federal government).

**Fig 14.15** The PTSD Checklist for DSM-5 (PCL-5). The standard self-report measure for PTSD symptom severity and monitoring: twenty items, one per DSM-5 symptom, each rated 0 (not at all) to 4 (extremely) over the past month, for a total of 0 to 80. A provisional cut-off of about 31 to 33 flags probable PTSD, but the National Center for PTSD holds the cut-off as provisional, so the score screens and tracks rather than diagnoses. Shown as the actual public-domain Veterans Affairs form.

## 21.1 The traumatic-stressor criterion: what counts as Criterion A

**The gateway.** PTSD is one of the few diagnoses that requires a specified *cause*. DSM-5-TR Criterion A demands exposure to actual or threatened death, serious injury or sexual violence in one of four ways: *directly experiencing* the event; *witnessing* it in person as it happened to others; *learning* that it happened to a close family member or friend (where the event was violent or accidental); or *repeated or extreme exposure to aversive details* of traumatic events, as in first responders or those handling human remains (DSM-5-TR 2022; Kaplan & Sadock CTP 2024). The fourth route does *not* apply to exposure through electronic media, television, films or pictures unless that exposure is work-related.

**How the systems frame it.** ICD-11 requires exposure to an event or situation of an *extremely threatening or horrific* nature, listing disasters, combat, serious accidents, torture, sexual violence, terrorism, assault or acute life-threatening illness, and includes witnessing the sudden violent injury or death of others and learning of the sudden violent death of a loved one (ICD-11 CDDR). A historically examinable point: DSM-5 *removed* the old DSM-IV Criterion A2 requirement that the person felt intense fear, helplessness or horror at the time, because many who later develop full PTSD (combat personnel, first responders) show muted emotion at the moment of trauma (Kaplan & Sadock CTP 2024).

**WORKED EXAMPLE**

A 34-year-old woman survives a bus accident in which two fellow passengers die. Over the following weeks she is ambushed by vivid images of the crushed vehicle that arrive unbidden and feel as if it is happening again; she cannot take a bus, detours around the accident road, sleeps two hours a night, startles violently at horns, and believes the world is now lethally dangerous and that she should have saved the others. This is Criterion A (directly experiencing a life-threatening accident) followed by re-experiencing, avoidance, negative cognitions and hyperarousal persisting beyond a month: post-traumatic stress disorder, not an understandable transient reaction.

### 21.2 The DSM-5 four symptom clusters (B, C, D, E)

**Four groups, minimum counts.** DSM-5-TR parses PTSD into four symptom clusters, each with a required minimum, all beginning or worsening after the trauma (DSM-5-TR 2022; Kaplan & Sadock CTP 2024). **Cluster B, intrusion** (one or more of five): intrusive distressing memories, traumatic nightmares, dissociative flashbacks, intense distress at reminders, and marked physiological reactivity to reminders. **Cluster C, avoidance** (one or more of two): avoidance of trauma-related thoughts or feelings, and avoidance of external reminders (people, places, activities). **Cluster D, negative alterations in cognitions and mood** (two or more of seven): dissociative amnesia for the event, persistent negative beliefs about self or the world, distorted blame, persistent negative emotional state, markedly diminished interest, detachment or estrangement, and an inability to feel positive emotions. **Cluster E, alterations in arousal and reactivity** (two or more of six): irritability or aggression, reckless or self-destructive behaviour, hypervigilance, exaggerated startle, concentration problems, and sleep disturbance.

**The threshold criteria.** On top of the four clusters, the full picture must last *more than one month* (Criterion F), cause clinically significant distress or functional impairment (Criterion G), and not be attributable to a substance or another medical condition (Criterion H) (DSM-5-TR 2022). The negative-cognitions cluster (D) is the one that was *new* in DSM-5, absent from DSM-IV, and it widens PTSD from a fear-based reaction into a disorder of altered worldview, mood and self-belief.

MNEMONIC

**TRAUMA → IANA (1·1·2·2)**

The four DSM-5 clusters and their minimum counts, in order B-C-D-E. **I**ntrusion / re-experiencing (need **1** of 5), **A**voidance (need **1** of 2), **N**egative alterations in cognitions and mood (need **2** of 7), **A**rousal and reactivity (need **2** of 6). Then the three threshold gates: over one month, distress/impairment, not substance or medical.

## 21.3 Cluster B in depth: re-experiencing in the here and now

**The pathognomonic feature.** What makes an intrusion a PTSD symptom rather than an ordinary bad memory is that it is lived **re-experiencing** in the present, not retrospective recall. The intrusive memory carries the emotional and physiological charge of the original event and is experienced as recurring and current, even while the person knows intellectually that the trauma is in the past (Kaplan & Sadock CTP 2024). At the extreme, a *dissociative flashback* (Criterion B3) transports the person back so completely that they lose awareness of present surroundings. Nightmares thematically related to the trauma and marked distress or bodily reactivity on encountering reminders complete the cluster.

**Not sufficient.** The reciprocal point, which discriminates PTSD from rumination and grief, is that merely reflecting on, ruminating about, or remembering the feelings of the event is *not* enough to meet the re-experiencing requirement; the event must be experienced as occurring again in the here and now (ICD-11 CDDR). This is the exact line that separates PTSD re-experiencing from the preoccupied recollection of prolonged grief disorder.

RULE **Present-tense, not past-tense.** The single discriminating feature of PTSD is that the trauma is re-experienced in the here and now (intrusive images, flashbacks, nightmares that feel current), not simply remembered as belonging to the past; ordinary distressing recall or rumination does not meet the re-experiencing criterion.

## 21.4 Clusters C, D and E: avoidance, negative cognitions, hyperarousal

**Avoidance.** Because the re-experiencing is so intolerable, the person makes effortful attempts to avoid its triggers, both external (people, places, conversations, activities) and internal (thoughts, feelings, bodily sensations tied to the event). This **avoidance** tends to *generalise* over time, new objects and situations progressively drawn into the avoided set, which can confine severely affected patients to a shrinking, no-surprises world (Kaplan & Sadock CTP 2024).

**Negative cognitions.** The Cluster D **negative cognitions** and mood run beyond the event itself into fixed, permanent-feeling beliefs, the world is *always* dangerous, other people are untrustworthy, the self is irreversibly damaged or to blame, alongside anhedonia, detachment, a persistent negative emotional state and, in some, dissociative amnesia for part of the event. These overlap with depression and mark how far PTSD reshapes the whole disposition, not just the trauma response.

**Hyperarousal.** Cluster E, the alterations in arousal and reactivity, is the state of a threat-detection system stuck on: **hyperarousal** with hypervigilance (unrelenting scanning for danger),

exaggerated startle, irritability and angry outbursts, poor concentration, sleep disturbance, and reckless or self-destructive behaviour (DSM-5-TR 2022; Kaplan & Sadock CTP 2024). The irritability and aggression are frequently aimed at family and close relationships, deepening the person's isolation.

**PEARL**

The four clusters have an inner logic worth stating in a viva. The trauma stays intolerably alive (*intrusion*); because it is intolerable it is walled off (*avoidance*); the effort to make sense of it corrodes belief about self, others and the world (*negative cognitions*); and a system that failed to keep the person safe once now never stands down (*hyperarousal*). Naming that chain shows you understand PTSD as a coherent post-exposure trap, not a checklist.

### 21.5 The ICD-11 three-cluster model and the DSM-5 divergence

**ICD-11 6B40. Icd-11 ptsd** deliberately narrows the picture to three core elements, all developing after the trauma and lasting *at least several weeks* (ICD-11 CDDR). First, *re-experiencing* the traumatic event in the present (vivid intrusive memories, flashbacks or nightmares experienced as occurring again in the here and now). Second, *deliberate avoidance* of reminders, whether internal (thoughts, memories) or external (people, situations). Third, *persistent perceptions of heightened current threat*, indicated for example by hypervigilance or an enhanced startle reaction. Function must be significantly impaired, or maintained only through significant extra effort.

**Why the divergence is examinable.** ICD-11 collapses DSM-5's negative-cognitions cluster and much of avoidance out of the core criteria, keeping only three tighter **symptom clusters**, and reserves the wider disturbances of affect regulation, self-concept and relationships for the separate diagnosis of complex PTSD. The consequence, repeatedly demonstrated in military and international samples, is that many people who meet DSM-5 PTSD do *not* meet ICD-11 PTSD and vice versa (Kaplan & Sadock CTP 2024). Neither system should therefore be used to exclude a distressed, impaired trauma survivor from care.

| FEATURE | DSM-5-TR (309.81 / F43.10) | ICD-11 (6B40) |
|---|---|---|
| **Number of clusters** | **Four (B intrusion, C avoidance, D negative cognitions/mood, E arousal)** | **Three (re-experiencing in the present, avoidance, persistent sense of current threat)** |
| **Re-experiencing** | Intrusive memories, nightmares, flashbacks, cued distress, cued physiological reactivity (need 1 of 5) | Re-experienced in the here and now (not mere recall); required core element |
| **Avoidance** | Internal and/or external reminders (need 1 of 2) | Deliberate avoidance of reminders; required core element |
| **Negative cognitions and mood** | Separate required cluster D (need 2 of 7) | Not a core criterion; features moved to complex PTSD |

| FEATURE | DSM-5-TR (309.81 / F43.10) | ICD-11 (6B40) |
|---|---|---|
| **Arousal / current threat** | Cluster E arousal and reactivity (need 2 of 6) | Expressed as persistent perceptions of heightened current threat (e.g. hypervigilance, startle) |
| **Duration threshold** | More than 1 month | At least several weeks |
| **Complex presentations** | Handled within PTSD plus dissociative subtype | Separate diagnosis: complex PTSD (6B41) |

The DSM-5-TR four-cluster and ICD-11 three-cluster architectures of PTSD side by side; the key divergence is that ICD-11 drops the negative-cognitions cluster from the core and spins complex reactions into complex PTSD, so a patient can meet one system and not the other (criteria verified against DSM-5-TR 2022 and the ICD-11 CDDR).

**GOOD TO REMEMBER**

- **Post-traumatic stress disorder (the syndrome):** the defining criterion is a qualifying trauma (Criterion A, actual or threatened death, serious injury or sexual violence, experienced, witnessed, learned of, or through repeated occupational exposure) followed by a characteristic syndrome lasting *more than one month* (DSM-5) or *at least several weeks* (ICD-11); the discriminator from ordinary distress is re-experiencing *in the here and now* plus a persistent sense of current threat, and the first step is to confirm the stressor and then map the clusters (DSM-5 four: intrusion, avoidance, negative cognitions, arousal; ICD-11 three: re-experiencing, avoidance, current threat).

### 21.6 The dissociative subtype and delayed-onset (delayed expression)

**Dissociative subtype.** DSM-5 recognises a **dissociative subtype** of PTSD: full PTSD criteria are met and, in addition, there are persistent or recurrent symptoms of *depersonalisation* (feeling detached from, or an outside observer of, one's own mind or body) or *derealisation* (a sense that surroundings are unreal, dreamlike or distorted) (DSM-5-TR 2022; Kaplan & Sadock CTP 2024). It marks a subgroup who respond to reminders not with hyperarousal but with dissociation, characterised biologically by *over*modulation of the amygdala; the World Mental Health Surveys estimated it at about **14.4%** of those with PTSD.

**Delayed expression.** The *with delayed expression* specifier applies when the full diagnostic criteria are not met until *at least 6 months* after the event, even though some symptoms may have appeared immediately (DSM-5-TR 2022). The examinable subtlety is that genuine delayed *onset* from a truly asymptomatic state is rare; most apparent delayed-onset cases are worsening of pre-existing subsyndromal symptoms or delayed help-seeking, often re-triggered by a reminder or new stressor (Kaplan & Sadock CTP 2024).

**GOOD TO REMEMBER**

- **Dissociative subtype:** full PTSD *plus* persistent depersonalisation or derealisation; the signature caution is that these patients dissociate rather than hyperarouse to reminders (amygdala overmodulation), and it accounts for about 14.4% of PTSD.
- **Delayed expression specifier:** full criteria first met at least 6 months after the trauma; true asymptomatic delayed onset is rare, so look for pre-existing subsyndromal symptoms rekindled by a reminder or fresh stressor.

### 21.7 Epidemiology and course

**Common trauma, selective disorder.** Lifetime trauma exposure is near-universal (roughly 50 to 89% in United States samples; about 70% in a 69,000-adult worldwide study), yet only a minority exposed develop PTSD (Kaplan & Sadock CTP 2024). The National Comorbidity Survey Replication put the *lifetime* prevalence of PTSD in United States adults at **6.8%** and past-year prevalence at 3.5%. The disorder is roughly twice to three times as common in women (lifetime 9.7% in women versus 3.6% in men), partly reflecting the very high conditional risk after sexual assault.

**Course.** Conditional risk after trauma varies sharply by trauma type (highest after sexual assault, lower after accidents). Symptoms in most trauma-exposed people wane over the first months; nearly half of those who develop PTSD recover within 3 months, but roughly a third run a *chronic* course persisting for months to years, with survivors of interpersonal violence taking longest to remit (DSM-5-TR 2022; ICD-11 CDDR; Kaplan & Sadock CTP 2024). Symptom severity can fluctuate, patients crossing in and out of formal diagnostic threshold in the early years.

### 21.8 The PCL-5: what it is and its verified provisional cut-off

**What it is.** The **pcl-5** (the PTSD Checklist for DSM-5, Weathers et al. 2013) is a 20-item self-report questionnaire, one item for each of the 20 DSM-5 PTSD symptoms, each rated 0 to 4 over the past month and summed to a total score of **0 to 80** (Kaplan & Sadock CTP 2024). It takes about 5 to 10 minutes, is free from the United States National Center for PTSD, and its items are ordered by DSM-5 cluster (items 1 to 5 = Cluster B, 6 to 7 = Cluster C, and so on), so it can also support a provisional cluster-based diagnosis by counting endorsed symptoms per domain.

**The cut-off to memorise.** The PCL-5 is a screen and severity monitor, *not* a diagnosis (the CAPS-5 or SCID structured interview remains the gold standard). A total-score cut of **31 to 33** has been suggested as indicative of *probable* PTSD, with the CAPS-5 or SCID structured interview remaining the diagnostic gold standard (Kaplan & Sadock CTP 2024). Refer to the accompanying scale plate for the reproduced instrument; the number to carry is the provisional cut-off of 31 to 33 on a 0 to 80 range, and the concept map of the clusters is set out in the figure alongside.

TRAP **Treating a PCL-5 score as a diagnosis.** A PCL-5 of 33 or more is *probable* PTSD and a prompt to assess, not a diagnosis; the diagnosis needs a clinical interview confirming Criterion A and the cluster pattern. Equally, do not miss complex PTSD hiding behind a borderline personality-disorder label, chronic interpersonal trauma with affect dysregulation, disturbed self-concept and relational difficulty on top of the core PTSD triad is complex PTSD (ICD-11 6B41), and mislabelling it forfeits trauma-focused treatment.

| 31 to 33 | 0 to 80 | 1 month | 6.8% |
|---|---|---|---|
| PCL-5 PROVISIONAL CUT-OFF FOR PROBABLE PTSD | PCL-5 TOTAL RANGE (20 ITEMS, EACH 0 TO 4) | DSM-5 MINIMUM DURATION (OVER ONE MONTH) | LIFETIME PTSD PREVALENCE (NCS-R, DSM-IV) |

### 21.9 The India device: the treatment gap, benzodiazepines, and the IPS line

**Where PTSD lands in Indian practice.** India carries a very wide anxiety/OCD/PTSD treatment gap: trauma is common (road-traffic and industrial accidents, interpersonal and domestic violence, disasters), but PTSD is under-recognised, frequently somatised, and reaches formal care late, with most sufferers never seeing a mental-health professional (echoing the international finding that over half of people with PTSD do not seek treatment). Into that gap has flowed the benzodiazepine over-prescription problem: distress after trauma is met with a long-term benzodiazepine (typically alprazolam), which touches none of the core clusters and is explicitly *not* a PTSD treatment. The Indian Psychiatric Society CPGs and every major guideline put trauma-focused psychotherapy first, then an SSRI or the SNRI venlafaxine, with prazosin (Minipress, Prazopress) for trauma nightmares and propranolol (Ciplar, Inderal) in the peri-trauma setting; the benzodiazepine has no role. Indian brands of the anchor drugs are named where they arise, escitalopram as Nexito or Rexipra, clomipramine as Anafranil, in the accompanying management note.

**INDIA**

The single highest-yield Indian point is that a benzodiazepine is the wrong answer for PTSD twice over: it is not a treatment for any PTSD cluster, and early post-trauma benzodiazepine use may actually *increase* the risk of developing PTSD (CANMAT 2014). Against India's wide trauma treatment gap and its benzodiazepine over-prescription problem, the IPS-aligned answer is trauma-focused psychotherapy first, then paroxetine, sertraline or venlafaxine, with prazosin (Minipress, Prazopress) reserved for nightmares, never alprazolam as maintenance.

**HOLD THIS**

PTSD needs a Criterion-A trauma followed, for more than one month (DSM-5) or at least several weeks (ICD-11), by the cluster pattern: DSM-5 has FOUR clusters (intrusion 1/5, avoidance 1/2, negative cognitions and mood 2/7, arousal and reactivity 2/6) while ICD-11 has THREE (re-experiencing in the here and now, deliberate avoidance, a persistent sense of current threat); the pathognomonic feature is re-experiencing *in the present* not mere recall; specifiers are the dissociative subtype (depersonalisation/derealisation, ~14.4%) and delayed expression (full criteria first met at least 6 months on); lifetime prevalence is 6.8% (women ~2 to 3x men); and the PCL-5 is a 20-item, 0 to 80 self-report screen with a probable-PTSD provisional cut of 31 to 33, never a substitute for the CAPS-5/SCID interview.

## 22 · The evidence-based treatment of PTSD

The **evidence-based treatment of ptsd** is one of the cleanest algorithms in the anxiety and trauma syllabus, and examiners reward the candidate who states its two hard rules in the first sentence: *trauma-focused psychotherapy is first-line, and a benzodiazepine is never used*. Everything else, the choice of SSRI, the role of venlafaxine, prazosin for nightmares, antipsychotic augmentation for the refractory case, hangs off that spine. This is a management topic, so the marks are in the lines of treatment, the licensed agents, the doses, the monitoring and the sequence, not in re-describing the clusters (those sit in the separate PTSD clinical-features note).

**Why it matters clinically.** Every major guideline converges: BAP (BAP 2014), NICE (NICE 2018), CANMAT (CANMAT 2014), the WFSBP (WFSBP 2022) and the United States VA/DoD (VA/DoD 2023) all place a manualised **trauma-focused psychotherapy** ahead of any drug, and reserve pharmacotherapy for the patient who declines, cannot access, or does not respond to that psychotherapy. The **first-line ptsd** drugs are a tight list, sertraline and paroxetine (both licensed for PTSD), and venlafaxine, with **prazosin** added specifically for trauma-related nightmares. The deep antidepressant pharmacology of these agents, their doses, adverse effects and interactions, is developed in the Mood disorders: pharmacotherapy notes, and the general exposure/CBT model in the Psychotherapies, clinical assessment, MSE and nosology notes; this note assembles them into the PTSD algorithm and keeps to what the guidelines actually say.

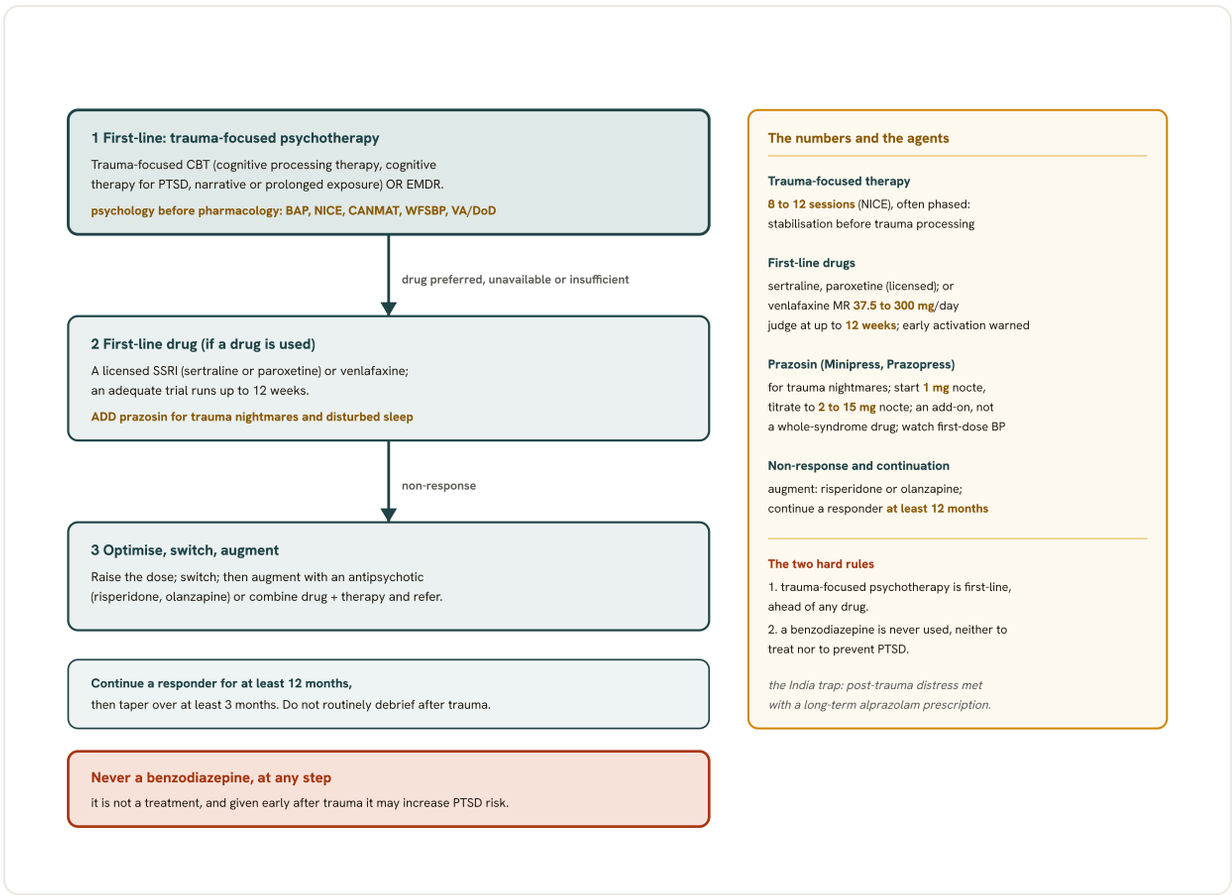



**Fig 14.16** The PTSD treatment algorithm. Its two hard rules open every answer: trauma-focused psychotherapy is first-line, and a benzodiazepine is never used. First-line is a manualised trauma-focused therapy, trauma-focused CBT or EMDR over 8 to 12 sessions, placed ahead of any drug by BAP, NICE, CANMAT, WFSBP and VA/DoD. Where a drug is used because the patient prefers it or therapy is unavailable or insufficient, the first-line agents are a licensed SSRI (sertraline or paroxetine) or venlafaxine (37.5 to 300 mg modified release), trialled up to 12 weeks, with prazosin (Minipress or Prazopress) added at 1 mg nocte and titrated to 2 to 15 mg for trauma nightmares. The non-responder has the dose raised, then a switch, then antipsychotic augmentation (risperidone, olanzapine) or a drug-plus-therapy combination with referral, and a responder continues for at least twelve months. The benzodiazepine is excluded throughout, a rule that collides directly with the Indian habit of meeting post-trauma distress with long-term alprazolam.

## 22.1 The governing principle: trauma-focused psychotherapy is first-line

**Psychology before pharmacology.** The single most examinable statement in PTSD management is that a trauma-focused psychological therapy, not a drug, is the first-line treatment. BAP instructs the clinician to "choose an evidence-based acute psychological treatment for chronic

PTSD: trauma-focused individual CBT or EMDR" (strength A) (BAP 2014). NICE goes further and makes it a *strong* recommendation to offer individual **trauma-focused cbt** or **emdr** in preference to drug treatment (NICE 2018), and the VA/DoD guideline states the point most explicitly of all: "recommend specific manualized trauma-focused psychotherapies over pharmacotherapy as first-line treatment for PTSD" (strong recommendation) (VA/DoD 2023). Drug treatment is offered where psychotherapy is declined, unavailable, or insufficient, not as an equal first option.

**What "trauma-focused" means.** The therapies that carry this recommendation are the ones that process the traumatic memory directly, cognitive processing therapy, cognitive therapy for PTSD, narrative exposure therapy and prolonged exposure therapy (the **tf-cbt** family), and eye movement desensitisation and reprocessing (**emdr**) (NICE 2018; VA/DoD 2023). Generic supportive counselling or non-trauma-focused CBT does not qualify. The WFSBP names CBT as "the psychotherapy with the best evidence for PTSD" (WFSBP 2022), and WHO mhGAP, the guideline that matters most for low-resource Indian settings, likewise puts individual or group trauma-focused CBT or EMDR first for adults and for children and adolescents (WHO mhGAP 2023).

RULE **Trauma-focused therapy first.** In PTSD the first-line treatment is a manualised trauma-focused psychotherapy, trauma-focused CBT (cognitive processing therapy, cognitive therapy for PTSD, narrative exposure, prolonged exposure) or EMDR; pharmacotherapy is second, offered only when psychotherapy is declined, unavailable or insufficient.

GOOD TO REMEMBER

- **Trauma-focused CBT / TF-CBT (the psychotherapy):** the mechanism is deliberate processing of the traumatic memory (cognitive processing, cognitive therapy for PTSD, narrative exposure, prolonged exposure) so the event is refiled as past rather than relived as present; it is FIRST-LINE for PTSD (offered before drugs), typically delivered over 8 to 12 sessions, more where there are multiple traumas, comorbid depression or dissociation (NICE 2018).
- **EMDR / eye movement desensitisation and reprocessing (the psychotherapy):** structured recall of the trauma paired with bilateral (eye-movement) stimulation to reprocess the memory; it is a first-line trauma-focused therapy alongside TF-CBT, and NICE offers it for PTSD presenting more than three months after a non-combat trauma over 8 to 12 sessions (NICE 2018; CANMAT 2014).

### 22.2 When to reach for drugs, and the two licensed SSRIs

**The indication for pharmacotherapy.** A drug is offered when the patient has a preference for medication over therapy, when a trauma-focused psychotherapy is not available or not acceptable, or when psychotherapy alone has been insufficient (NICE 2018; BAP 2014). NICE frames it precisely: "consider venlafaxine or an SSRI such as sertraline for adults with a diagnosis of PTSD if the person has a preference for drug treatment" (NICE 2018). Note the strength, this is a *weak* ("consider") recommendation, whereas the trauma-focused therapy is a *strong* ("offer") one.

**The ssri ptsd answer.** Of the SSRIs, two are licensed specifically for PTSD and are the drugs to name: sertraline and paroxetine. BAP identifies "paroxetine, sertraline or venlafaxine" as the first-

line pharmacological choice for chronic PTSD (strength A) (BAP 2014); CANMAT lists fluoxetine, paroxetine, sertraline and venlafaxine XR as first-line pharmacotherapy for core PTSD symptoms (CANMAT 2014); and VA/DoD, when a drug is used, offers "paroxetine, sertraline or venlafaxine" (strong) (VA/DoD 2023). The Maudsley concurs, an SSRI (paroxetine, sertraline or fluoxetine preferred) up to the maximum licensed dose is first-line drug treatment, and it repeats the rider that psychological approaches should be used before drugs (Maudsley 2021). The generic is primary in the note; the shared antidepressant brand list (fluoxetine, sertraline, fluvoxamine, paroxetine, escitalopram) is developed in the Mood disorders: pharmacotherapy notes.

**WORKED EXAMPLE**

A 40-year-old lorry driver, six months after a fatal highway collision, has nightly nightmares, cannot drive past the accident stretch and is hypervigilant and irritable. He declines "talking therapy" and asks for tablets. The evidence-based response is not a benzodiazepine and not reassurance. It is to explain that a trauma-focused psychotherapy would be the first choice, but that since he prefers medication, sertraline (starting low, titrating to the maximum tolerated licensed dose over several weeks) or paroxetine is appropriate; because trauma nightmares are prominent, prazosin can be added at night; and he is warned that a therapeutic effect may take up to 12 weeks to assess.

### 22.3 Venlafaxine: the SNRI first-line alternative

**Equal footing with the SSRIs.** The SNRI venlafaxine sits alongside the two licensed SSRIs as a first-line pharmacological option. BAP names it in the same breath as paroxetine and sertraline, noting that on meta-analysis "only paroxetine, sertraline and venlafaxine were superior to placebo" (strength A) (BAP 2014); the WFSBP recommends "SSRIs or the SNRI venlafaxine as first-line pharmacological treatment for PTSD" (WFSBP 2022); and NICE lists venlafaxine as the drug alternative to an SSRI (NICE 2018). On non-response to an SSRI, BAP's first step is to raise the dose (venlafaxine has post-hoc efficacy across trauma types), and only then to switch to another evidence-based treatment (BAP 2014).

**The dose to carry.** The Maudsley gives venlafaxine modified release **37.5 to 300 mg** daily for PTSD, recommended by NICE (Maudsley 2021). As with all these agents, an adequate trial is needed before declaring failure, guidelines advise that up to **12 weeks** may be required to assess pharmacological efficacy in PTSD (BAP 2014).

GOOD TO REMEMBER

- **Sertraline / paroxetine (SSRI, PTSD):** the two SSRIs licensed for PTSD and the first-line drug answer once psychotherapy is set aside; mechanism is serotonin re-uptake inhibition, the signature caution is that a full trial takes up to 12 weeks and early SSRI activation can transiently worsen symptoms, and continuation after response is for at least 12 months (BAP 2014; NICE 2018).
- **Venlafaxine (SNRI, PTSD):** the first-line SNRI alternative to the SSRIs, dosed as modified release 37.5 to 300 mg/day; the mechanism is dual serotonin-noradrenaline re-uptake inhibition, the caution is dose-related blood-pressure rise so monitor BP at higher doses, and on SSRI non-response raising the dose or switching to venlafaxine is the BAP-endorsed next step (BAP 2014; Maudsley 2021).

### 22.4 Prazosin: the targeted add-on for trauma nightmares

**What it treats and how.** Prazosin (Minipress, also marketed in India as Prazopress and Minipress XL) is a selective alpha-1 adrenergic blocker used *specifically* for trauma-related nightmares and sleep disturbance in PTSD, not for the disorder as a whole. It reduces central noradrenergic tone, damping the sympathetic surge that drives nightmares and the hyperaroused sleep of PTSD. CANMAT states it plainly: "for PTSD-associated trauma nightmares and poor sleep quality, use prazosin" (CANMAT 2014), and BAP notes that "prazosin augmentation reduces nightmares and other PTSD symptoms" (strength A) (BAP 2014). It is an *add-on* to a first-line SSRI or SNRI (or to psychotherapy), targeting one symptom cluster.

**The dose and the caution.** The Maudsley dose is **2 to 15 mg nocte**: initiate at 1 mg at night and titrate the dose gradually to reduce the risk of first-dose hypotension (Maudsley 2021). Because it is an antihypertensive, blood pressure and dizziness must be monitored (BAP 2014). Lead the drug with the Indian brand: prazosin is *Minipress* (and Prazopress) in Indian practice.

TRAP **Prazosin is not a PTSD cure-all.** Prazosin treats trauma nightmares and disturbed sleep, one symptom domain, and is an add-on, not a first-line agent for the whole disorder; do not offer it as monotherapy for core PTSD in place of a trauma-focused psychotherapy or a licensed SSRI/SNRI, and always start at 1 mg nocte to avoid first-dose hypotension.

GOOD TO REMEMBER

- **Prazosin (Minipress / Prazopress, alpha-1 blocker):** the targeted add-on for PTSD trauma nightmares and sleep disturbance; mechanism is alpha-1 adrenergic blockade lowering central noradrenergic tone, the signature caution is first-dose hypotension so start at 1 mg nocte and titrate to 2 to 15 mg nocte, and it augments (never replaces) first-line psychotherapy or an SSRI/SNRI (CANMAT 2014; Maudsley 2021).

### 22.5 The hard rule: no benzodiazepine in PTSD

**Not a treatment, and possibly harmful.** The most heavily tested negative rule in the whole topic is that a benzodiazepine is *not* used for PTSD, neither as treatment nor as prophylaxis. The VA/DoD makes it a strong recommendation "against the use of benzodiazepines for the treatment of PTSD" (VA/DoD 2023). CANMAT is equally firm and adds the mechanism of harm: "do not use benzodiazepines early after trauma to prevent PTSD; early benzodiazepine use may *increase* PTSD risk" (CANMAT 2014). NICE states that no drug treatment, "including

benzodiazepines", should be offered to prevent PTSD (NICE 2018), and BAP's do-not-use list for PTSD includes alprazolam (with citalopram, tiagabine and divalproex), which were not efficacious in placebo-controlled trials (BAP 2014).

**Why the error is so common.** Benzodiazepines touch none of the PTSD symptom clusters, they do not process the traumatic memory, do not reduce re-experiencing or avoidance, and blunt the emotional engagement that trauma-focused therapy depends on. Their apparent early calming effect is what makes the trap seductive, and their risk of dependence in a chronically distressed, often substance-comorbid population makes the error costly.

**INDIA**

This rule collides directly with Indian prescribing reality. Against a very wide anxiety/OCD/PTSD treatment gap, post-trauma distress is routinely met with a long-term benzodiazepine (typically alprazolam), the benzodiazepine over-prescription problem in Indian practice in one line. The guideline-anchored, IPS-aligned answer is the opposite: trauma-focused psychotherapy first, then sertraline, paroxetine or venlafaxine, with prazosin (Minipress, Prazopress) for nightmares, and no benzodiazepine, which is not merely ineffective but may raise the risk of PTSD when given acutely after trauma (CANMAT 2014). WHO mhGAP, designed for exactly these low-resource settings, keeps trauma-focused CBT or EMDR first (WHO mhGAP 2023).

### 22.6 The guideline-by-guideline algorithm

**Convergence across five guidelines.** The value of holding the guidelines side by side is that their agreement is the exam answer. Leading with BAP and NICE: first-line is trauma-focused psychotherapy (TF-CBT or EMDR); first-line drug, where a drug is used, is a licensed SSRI (sertraline or paroxetine) or venlafaxine; prazosin is added for nightmares; and the benzodiazepine is excluded. CANMAT, the WFSBP and VA/DoD add nothing that contradicts this and reinforce every element. The table alongside sets out the primary recommendations verbatim in structure so the concordance is visible.

| GUIDELINE (YEAR) | FIRST-LINE TREATMENT | FIRST-LINE DRUG (IF USED) | KEY ADD-ON / NOT-USED |
|---|---|---|---|
| **BAP (2014)** | Trauma-focused individual CBT or EMDR (strength A) | **Paroxetine, sertraline or venlafaxine (strength A)** | Prazosin for nightmares; do NOT use alprazolam, citalopram |
| **NICE (2018)** | Individual trauma-focused CBT or EMDR (strong "offer") | **Venlafaxine or an SSRI such as sertraline (weak "consider")** | Risperidone add-on if refractory; do NOT offer benzodiazepines, even to prevent PTSD |
| **CANMAT (2014)** | Individual trauma-focused CBT or EMDR (first-line) | **Fluoxetine, paroxetine, sertraline or venlafaxine XR (first-line)** | Prazosin for nightmares/sleep; do NOT use benzodiazepines (may increase risk) |

| GUIDELINE (YEAR) | FIRST-LINE TREATMENT | FIRST-LINE DRUG (IF USED) | KEY ADD-ON / NOT-USED |
|---|---|---|---|
| **WFSBP (2022)** | CBT (best-evidenced psychotherapy) | **SSRIs or the SNRI venlafaxine (first-line)** | Antipsychotic augmentation of an SSRI if unresponsive |
| **VA/DoD (2023)** | Manualised trauma-focused psychotherapy over pharmacotherapy (strong); PE / CPT / EMDR | **Paroxetine, sertraline or venlafaxine (strong)** | Recommend AGAINST benzodiazepines and against cannabis |

The evidence-based PTSD treatment algorithm across five guidelines: trauma-focused psychotherapy first everywhere, a licensed SSRI (sertraline/paroxetine) or venlafaxine as the first-line drug, prazosin added for nightmares, and the benzodiazepine excluded (recommendations verified against BAP 2014, NICE 2018, CANMAT 2014, WFSBP 2022 and VA/DoD 2023). The complete stepped algorithm is drawn in the accompanying figure.

### 22.7 The non-responder: augmentation and second-line agents

**Optimise, then switch, then augment.** When a first-line drug fails at an adequately tolerated dose, the BAP sequence is: first raise the dose, then switch to another evidence-based treatment (pharmacological or psychological), and only then, for the treatment-refractory case after dose optimisation and switching, combine an evidence-based drug with a trauma-focused psychotherapy and refer to specialist services (BAP 2014). The Maudsley second-line list (no fixed order, weaker evidence) is mirtazapine, phenelzine, amitriptyline, and the antipsychotics (Maudsley 2021).

**Antipsychotic augmentation.** For the disabling, unresponsive case, NICE says to "consider an antipsychotic such as risperidone in addition to psychological therapy" where symptoms have not responded and there is severe hyperarousal or psychotic symptoms (NICE 2018); the WFSBP endorses augmenting an SSRI with an antipsychotic for treatment-unresponsive PTSD (WFSBP 2022); and BAP notes RCT efficacy for risperidone and olanzapine as alternatives, with olanzapine or risperidone (or prazosin) as antidepressant-augmentation options in refractory PTSD (BAP 2014). Antipsychotics tend to help the intrusion symptoms (flashbacks, nightmares) more than avoidance or hyperarousal (Maudsley 2021).

**PEARL**

State the ladder as a single ordered line and you own the management viva: trauma-focused psychotherapy (TF-CBT or EMDR) first; if a drug is used, a licensed SSRI (sertraline, paroxetine) or venlafaxine, given an adequate trial of up to 12 weeks; add prazosin for nightmares; on non-response raise the dose, then switch, then augment with an antipsychotic (risperidone, olanzapine) or combine drug plus psychotherapy and refer; continue a responding drug for at least 12 months; and never a benzodiazepine at any step.

### 22.8 Prevention, early intervention and continuation

**Do not debrief.** A classic negative point: routine single-session or multiple-session psychological debriefing is *not* recommended to prevent PTSD after trauma, it does not help and may harm (BAP 2014; NICE 2018). For subthreshold symptoms within a month of the trauma, NICE advises active monitoring with follow-up within a month rather than immediate treatment (NICE

2018). Where preventive action is warranted after major trauma, BAP notes that propranolol (Ciplar, Inderal in India) or sertraline, or trauma-focused CBT, may be considered to reduce the emergence of post-traumatic symptoms (BAP 2014).

**How long to continue.** A drug that produces a response should be continued for at least **12 months** using a relapse-prevention approach (BAP 2014), with fluoxetine and sertraline having RCT evidence for longer-term relapse prevention. When stopping, taper gradually over an extended period (BAP suggests a minimum three-month taper) to avoid discontinuation and rebound symptoms (BAP 2014).

| 12 weeks | 12 months | 1 to 15 | 8 to 12 |
|---|---|---|---|
| TIME UP TO WHICH A PTSD DRUG TRIAL MAY BE NEEDED TO ASSESS EFFICACY (BAP) | MINIMUM CONTINUATION OF A RESPONDING DRUG | PRAZOSIN NOCTE RANGE, MG (START 1, TITRATE) | SESSIONS OF TRAUMA-FOCUSED CBT OR EMDR (NICE) |

### 22.9 The India device and the treatment gap

**Where the algorithm meets Indian practice.** Trauma is common in India (road-traffic and industrial accidents, interpersonal and domestic violence, disasters), yet PTSD is under-recognised, frequently somatised, and reaches formal care late. Trauma-focused psychotherapy, the first-line treatment everywhere, is scarce outside tertiary centres, which is precisely why the WHO mhGAP framing matters: it keeps trauma-focused CBT or EMDR first even in low-resource settings (WHO mhGAP 2023). The Indian Psychiatric Society clinical practice guidelines for the anxiety and stress-related disorders align with the international consensus, an SSRI as the first-line pharmacotherapy and a structured psychotherapy as the first-line psychological treatment, and the benzodiazepine reserved out of the PTSD pathway. Indian brands of the anchor drugs are named where they arise: prazosin as Minipress or Prazopress, propranolol as Ciplar or Inderal, with the shared SSRI antidepressant brand list carried in the Mood disorders: pharmacotherapy notes.

**TRAP** **Under-dosing and stopping too early.** A PTSD SSRI/SNRI trial must run to an adequate dose for long enough, up to 12 weeks may be needed before judging efficacy; abandoning the trial at three or four weeks, or never titrating past a token starting dose, manufactures a false "treatment failure" and pushes the prescriber toward the wrong answer (a benzodiazepine).

**HOLD THIS**

The evidence-based treatment of PTSD, in one line: trauma-focused psychotherapy FIRST (trauma-focused CBT, that is cognitive processing therapy / cognitive therapy for PTSD / narrative exposure / prolonged exposure, or EMDR), across BAP, NICE, CANMAT, WFSBP and VA/DoD; where a drug is used, a licensed SSRI (sertraline or paroxetine) or venlafaxine, trialled up to 12 weeks and continued at least 12 months if it responds; prazosin (Minipress/Prazopress) 1 mg nocte titrated to 2 to 15 mg for trauma nightmares; antipsychotic augmentation (risperidone, olanzapine) for the refractory case; and NEVER a benzodiazepine, which is not a treatment and may increase PTSD risk if given early after trauma.

## 23 · Complex PTSD

**Complex PTSD** is the diagnosis ICD-11 created for the survivor of trauma that was not a single event but a captivity: prolonged, repeated, inescapable harm, most damagingly in childhood. On top of the ordinary post-traumatic triad it adds a deeper wound, to the way the person regulates emotion, holds themselves in their own mind, and relates to other people. The examiner's target here is almost never the whole criterion list; it is one discrimination done cleanly. cptsd is the disorder most often mislabelled as a personality disorder, above all emotionally unstable (borderline) personality disorder, and the person who can hold the **distinction from personality disorder** in a viva is the person who will not forfeit a trauma survivor's access to trauma-focused treatment.




**Fig 14.17** ICD-11 complex PTSD contains the core PTSD triad, re-experiencing, avoidance and persistent current threat, plus affect dysregulation, negative self-concept and relational disturbance.

**Why it matters clinically.** **icd-11 complex** PTSD (6B41) is a *new* category, absent from DSM-5, built on Judith Herman's 1992 description of a syndrome in survivors of prolonged and repeated trauma. Its architecture is deliberately simple to state: the three core elements of PTSD *plus* three

**disturbances in self-organisation** (often abbreviated DSO), namely **affect dysregulation**, a persistent negative self-concept, and interpersonal difficulties. Two facts anchor every answer. First, you cannot have complex PTSD without first meeting PTSD; the DSO clusters sit on top of, never instead of, re-experiencing, avoidance and a sense of current threat. Second, the differential that examiners want is not PTSD versus complex PTSD (that is a severity-and-breadth step) but complex PTSD versus a personality disorder, because that is where real patients are misrouted. The core PTSD phenomenology is developed in the PTSD notes; the amygdala and fear-circuit substrate in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; the general exposure principle in the Psychotherapies, clinical assessment, MSE and nosology notes; the reactive attachment and disinhibited engagement disorders that can co-occur in maltreated under-5s in the Child behavioural, emotional and other disorders notes and the Child psychiatry: assessment, treatment, services and protection notes.

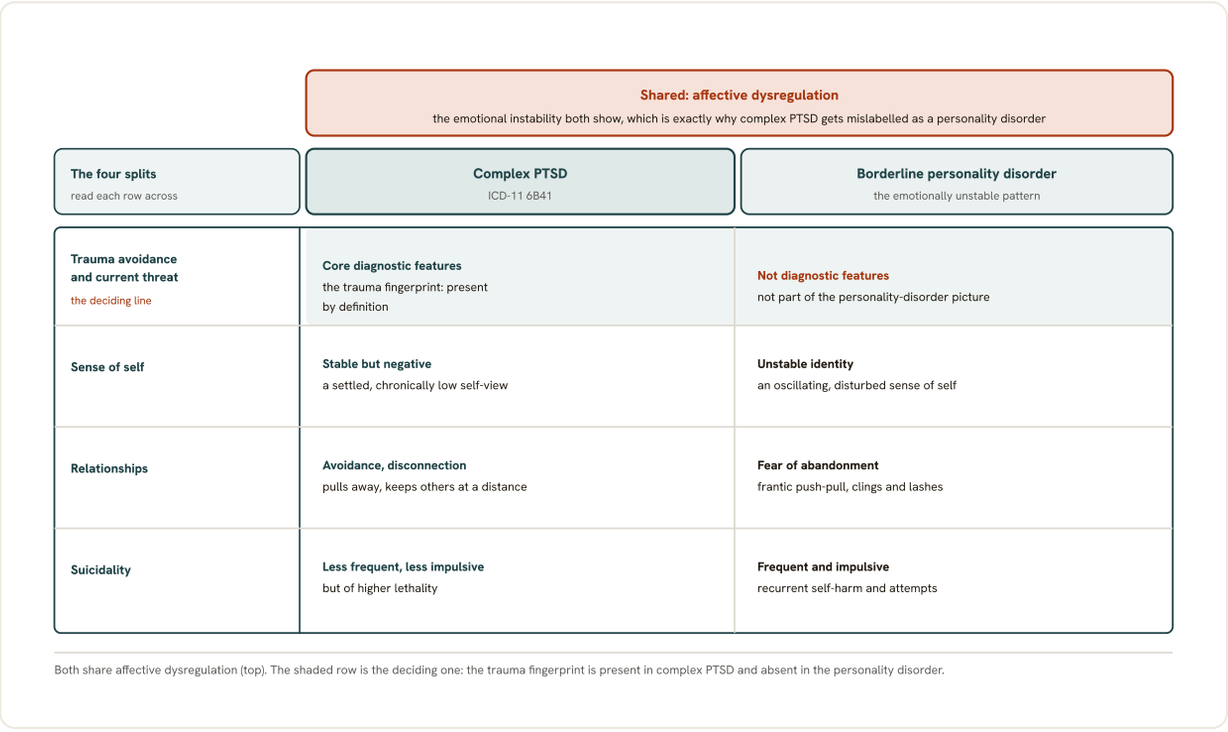



**Fig 14.18** Complex PTSD against borderline personality disorder. The two share their most visible symptom, affective dysregulation (the banner), which is exactly why a trauma survivor is so often mislabelled as borderline and the trauma missed. ICD-11 gives four lines that separate them. The deciding one, shaded, is the trauma fingerprint: trauma-reminder avoidance and a persistent sense of current threat are core to complex PTSD and are not diagnostic features of a personality disorder. Beneath it, the self-view is stable but negative in complex PTSD versus an unstable identity in the personality disorder; relationships are marked by avoidance and disconnection versus a fear of abandonment and push-pull instability; and suicidality is less frequent and less impulsive but of higher lethality, against the frequent, impulsive self-harm of the borderline pattern. Getting the label right unlocks trauma-focused treatment.

## 23.1 The definition: PTSD core plus three disturbances in self-organisation

**The whole diagnosis in one sentence.** Following the traumatic exposure, the person develops, for *at least several weeks*, all three core elements of PTSD *and* all three disturbances in self-organisation, with significant functional impairment (ICD-11 CDDR 6B41). The complex PTSD label is earned only when both halves are present; meet the PTSD triad alone and the diagnosis is PTSD (6B40), not complex PTSD.

**The two halves.** The *PTSD half* is the familiar triad: re-experiencing the event in the here and now (not mere recall), deliberate avoidance of reminders, and persistent perceptions of heightened current threat. The *self-organisation half* is the added signature of chronic entrapment: **affect dysregulation**, a persistent negative self-concept, and persistent difficulties in relationships. A single verbal-recall handle for the three DSO clusters is that the person cannot regulate feeling, cannot hold a workable view of the self, and cannot sustain closeness to others.

MNEMONIC

**PTSD + A S R**

Complex PTSD = the PTSD triad PLUS three disturbances in self-organisation. **A** = **A**ffect dysregulation (heightened reactivity, outbursts, reckless or self-destructive behaviour, dissociation under stress, emotional numbing). **S** = negative **S**elf-concept (persistent beliefs of being diminished, defeated or worthless, with pervasive shame, guilt or failure). **R** = **R**elationship difficulties (persistent trouble feeling close to or sustaining relationships with others). All three DSO clusters, on top of all three PTSD clusters.

### 23.2 The traumatic exposure: prolonged, repeated, inescapable

**The typical stressor.** Complex PTSD is generally a function of trauma that is *chronic, repeated and difficult or impossible to escape*, continuing over months or years: childhood physical, sexual or emotional abuse and severe neglect, domestic violence, torture, being trafficked, prolonged captivity or being a child soldier (ICD-11 CDDR; Herman 1992; Kaplan & Sadock CTP 2024). Exposure in early development carries a greater risk of complex PTSD (rather than PTSD) than does adult-onset trauma. Kaplan & Sadock frame simple PTSD as arising from a single or circumscribed event and complex PTSD from prolonged, inescapable stress, particularly in early childhood, complex precisely because of its extensive impact on all areas of development and functioning.

**The threshold caution.** Exposure to an extreme, prolonged or repetitive stressor from which escape is difficult does *not* in itself indicate complex PTSD; many people endure such stressors without developing any disorder (ICD-11 CDDR, boundary with normality). The presentation must meet all the diagnostic requirements. The stressor is the setting, not the diagnosis.

RULE **Two halves, both required.** Complex PTSD = the full PTSD triad PLUS all three disturbances in self-organisation (affect dysregulation, negative self-concept, interpersonal difficulty); no PTSD core, no complex PTSD, and DSO clusters without the PTSD triad point elsewhere.

### 23.3 Disturbance one: affect dysregulation

**What it looks like.** The presentation is characterised by severe and pervasive problems in **affect dysregulation** (ICD-11 CDDR). This runs in both directions on the arousal dial: *hyper*-regulation failure, showing as heightened emotional reactivity to minor stressors, violent outbursts, and reckless or self-destructive behaviour; and *hypo*-regulation, showing as emotional numbing, an inability to feel pleasure or positive emotion, and dissociative symptoms when under stress. A

telling ICD-11 detail is that, unlike in PTSD, the startle reaction in complex PTSD may be *diminished* rather than enhanced, the system shuts down rather than fires.

**Where affect dysregulation misleads.** This is the cluster that most resembles the affective instability of borderline personality disorder, and the overlap is genuine: affective dysregulation is a feature of both (ICD-11 CDDR). It is therefore the single cluster you must *not* read in isolation, because it is the shared ground on which the misdiagnosis is built. The discrimination lives in the accompanying figure, the complex-PTSD discriminator grid, and is unpacked in 23.6.

### 23.4 Disturbance two: a persistent negative self-concept

**What it looks like.** There are persistent beliefs about oneself as *diminished, defeated or worthless*, accompanied by deep and pervasive feelings of shame, guilt or failure related to the stressor (ICD-11 CDDR). The survivor of prolonged abuse may feel guilty for not having escaped, for having succumbed, or for not having prevented the suffering of others. The key adjective is *persistent*: this is a settled, stable, chronically low self-view, not a mood that swings across an afternoon.

**The discriminating quality.** Contrast this with the *unstable* sense of self of borderline personality disorder, where identity itself oscillates. In complex PTSD the self-view is bad but *steady*; in borderline PD it is bad and *unstable*. Stability of a negative self-concept is one of the cleanest single discriminators from a personality disorder (ICD-11 CDDR, boundary with personality disorder), and worth saying in exactly those words.

### 23.5 Disturbance three: interpersonal difficulties

**What it looks like.** There are persistent difficulties in sustaining relationships and in feeling close to others (ICD-11 CDDR). The characteristic pattern is *withdrawal*: the person consistently avoids, derides, or has little interest in relationships and social engagement. Where relationships do form they may be occasionally intense but hard to sustain. The organising theme is disconnection and avoidance of closeness.

**The discriminating quality.** This is the interpersonal cluster's crucial split from borderline PD. In complex PTSD, relationships are dominated by *avoidance and a general sense of disconnection*; they are *less* likely to be driven by fear of abandonment and the frantic, unstable, push-pull attachment that defines the borderline pattern (ICD-11 CDDR). Complex PTSD pulls away; borderline PD clings and lashes. That contrast (disconnection versus abandonment-driven instability) is the second clean discriminator.

**GOOD TO REMEMBER**

- **Complex PTSD (the syndrome, ICD-11 6B41):** the defining requirement is the full PTSD triad (re-experiencing in the here and now, deliberate avoidance, persistent sense of current threat) PLUS all three disturbances in self-organisation, affect dysregulation, a persistent negative self-concept, and interpersonal difficulties, lasting at least several weeks after prolonged/repeated/inescapable trauma, with functional impairment; the discriminator from PTSD is the added DSO clusters, and the first step is to confirm the PTSD core is present before adding the complex label.

### 23.6 The critical distinction: complex PTSD versus personality disorder

**Why the confusion is real.** Many people with a personality disorder have a history of childhood trauma, and affective dysregulation is a feature of both complex PTSD and personality disorder, particularly the borderline pattern (ICD-11 CDDR). The two therefore share their most visible symptom, emotional instability, which is exactly why the borderline label gets reached for and the trauma gets missed. ICD-11 lists four specific ways the profiles diverge, and these are the examinable discriminators.

**The four discriminators.** First, *avoidance of trauma reminders and a heightened sense of current threat* are core to complex PTSD and are *not* diagnostic features of a personality disorder, they are the trauma-specific fingerprint. Second, the *self-view*: complex PTSD shows a stable but persistently negative self-view, whereas an *unstable* sense of self is more typical of personality disorder. Third, *relationships*: complex PTSD is marked by avoidance and disconnection, personality disorder (borderline) by fear of abandonment and relationship instability. Fourth, *suicidality*: in personality disorder it tends to be frequent and impulsive, whereas in complex PTSD it tends to be less frequent, less impulsive, and of *higher lethality* (ICD-11 CDDR).

| FEATURE | COMPLEX PTSD (ICD-11 6B41) | PERSONALITY DISORDER, BORDERLINE PATTERN |
|---|---|---|
| **Trauma-reminder avoidance and heightened current threat** | **Core diagnostic features (the PTSD fingerprint)** | **Not diagnostic features** |
| **Sense of self** | **Stable but persistently negative self-view** | **Unstable sense of self / identity disturbance** |
| **Relationships** | Avoidance and general disconnection | Fear of abandonment, relationship instability (push-pull) |
| **Suicidality** | Less frequent, less impulsive, higher lethality | Frequent, often impulsive |
| **Affective dysregulation** | Present (shared feature) | Present (shared feature) |
| **Origin required for diagnosis** | Prolonged/repeated/inescapable trauma is typical, but the syndrome must be met | Enduring personality pattern; trauma frequent but not required |

The complex-PTSD-versus-borderline-personality-disorder discriminator grid; affective dysregulation is the shared feature that drives the misdiagnosis, and the four rows above it (trauma avoidance/current threat, stable-negative versus unstable self, disconnection versus abandonment, lethality pattern) are the ICD-11 lines that separate them (verified against ICD-11 CDDR 6B41, boundary with personality disorder). The concept map is set out in the figure alongside.

TRAP **Complex PTSD mislabelled as a personality disorder.** A childhood-abuse survivor with affective instability, low self-worth and stormy relationships is reflexively called borderline; the trauma-reminder avoidance, the persistent sense of current threat, the *stable* negative self-view and the disconnection (not abandonment-driven) pattern are the ICD-11 lines that make it complex PTSD, and the label change unlocks trauma-focused treatment.

**WORKED EXAMPLE**

A 27-year-old woman with a documented history of chronic childhood sexual abuse presents with intrusive flashbacks of the abuse that feel as if it is happening now, avoidance of anything that reminds her of it, and constant hypervigilance. On top of this she has explosive anger at trivial provocations alternating with numb emptiness, a fixed conviction that she is worthless and to blame, and a pattern of keeping everyone at arm's length. A previous clinician had labelled her borderline personality disorder on the emotional instability alone. The re-experiencing, the trauma-specific avoidance, the persistent current threat, the *stable* (not oscillating) negative self-view and the *avoidant* (not abandonment-driven) relational style make this complex PTSD (ICD-11 6B41), not a personality disorder, and reframe the whole treatment plan around the trauma.

**GOOD TO REMEMBER**

- **Complex PTSD versus personality disorder (the discrimination):** both share affective dysregulation, but complex PTSD is marked by trauma-reminder avoidance and a persistent sense of current threat (absent in PD), a *stable* negative self-view (versus an unstable sense of self), relationships dominated by avoidance/disconnection (versus fear of abandonment), and suicidality that is less frequent, less impulsive but of higher lethality; the trauma fingerprint is the deciding feature.

### 23.7 Management: trauma-focused psychotherapy first, guideline-anchored

**The management principle.** As for PTSD, the first-line treatment for complex PTSD is *trauma-focused psychotherapy*, not a drug (NICE 2018; BAP 2014). NICE 2018 covers complex presentations explicitly and directs an *urgent referral for specialist trauma assessment* when PTSD presents with severe dissociation or complex-PTSD features (pervasive affect dysregulation, severely disturbed self-concept, profound interpersonal difficulties), or when first-line trauma-focused therapy has failed or worsened symptoms. The evidence-based psychological treatments are individual trauma-focused CBT (cognitive processing therapy, cognitive therapy for PTSD, narrative exposure therapy or prolonged exposure) and EMDR (eye-movement desensitisation and reprocessing), each delivered over about **8 to 12 sessions**, extended where there are multiple traumas, comorbid depression or significant dissociation (NICE 2018). Clinically, complex PTSD often needs a *phased* approach: stabilisation and affect-regulation skills before trauma processing, because a poorly timed exposure can destabilise a dysregulated survivor.

**Where drugs sit.** Pharmacotherapy is an adjunct, not the spine. Where a drug is used, the first-line agents are the same evidence-based antidepressants as in PTSD: sertraline, paroxetine or the SNRI venlafaxine (BAP 2014; NICE 2018; CANMAT 2014). Adequate response can take up to **12 weeks** to judge, and responders should continue for at least **12 months** (BAP 2014). For trauma nightmares and disturbed sleep, prazosin (Minipress, Prazopress) is the targeted agent, an alpha-1 antagonist added for the nightmare symptom, not a whole-syndrome treatment (CANMAT 2014; BAP 2014). Where disabling hyperarousal or psychotic-level symptoms resist first-line care, NICE allows adjunctive risperidone alongside psychological therapy. The detailed SSRI/SNRI pharmacology (doses, adverse effects, interactions) sits in the Mood disorders: pharmacotherapy notes and is not re-derived here.

**GOOD TO REMEMBER**

- **Trauma-focused psychotherapy (the first-line treatment):** the principle is that complex PTSD, like PTSD, is treated *psychologically first*, with trauma-focused CBT or EMDR (about 8 to 12 sessions, often phased with stabilisation before processing); the signature caution is that a drug is an adjunct, and NICE 2018 directs urgent specialist referral for complex-PTSD features or when first-line therapy has failed or worsened.
- **Prazosin (Minipress, Prazopress):** an alpha-1 adrenergic antagonist; the one indication to anchor is trauma-related *nightmares* and sleep disturbance, added on to a first-line antidepressant, with blood pressure and dizziness the signature cautions, never a stand-alone PTSD treatment.

## 23.8 The India device: the trauma treatment gap, benzodiazepines, and the IPS line

**Where complex PTSD lands in Indian practice.** India carries a very wide anxiety, OCD and PTSD treatment gap, and complex PTSD sits at its blindest point. The precipitating traumas, chronic childhood abuse and neglect, prolonged domestic and interpersonal violence, are precisely those that are under-reported, stigmatised and reach formal care late, and the presentation, emotional instability with self-loathing and stormy relationships, is the one most readily misfiled as a personality disorder or dismissed. Into that gap flows the benzodiazepine over-prescription problem: a long-term benzodiazepine (typically alprazolam) is handed to a distressed survivor, touching none of the six clusters and, given early after trauma, potentially *increasing* the risk of PTSD (CANMAT 2014). The Indian Psychiatric Society CPGs, aligned with NICE and BAP, put trauma-focused psychotherapy first, then an SSRI (sertraline, paroxetine) or venlafaxine, with prazosin (Minipress, Prazopress) reserved for nightmares; the benzodiazepine has no maintenance role. Indian brands of the anchor antidepressants (sertraline, paroxetine, escitalopram) are named in the Mood disorders: pharmacotherapy notes.

**INDIA**

The highest-yield Indian point is that complex PTSD is doubly failed in Indian practice: the chronic-abuse trauma is under-recognised, and the affective-instability presentation is misrouted to a personality-disorder label (or an alprazolam prescription) instead of trauma-focused care. Against India's wide trauma treatment gap and its benzodiazepine over-prescription problem, the IPS-aligned answer is to name the complex PTSD, treat it psychologically first (trauma-focused CBT or EMDR), add an SSRI or venlafaxine only as an adjunct, use prazosin for nightmares, and never a benzodiazepine as maintenance.

| 6B41 | 3 + 3 | 8 to 12 | 12 months |
|---|---|---|---|
| ICD-11 CODE FOR COMPLEX PTSD (PTSD IS 6B40) | PTSD CORE CLUSTERS PLUS DISTURBANCES IN SELF-ORGANISATION | TRAUMA-FOCUSED CBT / EMDR SESSION RANGE (NICE 2018) | MINIMUM CONTINUATION OF DRUG IN RESPONDERS (BAP 2014) |

**HOLD THIS**

Complex PTSD (ICD-11 6B41, NOT in DSM-5) = the full PTSD triad (re-experiencing in the here and now, deliberate avoidance, persistent current threat) PLUS all three disturbances in self-organisation (affect dysregulation, a persistent negative self-concept, interpersonal difficulties), after prolonged/repeated/inescapable trauma; the critical discrimination is from borderline personality disorder, and although affective dysregulation is shared, complex PTSD is set apart by trauma-reminder avoidance and a persistent sense of current threat (absent in PD), a STABLE (not unstable) negative self-view, relationships driven by avoidance/disconnection (not fear of abandonment), and suicidality that is less impulsive but higher-lethality; treatment is trauma-focused psychotherapy FIRST (trauma-focused CBT or EMDR), with an SSRI or venlafaxine as adjunct and prazosin for nightmares, never a benzodiazepine.

## 24 · Acute stress reaction and acute stress disorder

These two labels sit in the hours-to-weeks window immediately after a terrifying event, and the whole examinable point is that they are *not* the same kind of thing. **Acute stress reaction** is the ICD-11's name for a **transient stress response**, a normal reaction to an exceptional stressor that the classification deliberately keeps *outside* the mental disorders; **acute stress disorder** is the DSM-5's *diagnosable* disorder in the first month after trauma. Getting the framing right, which is a disorder and which is not, which classification owns which, and how each relates to **ptsd**, is exactly what a viva examiner probes.

**Why it matters clinically.** Most people exposed to horror react and then recover; a minority stay ill. The early window is where clinicians must decide who is having an expectable acute stress reaction that needs support and time, and who is developing a disorder that needs treatment, without medicalising the first or missing the second. The trap that runs through the whole area is reflexively reaching for a benzodiazepine in the acute aftermath, which is not a treatment and may actually worsen the trajectory. The amygdala and fear-circuit anatomy that drives the acute autonomic storm is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; the full-blown syndrome and its clusters belong to the PTSD notes and are not re-derived here.

### 24.1 The core distinction: a reaction that is not a disorder versus a disorder

**Two different objects.** The single most tested fact is definitional. ICD-11 acute stress reaction (code **QE84**) is *not* considered a mental disorder: it is placed in Chapter 24, on factors influencing health status or contact with health services, precisely to signal that a transient response to an extreme stressor is normal (ICD-11 CDDR). It is only secondary-parented into the grouping of disorders specifically associated with stress to aid differential diagnosis. DSM-5-TR acute stress disorder (F43.0), by contrast, is a full trauma- and stressor-related *disorder* with operational criteria, a symptom threshold and a duration window (DSM-5-TR 2022; Kaplan & Sadock CTP 2024).

**The consequence for the two systems.** ICD-11 therefore has *no* "acute stress disorder" among its disorders: its stress grouping runs PTSD, complex PTSD, prolonged grief disorder and adjustment disorder, with acute stress reaction sitting outside as a non-disorder (ICD-11 CDDR).

DSM-5 *does* carry acute stress disorder as a diagnosis. So the same distressed trauma survivor in the first weeks may be given an ICD-11 "acute stress reaction" (a health-status factor, no disorder) or a DSM-5 "acute stress disorder" (a diagnosis), depending entirely on which manual is used.

RULE **Reaction is normal; disorder is diagnosable.** ICD-11 acute stress reaction is a transient normal response classified *outside* the mental disorders (Chapter 24, QE84); DSM-5 acute stress disorder is a diagnosable disorder in the first month. If the manual is ICD-11 there is no "acute stress disorder" to give.

### 24.2 Acute stress reaction: what it is and its time frame

**The ICD-11 description.** Acute stress reaction is the development of transient emotional, somatic, cognitive or behavioural symptoms after exposure to an event or situation, short- or long-lasting, of an *extremely threatening or horrific* nature (natural or human-made disasters, combat, serious accidents, sexual violence, assault) (ICD-11 CDDR). The picture is mixed and shifting: autonomic signs of anxiety (tachycardia, sweating, flushing), being in a daze, confusion, sadness, anxiety, anger, despair, over-activity, inactivity, social withdrawal, or at the extreme a stupor. Crucially, "the response to the stressor is considered to be normal given the severity of the stressor, and usually begins to subside within a few days after the event or following removal from the threatening situation".

**The classic ICD-10 timings to carry.** The older ICD-10 acute stress reaction, still the version most exam stems use, sets sharp temporal anchors: symptoms appear *within minutes* of the exceptional stressor and resolve rapidly, typically within a few hours once the stressor is removed, and within **1 to 3 days** even if it is not, so that a transient reaction lasts considerably less than **48 hours** (Companion to Psychiatric Studies; ICD-10). It is more likely to develop, and to be more severe, in the presence of physical exhaustion or at the extremes of age. The point that unifies both editions is transience: this is a storm that passes.

**WORKED EXAMPLE**

A previously well 40-year-old man is pulled from a collapsed scaffold at a construction site. For the next couple of hours he is dazed and disoriented, cannot recall parts of the rescue, is tremulous, tachycardic and sweating, alternates between agitation and withdrawal, and briefly cannot take in what is said to him. By the evening, moved to a quiet ward and reassured, he is oriented, calmer and communicative, and by the following morning he is largely settled. This is an *acute stress reaction*, a transient stress response that subsided once he was removed from the threat, not a disorder; the correct management was safety, orientation and support, not a benzodiazepine drip.

**GOOD TO REMEMBER**

- **Acute stress reaction (the syndrome):** the defining criterion is a transient, mixed, rapidly shifting emotional/autonomic/dissociative reaction beginning within minutes of an *exceptional* stressor and resolving fast (within hours to 1 to 3 days, typically under 48 hours); the discriminator is that it is judged *normal given the severity of the stressor* and is classified *outside* the mental disorders in ICD-11 (QE84, Chapter 24), so the first step is support and safety, not diagnosis or a drug.

### 24.3 Acute stress disorder: the DSM-5-TR criteria and the 9-of-14 rule

**The gateway and the threshold.** DSM-5-TR acute stress disorder requires the same Criterion A traumatic stressor as PTSD (exposure to actual or threatened death, serious injury or sexual violence, experienced, witnessed, learned of, or through repeated occupational exposure to aversive details), and then a symptom threshold that is distinctive: the presence of **nine (or more) of 14** symptoms drawn from *any* of five categories, all beginning or worsening after the trauma (DSM-5-TR 2022). The five categories are *intrusion*, *negative mood*, *dissociation*, *avoidance* and *arousal*. Because the nine can come from any mix of the five, the symptom profile of two people with the same diagnosis can look very different.

**The 14 symptoms, by category.** Intrusion (four): intrusive distressing memories, distressing dreams, dissociative flashback reactions, and intense/prolonged distress or marked physiological reactivity to reminders. Negative mood (one): a persistent inability to experience positive emotions. Dissociation (two): an altered sense of reality of self or surroundings (derealisation/depersonalisation), and inability to remember an important aspect of the event (dissociative amnesia). Avoidance (two): effortful avoidance of internal reminders (memories, thoughts, feelings) and of external reminders. Arousal (five): sleep disturbance, irritability or angry outbursts, hypervigilance, concentration problems, and exaggerated startle (DSM-5-TR 2022).

> **COMMON TRAP**
>
> Assuming acute stress disorder still *requires* dissociation. DSM-IV demanded a set number of dissociative symptoms, so ASD was historically taught as "the dissociative early trauma reaction". DSM-5 deliberately dropped that requirement: dissociation is now just *one of five* symptom categories, and the nine symptoms can be reached with none from the dissociation category at all (DSM-5-TR 2022; Kaplan & Sadock CTP 2024). Marking a survivor as "not ASD because they did not dissociate" is a DSM-IV-era error.

### 24.4 The time frames that separate acute stress disorder from PTSD

**The window is the whole point.** Acute stress disorder is a diagnosis of the *first month*. DSM-5-TR fixes the duration of the disturbance at **3 days to 1 month** after trauma exposure: symptoms typically begin immediately, but persistence for at least three days is needed before the label applies, and the diagnosis cannot outlive the first month (DSM-5-TR 2022). This is the mirror image of PTSD, whose duration criterion is *more than* one month. The two are, by construction, temporally exclusive: before the third day, neither applies; from three days to a month it is ASD; beyond a month it is PTSD.

**The floor and the ceiling.** The *floor* of three days exists so that the transient acute stress *reaction* of the first hours and days is not over-diagnosed as a disorder. The *ceiling* of one month exists because at that point the diagnostic question changes: if the full syndrome is still present after a month, the correct label is PTSD, and ASD is retired. A patient who is symptomatic at five weeks does not "still have ASD"; they have crossed into PTSD (or another diagnosis) (DSM-5-TR 2022; Tasman Psychiatry).

| FEATURE | ACUTE STRESS REACTION (ICD-11 QE84) | ACUTE STRESS DISORDER (DSM-5-TR F43.0) | PTSD |
|---|---|---|---|
| **Status** | **Not a mental disorder (Chapter 24, factors influencing health)** | **A diagnosable disorder** | A diagnosable disorder |
| **Owning system** | ICD-11 (also ICD-10 F43.0) | DSM-5 (no ASD exists in ICD-11) | Both DSM-5 and ICD-11 |
| **Onset** | Within minutes of the stressor | May begin immediately; label needs at least 3 days | Any time; delayed expression if first met after 6 months |
| **Duration** | Hours to 1 to 3 days (typically under 48 hours) | 3 days to 1 month | More than 1 month |
| **Symptom rule** | Transient, mixed, shifting; no operational count | 9 or more of 14 across 5 categories | DSM-5 four clusters (or ICD-11 three) |
| **Framing** | Normal response given stressor severity | Early diagnosable syndrome; imperfect predictor of PTSD | Established disorder of persistent re-experiencing |

The three labels placed on one timeline from the exceptional event outward; the load-bearing points are that ICD-11 acute stress reaction is a non-disorder, DSM-5 acute stress disorder occupies the 3-day-to-1-month window with a 9-of-14 rule, and PTSD takes over beyond a month (verified against ICD-11 CDDR, DSM-5-TR 2022 and Tasman Psychiatry).

**GOOD TO REMEMBER**

- **Acute stress disorder (the syndrome):** the defining criterion is a Criterion-A trauma followed by *9 or more of 14* symptoms from any of five categories (intrusion, negative mood, dissociation, avoidance, arousal), lasting *3 days to 1 month*; the discriminator from PTSD is purely the time window (ASD under a month, PTSD over a month) and from ICD-11 acute stress reaction is that ASD is a diagnosable *disorder*, while dissociation is no longer mandatory (contrast DSM-IV); the first step is to confirm the stressor, then count symptoms across the five categories.

## 24.5 The relationship to PTSD: who progresses, and the sensitivity problem

**The asd trauma pathway.** Acute stress disorder was introduced partly to flag people early on the road to PTSD, and it does carry predictive weight: roughly **half of those meeting ASD criteria go on to develop PTSD** (Tasman Psychiatry; Bryant et al. 2011). That is a clinically meaningful signal, and a positive ASD diagnosis in the first month should prompt monitoring and access to care.

**Why it is a poor screen.** The examinable subtlety is the other direction: *many people who develop PTSD never met criteria for ASD* in the first month (Tasman Psychiatry; Bryant et al. 2011). Some progress to PTSD from subthreshold early symptoms, and some show a delayed course. So ASD has reasonable positive predictive value but *poor sensitivity* as a screen for eventual PTSD: a clean first month does not rule out PTSD later, which is why guidelines favour active monitoring over reassurance-and-discharge for symptomatic trauma survivors (NICE 2018). ASD prevalence itself

varies by trauma type, estimated at about **13 to 25%** of motor-vehicle-accident survivors (Tasman Psychiatry).

**TRAP** **Treating absent ASD as reassurance.** A survivor who did not meet acute stress disorder criteria in the first month is *not* safe from PTSD; many who develop PTSD never had ASD. Do not discharge with false reassurance; use active monitoring with follow-up within a month for subthreshold symptoms (NICE 2018).

| | | | |
|---|---|---|---|
| **3 days to 1 month**<br>DSM-5 ACUTE STRESS DISORDER DURATION WINDOW | **9 of 14**<br>DSM-5 ASD SYMPTOM THRESHOLD (ACROSS 5 CATEGORIES) | **~50%**<br>OF ASD CASES PROGRESS TO PTSD | **under 48 hours**<br>TYPICAL DURATION OF AN ICD ACUTE STRESS REACTION |

### 24.6 Early management: what to do, and the benzodiazepine and debriefing traps

**Support first, drugs rarely.** The evidence-based early response is psychological first aid, safety, orientation, practical support and reconnection, with active monitoring and follow-up within a month for anyone with subthreshold symptoms (NICE 2018). Where the early syndrome is established, an *individual trauma-focused CBT* intervention is the treatment of choice, not a drug (NICE 2018). Two things are explicitly *not* to be done routinely: single-session psychological debriefing is ineffective at preventing PTSD and may increase the risk of developing it (BAP 2014; NICE 2018; Companion), and drug treatments, including benzodiazepines, should not be offered to *prevent* PTSD after trauma (NICE 2018).

**The benzodiazepine point, stated twice over.** A benzodiazepine is the wrong reflex in the acute aftermath for two separate reasons: it treats none of the core trauma symptoms, and early post-trauma benzodiazepine use may actually *increase* the risk of subsequently developing PTSD (CANMAT 2014). Where a specific peri-trauma preventive is considered at all, the guideline-supported options are trauma-focused CBT, or, more tentatively, propranolol (Ciplar, Inderal) or sertraline, never an early anxiolytic (BAP 2014). For established PTSD later, prazosin (Minipress, Prazopress) is the anchor for trauma nightmares (CANMAT 2014), but that belongs to the management of the persistent disorder, not the acute reaction.

**GOOD TO REMEMBER**

- **Early trauma management (the principle):** the mechanism is that recovery is the norm, so the first-line move is psychological first aid plus active monitoring, with individual trauma-focused CBT if an early syndrome is established (NICE 2018); the signature caution is that routine single-session debriefing may *increase* PTSD risk and a benzodiazepine both fails to treat trauma symptoms and may raise PTSD risk (CANMAT 2014), so the one thing not to do is sedate-and-debrief.

### 24.7 The India device: the treatment gap, benzodiazepine over-prescription and the IPS line

**Where this lands in Indian practice.** India carries a wide anxiety, OCD and PTSD treatment gap, and the early trauma window is exactly where it shows. Trauma is common (road-traffic and industrial accidents, disasters, interpersonal and domestic violence), distress after it is frequently somatised, and the reflex in over-stretched settings is a short course, then a long course, of a

benzodiazepine, usually alprazolam. Against that benzodiazepine over-prescription problem, the acute stress reaction is precisely the presentation that should *not* be medicalised: it is a transient normal response, best met with support and safety, and any benzodiazepine given past marked acute agitation risks doing harm rather than good (CANMAT 2014). The Indian Psychiatric Society CPGs, in line with NICE and BAP, put psychological first aid and active monitoring first, trauma-focused psychotherapy for established syndromes, and reserve pharmacotherapy for the persistent disorder, where the anchors are the SSRIs and the SNRI, with prazosin (Minipress, Prazopress) for nightmares and no maintenance benzodiazepine.

**INDIA**

The highest-yield Indian point: an acute stress reaction is a *non-disorder* that needs support, not a prescription, yet it is a common trigger for the benzodiazepine over-prescription seen across Indian practice. Set against India's wide trauma treatment gap, the IPS-aligned answer is psychological first aid and active monitoring first, trauma-focused CBT for an established early syndrome, and an SSRI (not alprazolam) only if the illness persists into PTSD, with prazosin (Minipress, Prazopress) reserved for nightmares.

**PEARL**

Line the whole area up on one clock and it becomes un-confusable. Minutes-to-hours after the event, mixed and settling: acute stress reaction (a normal response, not a disorder, ICD-11 QE84). Three days to a month, 9 of 14 symptoms: acute stress disorder (a DSM-5 diagnosis; ICD-11 has none). Beyond a month, the cluster syndrome: PTSD. Reaction, disorder-in-the-month, disorder-past-the-month, in that order.

**HOLD THIS**

Acute stress reaction is a *transient normal response* to an exceptional stressor, coded QE84 in ICD-11 *outside* the mental disorders (Chapter 24), beginning within minutes and settling within hours to 1 to 3 days (under 48 hours); acute stress disorder is a *DSM-5 diagnosable disorder* requiring a Criterion-A trauma plus 9 or more of 14 symptoms across five categories (intrusion, negative mood, dissociation, avoidance, arousal) lasting 3 days to 1 month, with dissociation no longer mandatory (unlike DSM-IV); ICD-11 has no acute stress disorder at all; about half of ASD cases progress to PTSD but many who develop PTSD never had ASD (poor sensitivity); and the acute management is psychological first aid plus active monitoring, never routine debriefing and never a benzodiazepine, which may raise PTSD risk.

## 25 · Adjustment disorder

**Adjustment disorder** is the diagnosis for a **maladaptive reaction** to an identifiable life event that is more than an expectable upset but less than a full syndrome such as a depressive episode or generalised anxiety disorder. The whole examinable tension sits in that middle ground: the reaction is real and can carry genuine morbidity (including suicide risk), yet the same clinical picture, in a slightly milder form, is simply *normal human distress* that must *not* be labelled a disorder. Getting the frame right, that a stressor is necessary but not sufficient, is exactly what a viva examiner probes.

**Why it matters clinically.** Adjustment disorder is one of the most frequently used diagnoses in consultation-liaison, primary-care and emergency settings, and it is also one of the most easily misused. The icd-11 changes reconceptualised it around two positive features, preoccupation with the stressor and a *failure to adapt*, moving it away from being a pure residual "not-quite-anything-else" category. The examinable traps run in two opposite directions at once: over-diagnosing a normal, self-limiting adaptive reaction as a disorder, and under-recognising that a genuine adjustment disorder carries a real, impulsive self-harm risk that can be greater per-episode than in major depression. The amygdala and fear-circuit anatomy that underpins the stress response is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; the wider CBT and problem-solving model that anchors treatment belongs to the Psychotherapies, clinical assessment, MSE and nosology notes and is not re-derived here.

### 25.1 The core concept: a maladaptive reaction bounded by a stressor at one end and another disorder at the other

**The sine qua non.** A precipitating stressor is the one non-negotiable requirement: adjustment disorder is the development of emotional or behavioural symptoms *in response to* an identifiable psychosocial stressor (Kaplan & Sadock CTP 2024). The stressor can be single (a relationship ending, job loss, a new medical diagnosis) or multiple, acute or continuous, and of *any* severity, it need not be extreme or life-threatening (DSM-5-TR 2022; ICD-11 CDDR). Unlike almost every other DSM-5-TR disorder, there is *no* defined symptom checklist; the diagnosis is defined by its relationship to the stressor, not by a signature symptom profile.

**Bounded on both sides.** The diagnosis is squeezed between two thresholds. At the lower boundary it must be more than normality: a **maladaptive reaction** with marked distress out of proportion to the stressor, *or* significant functional impairment (DSM-5-TR 2022). At the upper boundary it must be less than another disorder: if the picture meets criteria for a depressive episode, an anxiety disorder, PTSD or acute stress disorder, that diagnosis is made *instead*, and adjustment disorder is not given (ICD-11 CDDR; DSM-5-TR 2022). This is why it has historically read as a residual category, the diagnosis you reach when the reaction is clearly abnormal but fits no better-defined box.

RULE **Stressor necessary, but never sufficient.** No identifiable stressor, no adjustment disorder; but a stressor plus a full syndrome (depressive episode, an anxiety disorder, PTSD) means you diagnose *that* disorder, not adjustment disorder.

### 25.2 The ICD-11 reconceptualisation: preoccupation and failure to adapt as the positive core

**From residual to defined.** The single most testable point of the icd-11 changes is that adjustment disorder was given a *positive* core rather than being left as a leftover category. ICD-11 defines it as a **maladaptive reaction** to an identifiable psychosocial stressor whose two defining features are preoccupation with the stressor (excessive worry, recurrent distressing thoughts about the stressor, or constant rumination about its implications) and a *failure to adapt* that

produces significant impairment (ICD-11 CDDR). Symptoms of preoccupation characteristically worsen with reminders of the stressor and can drive avoidance of those reminders.

**What this changes in practice.** Because the core is now preoccupation-plus-failure-to-adapt, ICD-11 does *not* require the old affective subtypes: depressive and anxiety symptoms are listed as common *additional* features, not as the defining substance (ICD-11 CDDR). Two further ICD-11-specific anchors are examinable. First, ICD-11 tightens the onset window: symptoms usually emerge **within one month** of the stressor, tighter than the DSM-5-TR three-month window (ICD-11 CDDR; Kaplan & Sadock CTP 2024). Second, ICD-11 explicitly places adjustment disorder inside the grouping of "disorders specifically associated with stress" (code **6B43**) alongside PTSD, complex PTSD and prolonged grief disorder, all of which require a stressor by definition.

**PEARL**

The ICD-11 mnemonic for the core is simply *preoccupation plus failure to adapt*. If a candidate can only name one ICD-11 change, name that the diagnosis is now built on those two positive features rather than being a residual "everything else after a stressor" bin, with onset within one month rather than three.

**GOOD TO REMEMBER**

- **Adjustment disorder (the syndrome):** the defining criterion is a *maladaptive reaction to an identifiable psychosocial stressor* whose ICD-11 core is *preoccupation with the stressor* (worry, recurrent distressing thoughts, rumination) plus a *failure to adapt* causing significant impairment; the discriminators are that a stressor is *necessary* (below) and that it must not meet criteria for any better-defined disorder (above); the first step is to establish the stressor, confirm the reaction is out of proportion or impairing, and exclude a full depressive/anxiety/PTSD syndrome (ICD-11 CDDR; DSM-5-TR 2022).

### 25.3 The time frame: the onset window and the 6-month resolution rule

**Onset and resolution.** The temporal architecture is heavily examined. DSM-5-TR requires onset **within 3 months** of the stressor; ICD-11 tightens this to usually **within one month** (DSM-5-TR 2022; ICD-11 CDDR). Both systems agree the exit rule: once the stressor *or its consequences* have terminated, symptoms resolve **within 6 months** (DSM-5-TR 2022; ICD-11 CDDR). If symptoms outlast the stressor by more than six months, the diagnosis should generally be changed to a more appropriate one.

**Acute versus persistent.** DSM-5-TR carries two duration specifiers: *acute* (symptoms under six months) and *persistent (chronic)* (six months or longer). The persistent specifier applies only when the stressor itself is ongoing or has enduring consequences, a chronic disabling illness, or the lasting financial and emotional sequelae of a divorce, so that the diagnosis can legitimately run longer than six months *because the stressor has not actually ended* (DSM-5-TR 2022). The rule is not contradicted: what resolves within six months is the reaction after the stressor terminates.

| FEATURE | ICD-11 (6B43) | DSM-5-TR |
|---|---|---|
| **Onset after stressor** | **Usually within one month** | **Within 3 months** |
| **Defining core** | Preoccupation with the stressor + failure to adapt | Emotional/behavioural symptoms, marked distress out of proportion or functional impairment |
| **Resolution** | Within 6 months of stressor/consequences ending | Within 6 months of stressor/consequences ending |
| **Subtypes / specifiers** | No affective subtypes; depression/anxiety are additional features | Five specifiers; plus acute vs persistent (chronic) |
| **Relation to other disorders** | Not diagnosed if another disorder accounts for symptoms | Must not meet criteria for another disorder; not merely an exacerbation of a pre-existing one |

The two classifications side by side; the load-bearing contrasts are the tighter one-month ICD-11 onset window versus the DSM-5-TR three-month window, the shared six-month resolution rule, and ICD-11's positive preoccupation-plus-failure-to-adapt core replacing the DSM affective subtypes (verified against ICD-11 CDDR and DSM-5-TR 2022).

### 25.4 The DSM-5-TR subtypes: six specifiers by predominant symptom

**The five plus one.** Where ICD-11 dropped subtypes, DSM-5-TR retains a specifier for the *predominant* disturbance (DSM-5-TR 2022): *with depressed mood* (F43.21, low mood, tearfulness, hopelessness); *with anxiety* (F43.22, nervousness, worry, jitteriness, or in children separation anxiety); *with mixed anxiety and depressed mood* (F43.23); *with disturbance of conduct* (F43.24, e.g. rule-breaking, reckless behaviour); *with mixed disturbance of emotions and conduct* (F43.25); and *unspecified* (F43.20) for maladaptive reactions not classifiable as one of the above. The most common subtypes in adults carry prominent depressive symptoms.

**Why the conduct subtypes matter.** The conduct and mixed-conduct subtypes are the examinable reminder that adjustment disorder is not only an affective category: in adolescents it may present as acting-out, risk-taking or a rise in substance use, and in children the preoccupation is often not verbalised but somatised (stomach pains, headaches) or shown as regression, oppositional behaviour, tantrums or increased clinginess (ICD-11 CDDR; DSM-5-TR 2022). The reactive attachment and disinhibited engagement disorders that also follow adverse early environments are covered in the Child behavioural, emotional and other disorders notes and the Child psychiatry: assessment, treatment, services and protection notes.

MNEMONIC

**DAMCU**

DSM-5-TR adjustment disorder specifiers: **D**epressed mood, **A**nxiety, **M**ixed anxiety and depressed mood, **C**onduct disturbance, mixed emotions-and-conduct (the sixth being **U**nspecified). Five named subtypes plus unspecified.

### 25.5 Do not over-diagnose: the boundary with a normal adaptive reaction

**The lower threshold is the hard part.** The central clinical skill, and the most tested single caution, is distinguishing a diagnosable disorder from an expectable, self-limiting response to a hard event. A precipitant plus some distress is *not* a disorder: emotional reactions to negative life events that are *not* markedly out of proportion and do *not* cause significant impairment should not be diagnosed as adjustment disorder (ICD-11 CDDR). Transient responses that settle within a few days do not warrant the label at all, and where a reaction is judged normal given the severity of an extreme stressor, ICD-11 directs you to the non-disorder category *acute stress reaction* (QE84) rather than any diagnosis (ICD-11 CDDR).

**The bereavement carve-out.** Ordinary grief is explicitly excluded: DSM-5-TR requires that the symptoms do not represent normal bereavement and are not better explained by *prolonged grief disorder* (DSM-5-TR 2022). Adjustment disorder may still be diagnosed after a death when the intensity, quality or persistence of the grief exceeds what age, cultural and religious norms would expect and the picture does not meet prolonged grief criteria, but the default assumption is that grief is normal. Over-medicalising life's difficulties, treating "problems of daily living" as illness because families, community and religious supports have thinned, is the error the diagnosis most invites (Kaplan & Sadock CTP 2024).

TRAP **Labelling normal distress a disorder.** A stressor plus understandable upset is *not* adjustment disorder unless the reaction is markedly out of proportion *or* functionally impairing; transient distress settling in days, and ordinary bereavement, are normal, not diagnoses.

### 25.6 The self-harm signal: why "minor" is a dangerous word here

**The other-direction error.** Set against the over-diagnosis trap is its mirror image, dismissing a genuine adjustment disorder as trivial. Several studies link adjustment disorder to significant suicidal ideation and behaviour that belies its "minor and self-limiting" reputation (Kaplan & Sadock CTP 2024). Among emergency-department self-harm presentations, patients with adjustment disorder made *more impulsive* acts, were more often associated with alcohol use, and reached self-harm *earlier* in the course of illness than patients with major depression; poisoning is the commonest method (Kaplan & Sadock CTP 2024). Subthreshold conditions can therefore carry high morbidity, including death.

**The clinical consequence.** This is why ongoing suicide risk assessment is written into management, and why acuity, not the size of the stressor, drives the decision to intervene. A rapidly escalating reaction with impulsivity and alcohol involvement warrants crisis intervention and case management on a short-term basis to preclude suicide attempts and, where necessary, to avoid hospitalisation (Kaplan & Sadock CTP 2024).

**HOLD THIS**

Adjustment disorder is a *maladaptive reaction to an identifiable stressor* with, in ICD-11, *preoccupation with the stressor plus failure to adapt* at its core; onset is within one month (ICD-11) or three months (DSM-5-TR) and it resolves within six months of the stressor ending; it requires a stressor but is trumped by any full disorder (depression, an anxiety disorder, PTSD) and must be more than normal distress or grief; treatment is *predominantly psychological* (supportive, problem-solving, CBT) with drugs only complementary and symptom-targeted, and, despite its "minor" reputation, it carries a real impulsive self-harm risk that mandates active suicide-risk assessment.

## 25.7 Management: the predominantly psychological approach

**Psychosocial strategies are the mainstay.** The guiding principle is that adjustment disorder is treated *predominantly psychologically*: understand the meaning of the stressor to the patient, minimise its day-to-day impact, and mobilise adaptive coping (Kaplan & Sadock CTP 2024). The evidence base is thin, and the first formal systematic review appeared only in 2018, but the psychotherapeutic options that have been used are supportive psychotherapy, cognitive-behavioural and behavioural approaches, *problem-solving* techniques, relaxation training and brief psychodynamic work. Treatment is often short-term, with crisis intervention and case management layered on where acuity or self-harm risk demands it. Notably, in a Cochrane meta-analysis, *problem-solving therapy* was the intervention that reduced the time for patients to return to work.

**The India device.** Adjustment disorder is a high-volume presentation in Indian consultation-liaison and primary-care practice, exactly the settings where the benzodiazepine over-prescription problem is most entrenched: the reflex to reach for alprazolam or another short-acting benzodiazepine for a distressed patient after a life event. That reflex is doubly wrong here, the disorder is predominantly psychological, and even short-term benzodiazepine use in adjustment disorder can produce withdrawal (Kaplan & Sadock CTP 2024). Against India's wide anxiety, OCD and PTSD treatment gap, and in line with the Indian Psychiatric Society clinical practice guidelines, the answer is psychological support and problem-solving first, with medication reserved and symptom-targeted, never a benzodiazepine as the default.

**INDIA**

The highest-yield Indian point: adjustment disorder is one of the commonest reasons a distressed patient after a life event is handed a benzodiazepine (typically alprazolam) in Indian outpatient and consultation-liaison practice. It is precisely the presentation that should *not* be medicalised by reflex: the mainstay is psychological (supportive work, problem-solving, brief CBT), and even short-term benzodiazepine use risks withdrawal. The IPS-aligned line is support and problem-solving first, and any drug used briefly and only for a specific symptom such as severe insomnia or anxiety.

**GOOD TO REMEMBER**

- **Psychological treatment (the principle):** the mechanism is to help the patient process the *meaning* of the stressor and mobilise adaptive coping, so the first-line move is a brief, predominantly psychological intervention, supportive psychotherapy, problem-solving therapy (the option shown to speed return to work) or brief CBT; the signature caution is that adjustment disorder carries a real impulsive self-harm risk, so ongoing suicide-risk assessment and, where acuity demands, crisis intervention are built in; the first-line indication is essentially every case, with drugs kept complementary (Kaplan & Sadock CTP 2024).

## 25.8 Pharmacotherapy: complementary, symptom-targeted, and benzodiazepine-sparing

**Where drugs fit.** Medication is a *complementary*, not a primary, treatment, and the evidence is limited: Stein's 2018 systematic review of adjustment-disorder pharmacotherapy could identify only 11 randomised trials (Kaplan & Sadock CTP 2024). The sensible practice is to target specific symptoms in the short run, for example treating severe insomnia or severe anxiety, and to use an antidepressant for moderate-to-severe depressive symptoms. SSRI agents (fluoxetine, sertraline, escitalopram, and the others) have been found useful for some subthreshold depressive symptoms and certain subtypes, but the evidence for improving depressive symptoms in adjustment disorder is genuinely scant, and psychosocial strategies remain the mainstay.

**The benzodiazepine problem.** Benzodiazepines carry particular caution: even short-term use in adjustment disorder can result in withdrawal, and the harms are magnified in older adults and those with a substance use disorder (Kaplan & Sadock CTP 2024). Comparator studies in the anxiety subtype found the non-benzodiazepine GABA-A modulator etifoxine (a European agent) at least as effective as, and better tolerated than, lorazepam or alprazolam, with less rebound anxiety on discontinuation, a useful teaching point on why a benzodiazepine is not the natural first drug even when anxiety predominates. The deeper anxiolytic and hypnotic pharmacology, benzodiazepines, buspirone (Buspin), pregabalin and dependence, is developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; the SSRI pharmacology and dosing sit in the Mood disorders: pharmacotherapy notes.

**COMMON TRAP**

Reaching for a benzodiazepine as the default treatment for adjustment disorder because the patient is anxious after a life event. It is not first-line, the disorder is predominantly psychological; and even short-term benzodiazepine use here can cause withdrawal, especially in older adults and those with substance use disorder (Kaplan & Sadock CTP 2024). Target the specific symptom briefly if you must, but the mainstay is psychological, not sedative.

| 1 month | 3 months | 6 months | 5 to 20% |
|---|---|---|---|
| ICD-11 USUAL ONSET WINDOW AFTER THE STRESSOR | DSM-5-TR ONSET WINDOW AFTER THE STRESSOR | RESOLUTION ONCE THE STRESSOR/CONSEQUENCES END | OF OUTPATIENT MENTAL-HEALTH CASES (PRINCIPAL DIAGNOSIS) |

**WORKED EXAMPLE**

A previously well woman in her forties, redeployed under sustained pressure during a public-health crisis, presents after a low-lethality overdose. She has no prior psychiatric history. Over the preceding weeks she has been unable to sleep, tearful at work and at home, withdrawing from family meals, and consumed by worry about the risk she poses to an immunosuppressed spouse, clear *preoccupation with the stressor* and a failing effort to adapt, but she does not meet full criteria for a depressive episode. This is an adjustment disorder. Management was crisis intervention in the emergency setting, then individual psychotherapy and a peer-support group over about six months, with an antidepressant added as a *complement* and planned to taper once the stressor eased, not a standing benzodiazepine. She reached full remission. The example carries the whole topic: preoccupation-plus-failure-to-adapt below the depressive-episode threshold, a real self-harm risk, and psychological treatment first with medication complementary.

## 26 · The neurobiology of trauma and fear conditioning

The **neurobiology of trauma** is the story of a normal survival-learning system pushed past its design limits by an overwhelming event. A trauma is, in learning terms, an intensely aversive experience that teaches the brain, fast and durably, that a set of previously neutral cues now predict catastrophe. The machinery that does this learning is the same amygdala-centred fear circuit that underlies ordinary anxiety, but in post-traumatic stress disorder (PTSD) the learning is too strong, the context that should contain it fails, the stress-hormone axis that should switch off the alarm is dysregulated, and the prefrontal brake that should teach safety again is too weak to do so. Examiners test this as a four-part story, and the candidate who can name the four parts in order, amygdala over-learning, hippocampal context failure, HPA-axis dysregulation and impaired prefrontal extinction, has the answer.

**Why it matters clinically.** The neurobiology is not decoration; it is the rationale for every effective treatment. Trauma-focused psychotherapy works because it drives **fear extinction** through the prefrontal cortex, the very process that is impaired; a beta-blocker was trialled to blunt the noradrenergic consolidation of the traumatic memory; prazosin dampens the noradrenergic hyperarousal that fires the nightmares. Two ideas anchor the whole answer. First, PTSD is a disorder of fear learning that will not extinguish, an over-consolidated fear memory in the **amygdala** that the prefrontal cortex cannot teach the brain to switch off. Second, the disorder is maintained, not merely triggered, so the neurobiology explains why symptoms persist for years after the threat is gone. The detailed fear-circuit anatomy and the receptor systems are developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; this topic applies that anatomy to trauma and refers to the accompanying figure for the fear-conditioning map.

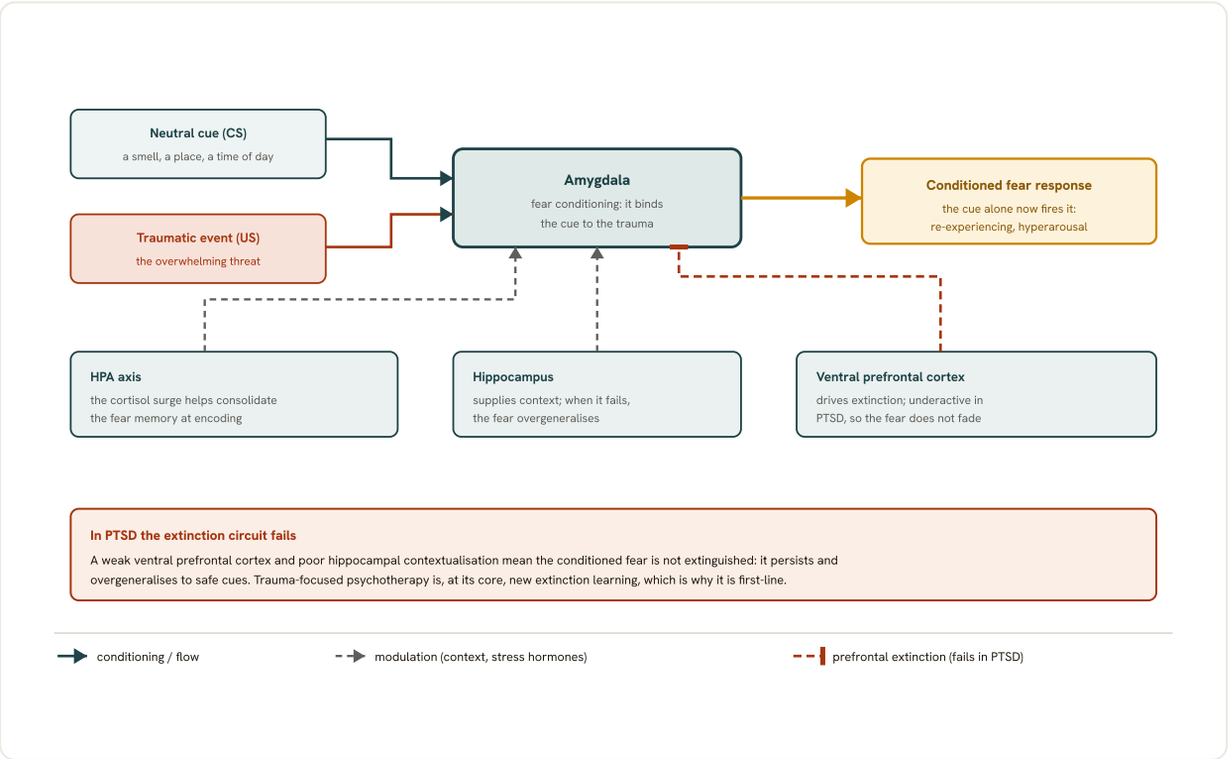



**Fig 14.19** The neurobiology of trauma: fear conditioning and its failed extinction. At the moment of trauma the amygdala binds a neutral cue (the conditioned stimulus) to the overwhelming event (the unconditioned stimulus), so that afterwards the cue alone fires the conditioned fear response, the re-experiencing and hyperarousal of PTSD. The HPA-axis cortisol surge helps consolidate that memory, and the hippocampus normally tags it with a safe context. Recovery depends on extinction, new learning driven by the ventral prefrontal cortex that the cue is now safe. In PTSD the prefrontal extinction circuit is underactive and the hippocampus contextualises poorly, so the fear neither fades nor stays tied to its original context but overgeneralises; trauma-focused psychotherapy works precisely because it is new extinction learning. The pathway anatomy is taught in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes.

## 26.1 Fear conditioning: how a neutral cue becomes a trauma trigger

**The learning paradigm to state first.** **fear conditioning** is Pavlovian associative learning applied to threat. In the laboratory paradigm an animal meets an intrinsically neutral cue, the conditioned stimulus (CS, for example a tone), paired repeatedly with an inherently aversive **unconditioned stimulus** (UCS, for example a shock); after conditioning, the cue that was paired with the shock (the CS+) alone provokes the defensive response, freezing and a heightened startle, while the cue never paired with shock (the CS-) does not (Kaplan & Sadock CTP 2024). The neutral cue has acquired threat value.

**The clinical translation.** This is exactly what trauma does. The horn, the smell of diesel, the particular light of the time of day, whatever neutral cue happened to be present at the moment of the trauma (the UCS), becomes a conditioned fear cue (the CS+) that thereafter fires the full alarm on its own. The intrusive re-experiencing and the cue-driven panic of PTSD are conditioned fear responses to CS+ cues laid down at the time of the event; naming the trauma as the UCS and the trigger as the CS+ is the cleanest way to explain a flashback in circuit terms.

**WORKED EXAMPLE**

A survivor of a road collision hears a car horn (a neutral cue, present at the crash) and is instantly flooded with terror, a pounding heart and a vivid sense of the impact. In conditioning language the crash was the UCS, the horn was a co-occurring cue that became the CS+, and the terror is the conditioned fear response the CS+ now evokes on its own. The lateral amygdala is where that horn-to-terror association was welded, and it is why an apparently trivial sound reliably reproduces the alarm.

### 26.2 The amygdala: where the trauma memory is welded

**The site of the plasticity.** The amygdala, specifically its lateral nucleus, is the convergence point where the conditioned cue (CS) and the aversive trauma (UCS) meet; single lateral-nucleus neurons receive both streams, and the strengthening of that synapse (long-term potentiation) is the physical substrate of the acquired fear (Kaplan & Sadock CTP 2024). The central nucleus is the output that broadcasts the fear response to the hypothalamus, brainstem and periaqueductal grey. In trauma this plasticity is excessive: an overwhelming UCS produces an over-consolidated, easily reactivated fear memory.

**The imaging signature.** The consequence is an amygdala set on a hair trigger. Functional imaging in PTSD shows amygdala *hyper*-responsivity to trauma-related and even to generally threatening or fearful stimuli, the neural correlate of the exaggerated startle and the sense of current threat (Kaplan & Sadock CTP 2024; New Oxford Textbook of Psychiatry). The amygdala is the hub whose over-reactivity the whole rest of the circuit is meant to, and in PTSD fails to, restrain.

**RULE** **PTSD is over-consolidated fear learning the brain cannot extinguish.** A hyper-responsive amygdala holds an over-strong trauma memory (excessive fear conditioning), a failing hippocampus lets the fear generalise out of its context, a dysregulated HPA axis fails to shut the alarm down, and a weak ventromedial prefrontal cortex cannot drive the extinction that would teach safety; the fear therefore persists.

### 26.3 The hippocampus: encoding the context, and why it fails

**Context is the hippocampus's job.** The hippocampus supplies the *context* of a fear memory, the where and when that tells the brain whether a cue is dangerous *here and now* or only in the original setting; it is central to contextual fear conditioning and to telling a safe context apart from a dangerous one (Kaplan & Sadock CTP 2024). Intact hippocampal gating is what normally keeps a conditioned fear tied to its proper situation.

**How the failure produces PTSD symptoms.** When hippocampal contextual processing fails, the fear *over-generalises*: cues that merely resemble the trauma, or occur in plainly safe settings, trigger the full response, which is the over-generalisation that characterises PTSD (Kaplan & Sadock CTP 2024; New Oxford Textbook of Psychiatry). The hippocampus is also uniquely vulnerable to the very stress hormones the trauma unleashes: the dentate-gyrus to CA3 system is delicately balanced, and chronic stress lasting longer than about three weeks impairs hippocampal-dependent function while it *enhances* amygdala-dependent fear conditioning, a

double hit that reduced hippocampal volume in PTSD may reflect (New Oxford Textbook of Psychiatry). A weaker context signal and a stronger fear memory pull in the same maladaptive direction.

PEARL

Read the amygdala and the hippocampus as a pair of opposing dials that trauma turns the wrong way. Stress *up*-regulates amygdala fear conditioning and *down*-regulates hippocampal contextual function at the same time. The result is a fear memory that is both too strong (amygdala) and untethered from its context (hippocampus), so it fires everywhere. That single sentence answers "why does PTSD generalise?"

### 26.4 The HPA axis: the stress-hormone response and its dysregulation

**The axis and its normal job.** The **hpa axis** (hypothalamic-pituitary-adrenal axis) is the body's neuroendocrine stress response: the amygdala drives the hypothalamic paraventricular nucleus to release corticotropin-releasing hormone, which drives pituitary ACTH, which drives adrenal cortisol; cortisol then normally feeds back to terminate the stress response (Kaplan & Sadock CTP 2024; New Oxford Textbook of Psychiatry). Cortisol's job is, in part, to switch the alarm *off* once the threat has passed.

**The dysregulation in PTSD, and its counter-intuitive direction.** The examinable, counter-intuitive fact is that PTSD is associated with *low* or normal, not high, basal cortisol, together with an *enhanced* negative-feedback sensitivity of the axis (an exaggerated cortisol suppression to dexamethasone), a pattern distinct from the high-cortisol picture of melancholic depression (Kaplan & Sadock CTP 2024). Because glucocorticoids are thought to facilitate extinction learning and to constrain the over-consolidation of a traumatic memory, an inadequate cortisol response at the time of trauma is one proposed reason the fear memory is laid down too strongly and extinguishes too poorly; consistent with this, giving stress-dose hydrocortisone around a major stressor has been trialled to reduce later PTSD, hypothesised to work by facilitating extinction and impairing consolidation of the traumatic event (New Oxford Textbook of Psychiatry). The HPA story is thus not "too much stress hormone" but a dysregulated axis that fails to do its off-switch and extinction-facilitating work.

TRAP **Assuming PTSD means high cortisol.** Unlike melancholic depression, chronic PTSD is classically associated with *low or normal* basal cortisol and *enhanced* glucocorticoid negative feedback (exaggerated dexamethasone suppression); writing "raised cortisol" is the reflex error that loses the mark. The maladaptive point is a failure of the axis to regulate and to facilitate extinction, not a simple excess.

### 26.5 Fear extinction: the safety learning that is meant to switch the alarm off

**What extinction is, and is not.** **fear extinction** is the learned reduction of a conditioned fear when the CS+ is presented repeatedly *without* the UCS, so the cue no longer predicts the threat (Kaplan & Sadock CTP 2024). The crucial point, and a favourite examiner distinction, is that extinction does *not* erase the original fear memory; it creates a new, competing "safety" memory that *inhibits* the fear response. Because the original memory persists, extinguished fears can

return (spontaneous recovery, renewal in a new context, reinstatement after a stressor), which is exactly why PTSD relapses under stress and why treatment is safety-learning rather than deletion.

**The prefrontal driver.** Extinction is driven by the ventromedial prefrontal cortex (the infralimbic region in the rodent), which acts through the amygdala's GABAergic intercalated cells to inhibit central-nucleus output; a strong ventromedial prefrontal brake means a fear that can be extinguished (Kaplan & Sadock CTP 2024). This is the mechanism that trauma-focused exposure therapy recruits: repeated exposure to trauma-related cues in a safe environment leads to extinction of the fear response, and agents such as D-cycloserine (an NMDA partial agonist) have been trialled to enhance that extinction learning (New Oxford Textbook of Psychiatry).

MNEMONIC

### A-H-H-E: the four-hit story of trauma

Run the neurobiology of trauma in four beats. **A**mygdala over-learns the fear (excessive conditioning, hyper-responsivity). **H**ippocampus loses the context, so fear over-generalises. **H**PA axis is dysregulated (low/normal cortisol, over-sensitive feedback, extinction not facilitated). **E**xtinction fails because the ventromedial prefrontal brake is too weak. Naming A-H-H-E in order is the fastest route to the "neurobiology of PTSD" marks.

## 26.6 Impaired prefrontal extinction: the lesion that maintains PTSD

**The core maintaining lesion.** The single circuit abnormality that best explains why PTSD *persists* is impaired prefrontal-mediated extinction. In PTSD the ventromedial prefrontal cortex is *hypo*-active and structurally reduced, while the amygdala is hyperactive; a weak prefrontal brake over a hot amygdala means the fear cannot be taught to switch off, and the conditioned response keeps firing to trauma cues long after any real danger has gone (Kaplan & Sadock CTP 2024; New Oxford Textbook of Psychiatry). Where ordinary recovery from a frightening event is spontaneous extinction, PTSD is the failure of that extinction.

**Why this is the treatment target.** This is why trauma-focused psychotherapy, not sedation, is the first-line treatment: exposure-based and cognitive trauma therapies work by driving successful extinction and safety learning through the prefrontal cortex, correcting the very process that is impaired. It is also why a benzodiazepine is the wrong tool, it sedates the alarm and can actually impair the extinction learning on which recovery depends, and why it has no place in PTSD treatment. The general exposure principle and the CBT model are developed in the Psychotherapies, clinical assessment, MSE and nosology notes; the licensed drugs, doses and the trauma-focused-first algorithm sit in the PTSD-treatment material.

**WORKED EXAMPLE**

Two soldiers survive the same ambush. Weeks later one no longer startles at a slammed door: repeated safe exposures let his ventromedial prefrontal cortex extinguish the conditioned fear. The other still dives for cover, has nightly nightmares and cannot enter a car park: his amygdala holds an over-consolidated fear his under-active prefrontal cortex cannot extinguish, his hippocampus has let the fear generalise to any enclosed space, and his HPA axis is not doing its off-switch work. The difference between recovery and PTSD is, in circuit terms, whether prefrontal extinction succeeds.

## 26.7 Noradrenaline and the consolidation of the traumatic memory

**The amplifier that also fixes the memory.** The noradrenergic system, driven by the locus coeruleus, does two things in trauma: it produces the peripheral hyperarousal (tachycardia, hypervigilance, exaggerated startle) that the patient feels, and, because noradrenaline released during a highly emotional event enhances the amygdala consolidation of that memory, it helps weld the traumatic memory in *more* strongly (Kaplan & Sadock CTP 2024; New Oxford Textbook of Psychiatry). A trauma is remembered vividly in part because it was encoded under a noradrenergic surge.

**The pharmacological corollary.** This gives two rational, noradrenergically targeted interventions. Peri-traumatic propranolol (Ciplar, Inderal), a beta-blocker, was trialled to blunt amygdala fear conditioning and the noradrenergic consolidation of the memory, though the evidence is insufficient to recommend it routinely for prevention (New Oxford Textbook of Psychiatry). And prazosin (Minipress, Prazopress), an alpha-1 antagonist, dampens the central noradrenergic hyperarousal that drives PTSD nightmares, which is why it is the targeted add-on for trauma-related nightmares. Both are the neurobiology cashed out as treatment; the prescribing detail sits in the PTSD-treatment material.

**GOOD TO REMEMBER**

- **Propranolol / prazosin (the noradrenergic handles on trauma):** noradrenaline released during trauma both drives the hyperarousal and strengthens amygdala consolidation of the memory; propranolol (Ciplar, Inderal), a beta-blocker, was trialled peri-traumatically to blunt that consolidation but is not routinely recommended for prevention, whereas prazosin (Minipress, Prazopress), an alpha-1 blocker, is the established add-on for trauma nightmares by lowering central noradrenergic tone, the signature caution for prazosin being first-dose hypotension.

## 26.8 Putting the trauma circuit together

**One loop, four failures.** The neurobiology of trauma is a single fear-learning loop failing at four nodes at once. The amygdala over-learns the fear (excessive conditioning, hyper-responsivity); the hippocampus loses the context, so fear generalises; the HPA axis is dysregulated and fails to facilitate the extinction and shut-down it should; and the ventromedial prefrontal cortex cannot drive the extinction that would teach safety. The result is a conditioned fear response, laid down under a noradrenergic surge, that fires to trauma cues indefinitely. Every effective treatment targets one of these nodes, and trauma-focused psychotherapy targets the most important, the failed prefrontal extinction.

| NODE / PROCESS | WHAT IT DOES NORMALLY | THE FAILURE IN TRAUMA / PTSD |
|---|---|---|
| **Amygdala (lateral nucleus)** | Welds the CS-UCS association; site of fear conditioning (LTP) | Over-consolidated fear memory; amygdala hyper-responsive to threat |
| **Amygdala (central nucleus)** | Broadcasts the fear response to hypothalamus, brainstem, PAG | Drives exaggerated startle and sense of current threat |
| **Hippocampus** | **Supplies the context of the fear memory** | Context fails, so fear over-generalises to safe cues; reduced volume |
| **HPA axis** | Cortisol terminates the stress response; facilitates extinction | **Low/normal cortisol, enhanced feedback; extinction not facilitated** |
| **Ventromedial prefrontal cortex** | **Drives fear extinction via GABAergic intercalated cells** | Hypo-active and reduced; extinction fails, fear persists |
| **Locus coeruleus / noradrenaline** | Arousal; enhances emotional memory consolidation | Hyperarousal and over-strong memory encoding; nightmares |
| **Extinction learning** | New safety memory inhibits the conditioned fear | **Impaired; the target of trauma-focused psychotherapy** |

The trauma fear-learning loop node by node, mapped to its normal role and its PTSD failure; the accompanying figure draws the fear-conditioning sequence (neutral cue paired with the trauma UCS becomes a CS+ that fires the amygdala, with the hippocampal context signal and the prefrontal extinction brake shown as the two systems that fail). Grounded in Kaplan & Sadock CTP 2024 and the New Oxford Textbook of Psychiatry.

## 26.9 Definitions to fix the vocabulary

### Fear conditioning

Pavlovian learning in which a neutral cue (CS), paired with an aversive event (UCS), acquires the power to evoke a fear response.

### Conditioned stimulus (CS+)

The cue that was paired with the trauma and now triggers the conditioned fear on its own; the CS- is the unpaired cue that does not.

### Unconditioned stimulus (UCS)

The intrinsically aversive event, the trauma itself, that gives the cue its threat value.

### Fear extinction

Learned reduction of a conditioned fear when the CS+ is met without the UCS; a new safety memory that inhibits, not erases, the fear.

### HPA axis

Hypothalamic-pituitary-adrenal stress axis (CRH to ACTH to cortisol); cortisol normally terminates the stress response and facilitates extinction.

### Contextual fear

Fear tied by the hippocampus to a specific setting; its failure produces the over-generalisation of PTSD.

**GOOD TO REMEMBER**

- **PTSD (the syndrome, in circuit terms):** the defining neurobiology is a conditioned fear memory over-consolidated in a hyper-responsive amygdala, stripped of its context by a failing hippocampus (fear over-generalises), against a dysregulated HPA axis (low/normal cortisol, enhanced negative feedback) and a hypo-active ventromedial prefrontal cortex that cannot drive extinction; the discriminator from ordinary recovery is that recovery *is* spontaneous extinction while PTSD is its failure, and the first therapeutic step is trauma-focused psychotherapy to re-drive prefrontal extinction (never a benzodiazepine, which impairs it).

**INDIA**

Trauma is abundant in India, road-traffic collisions, interpersonal and gender-based violence, disasters and displacement, yet PTSD is under-recognised and under-treated, folded into vague "tension" or somatic complaints and rarely reaching a psychiatrist; the trauma treatment gap is wide. Into that gap steps the country's **benzodiazepine over-prescription** problem: alprazolam and clonazepam are handed out freely, often long-term, for post-trauma distress and insomnia, which is precisely the wrong move on this circuit, because a benzodiazepine sedates the alarm and can *impair* the prefrontal extinction learning on which real recovery depends, while breeding dependence. The neurobiology gives the corrective the Indian Psychiatric Society CPG endorses: trauma-focused psychotherapy first (it drives the failed extinction), an SSRI or SNRI where a drug is needed, and prazosin (Minipress, Prazopress) for nightmares, with no role for a benzodiazepine. The correct answer treats the fear circuit, not merely the sleeplessness.

| 4 | 2 | 21 |
|---|---|---|
| FAILING NODES TO NAME (AMYGDALA, HIPPOCAMPUS, HPA AXIS, PREFRONTAL EXTINCTION) | TRAUMA-CONDITIONING TERMS (CS+ TRIGGER, UCS TRAUMA) | DAYS OF STRESS BEYOND WHICH HIPPOCAMPAL FUNCTION IS IMPAIRED AND AMYGDALA CONDITIONING ENHANCED |

**HOLD THIS**

PTSD is a fear-conditioning disorder: an overwhelming trauma (UCS) welds an over-strong conditioned fear to neutral cues (CS+) in a hyper-responsive amygdala, a failing hippocampus lets that fear generalise out of its context, a dysregulated HPA axis (low or normal cortisol, over-sensitive feedback) fails to facilitate extinction, and a hypo-active ventromedial prefrontal cortex cannot drive the fear extinction that would teach safety, so the alarm keeps firing, which is why trauma-focused psychotherapy (re-driving prefrontal extinction) is first-line and a benzodiazepine, which impairs extinction, is never used.

## 27 . Prolonged grief disorder

**A new diagnosis, and an examiner's favourite trap.** **Prolonged grief disorder** is the pathological grief syndrome added to DSM-5-TR (2022) and codified in ICD-11 (6B42). It sits in the trauma- and stress-related grouping alongside PTSD and adjustment disorder, and its whole diagnostic weight rests on one judgement: has grief persisted, and become impairing, *beyond* what this person's social, cultural and religious context would expect. The syndrome the DSM once parked in an appendix as **persistent complex bereavement disorder** is now a full disorder.

The examiner is rarely testing whether you can list the symptoms. They are testing whether you know the **time thresholds**, whether you can separate this from a depressive episode and from PTSD, and above all whether you resist the reflex to medicalise ordinary mourning. Grief is not a disease; only a grief response that is atypically prolonged, pervasive and disabling earns the label.

### 27.1 What the entity is

**Persistent grief.** Both classifications describe a **persistent and pervasive grief response** following the death of someone close, dominated by yearning or longing for the deceased, or by persistent preoccupation with them, and carrying intense emotional pain (sadness, guilt, anger, denial, difficulty accepting the death, emotional numbness, a sense that part of the self has died). This is **persistent grief** that has not moved through the normal arc of adaptation. The phrase to memorise is that the response **markedly exceeds expected social, cultural or religious norms** for that person's context.

### 27.2 The time thresholds (where marks are won and lost)

**Two systems, two numbers.** The single highest-yield discriminator is duration, and the two manuals differ. ICD-11 requires the response to have persisted for an atypically long time, giving a floor of at least **6 months** (longer in some cultural contexts). DSM-5-TR sets a higher adult bar: the death must have occurred at least **12 months** ago for adults (at least **6 months** for children and adolescents), and the symptoms must have been present nearly every day for at least the last month. Do not conflate the two.

| FEATURE | ICD-11 (6B42) | DSM-5-TR |
|---|---|---|
| **Minimum time since death** | **at least 6 months** | **12 months adult; 6 months child/adolescent** |
| **Core (one or both)** | yearning/longing OR preoccupation with the deceased | intense yearning OR preoccupation, nearly every day, last month |
| **Accompanying symptoms** | intense emotional pain features (not a fixed count) | at least 3 of 8 (identity disruption, disbelief, avoidance of reminders, intense emotional pain, difficulty reintegrating, emotional numbness, life feels meaningless, intense loneliness) |
| **Threshold clause** | exceeds cultural/religious norms; impairment | exceeds social/cultural/religious norms; distress or impairment |

The ICD-11 versus DSM-5-TR criteria for prolonged grief disorder; the duration cells are the ones examiners probe.

RULE **Duration is the discriminator.** ICD-11 says at least 6 months; DSM-5-TR says at least 12 months in adults. Anchor every answer to the number for the system being asked.

### 27.3 Do not pathologise normal grief too early

**The threshold is cultural, not just temporal.** Normal bereavement is protective, self-limiting and does not need psychiatric treatment. Both manuals build in a boundary-with-normality clause: a grief reaction within the normative period for the person's cultural and religious context

is *normal*, and it is often decisive to ask whether family, friends and community who share that perspective regard the duration or intensity as abnormal. Some traditions prescribe grieving that lasts a year or is deliberately reawakened at the first anniversary; a mourning ritual that runs its expected course is not a disorder.

TRAP **Labelling grief as disorder from the outset.** Applying the diagnosis before the time threshold, or against the person's own cultural norm, medicalises normal mourning; the community's reading of what is abnormal is part of the criterion.

### 27.4 Discriminating from depression and PTSD

**Circumscribed versus pervasive.** Prolonged grief disorder and a depressive episode share sadness, loss of interest, social withdrawal, guilt and suicidal ideation, but grief is *focused on the loss*: the yearning, the difficulty accepting the death, the sense that part of the self has died, feeling bitter about the loss. Depressive cognitions spread across many areas of life and pervasive low mood dominates. Against PTSD, the person with prolonged grief may be preoccupied with the circumstances of the death but does **not re-experience** them as happening again in the here and now; intrusive re-experiencing, flashbacks and hyperarousal point to PTSD. All three can co-occur, and each should be diagnosed where full criteria are met.

WORKED EXAMPLE

A widow, 14 months after her husband's death, still lays out his belongings untouched, longs for him nearly every day, cannot re-engage with friends and feels life is meaningless, yet finds joy in her grandchildren and has no pervasive anhedonia. The distress is circumscribed to the loss and the death is more than 12 months past: this meets DSM-5-TR prolonged grief disorder, not a depressive episode. Had she instead relived the moment of his death as if it were happening now, PTSD would move up the list.

### 27.5 Where it fits in the Indian setting

**An old idea with a new name.** Indian teaching has long distinguished normal grief from a **morbid grief reaction** (Gurmeet Singh's classification), where symptoms of grief are exaggerated or run prolonged beyond about 6 months without spontaneous recovery. Bowlby's three phases of response to loss, protest then despair then detachment, remain the standard developmental scaffold. The clinical caution matters in Indian practice: with the anxiety, OCD and PTSD treatment gap already leaving most common mental disorders untreated, scarce specialist time should not be spent converting culturally sanctioned mourning into a prescription, and the reflex to reach for a **benzodiazepine** for an acutely grieving person, a real over-prescription problem in Indian outpatient settings, is exactly the wrong move.

**INDIA**

Gurmeet Singh's normal-versus-morbid grief distinction predates the ICD-11 entity and is the classification most likely quoted by Indian examiners. Frame prolonged grief disorder as the formalised successor to morbid/pathological grief, hold the cultural-norm clause front and centre in a society with elaborate, lengthy mourning customs, and keep benzodiazepines off the acute-grief prescription.

### 27.6 Management: grief-targeted psychotherapy first

**Talk before pills.** Uncomplicated grief needs no drug. For prolonged grief disorder the evidence base points to a grief-targeted psychotherapy as first-line, most notably complicated grief treatment (the manualised approach developed by Shear), a structured therapy that combines processing the reality of the loss with re-engaging in life, and family-focused grief therapy in palliative and bereavement settings. Antidepressants are **not** routinely indicated for the grief itself: trials of SSRIs for grief have been largely negative, and drug treatment is reserved for a co-occurring depressive episode or another comorbid disorder diagnosed in its own right. If an antidepressant is used for comorbid depression, it is an SSRI (for example sertraline; Indian brands include the generic across many manufacturers), not a benzodiazepine.

**RULE** **Grief-targeted psychotherapy is the treatment; drugs treat comorbidity.** Complicated grief treatment first; reserve an SSRI for a diagnosed comorbid depression, and never a benzodiazepine for the grief.

| 6 | 12 | 3 of 8 | 9 to 25% |
|---|---|---|---|
| ICD-11 MINIMUM MONTHS SINCE DEATH | DSM-5-TR ADULT MINIMUM MONTHS | DSM-5-TR ACCOMPANYING SYMPTOMS | BEREAVED OLDER ADULTS DEVELOPING PGD |

**MNEMONIC**

**GRIEF-TOO-LONG**

**G**rief that is persistent and pervasive; **R**eaction exceeds cultural/**R**eligious norms; **I**ntense yearning or preoccupation is the core; **E**motional pain plus impairment; **F**ocused on the loss (not pervasive like depression, not re-experienced like PTSD); **TOO LONG** = past the threshold (6 months ICD-11, 12 months DSM-5-TR adult). The unpacking is the diagnosis.

**PEARL**

The term you will meet under several names points to one construct: **complicated grief**, morbid/pathological grief, persistent complex bereavement disorder (the old DSM-5 appendix name) and prolonged grief disorder (the current entity) are the historical layers of the same idea. When an examiner uses any of these, answer with the current ICD-11/DSM-5-TR criteria.

**GOOD TO REMEMBER**

- **Prolonged grief disorder (syndrome):** defining criterion is a persistent, pervasive grief response with intense **yearning** or preoccupation with the deceased that exceeds the person's cultural/religious norm; time floor is at least 6 months (ICD-11) or at least 12 months in adults (DSM-5-TR); the first discriminator against depression is that grief is circumscribed to the loss, and against PTSD that the death is not re-experienced in the here and now.
- **Complicated grief treatment (psychotherapy):** the grief-targeted, manualised first-line therapy (Shear); principle is to process the reality of the loss while restoring engagement with life; signature caution is that antidepressants do not treat the grief itself and are reserved for comorbid depression, never a benzodiazepine for acute grief.

**HOLD THIS**

Prolonged grief disorder = persistent yearning/preoccupation exceeding the cultural norm, at least 6 months (ICD-11) or at least 12 months in adults (DSM-5-TR) after the death; treat with grief-targeted psychotherapy, and do not pathologise normal mourning too early.

## 28 · Reactive attachment and disinhibited social engagement disorders

**Two disorders of early, severe neglect.** These are the trauma- and stressor-related disorders of the very young child. **Reactive attachment disorder** (RAD) is the emotionally withdrawn, inhibited pattern toward caregivers; **disinhibited social engagement disorder** (DSED, formerly disinhibited attachment disorder of childhood) is indiscriminate, over-familiar behaviour toward strangers. Both are named here briefly and criterion-led; the fuller developmental and services detail belongs to the child-psychiatry chapters. The exam hook is that both require the same aetiology, **early neglect** so severe it prevents normal attachment forming, yet they express in opposite behavioural directions.

Keep two nosological facts in front of you. First, DSM-5-TR and ICD-11 agree in splitting the old single "attachment disorder" into these two distinct disorders, both classed as trauma- and stressor-related, not as childhood-onset residuals. Second, both need a developmental age of at least **9 months** (the level at which selective attachment becomes possible) and onset before **5 years**. This note stays deliberately short: the point is the criterion, the discrimination, and the fact that treatment is psychosocial, never a drug.

### 28.1 Shared aetiology: pathogenic care

**Insufficient care is the gateway criterion for both.** Neither disorder is diagnosed without a documented history of a pattern of extremes of insufficient care: persistent social neglect or deprivation of basic emotional needs, repeated changes of primary caregiver that limit stable attachment (for example serial foster placements), or rearing in unusual settings with high child-to-caregiver ratios (institutions) that severely limit selective attachment. This "grossly insufficient care" is presumed causative, so the history of **early neglect** is not optional context, it is a criterion. Absent such a history the diagnoses are rare and, if made, are likely wrong.

### 28.2 Reactive attachment disorder: the withdrawn, inhibited child

**Minimal comfort-seeking and minimal response to comfort.** The child with RAD shows a consistent pattern of emotionally withdrawn, inhibited behaviour toward adult caregivers: rarely or minimally seeking comfort when distressed, and rarely or minimally responding to comfort when it is offered. Layered on this is a persistent social and emotional disturbance, reduced positive affect, and episodes of unexplained irritability, sadness or fearfulness even during non-threatening interactions. It is, in effect, the absence of an organised attachment where one should have formed. RAD is diagnosed only when the child does not meet criteria for autism spectrum disorder.

### 28.3 Disinhibited social engagement disorder: the over-familiar child

**Indiscriminate over-familiarity with unfamiliar adults.** The child with DSED actively approaches and engages unfamiliar adults with a striking lack of the normal stranger wariness that emerges between **7 to 9 months**. DSM-5-TR requires at least **two** of: reduced or absent reticence in approaching unfamiliar adults; overly familiar verbal or physical behaviour beyond age-appropriate and culturally sanctioned bounds; diminished or absent checking back with the caregiver after venturing away, even in unfamiliar settings; and willingness to go off with an unfamiliar adult with little or no hesitation. The behaviour is sometimes called "indiscriminate friendliness," but it is intrusive, boundary-crossing, and self-endangering rather than sociable.

### 28.4 RAD versus DSED versus their mimics

**Two discriminators carry the marks.** First, the direction of behaviour: RAD is under-social and withdrawn, DSED is over-social and disinhibited. Second, the exclusion pattern: **autism spectrum disorder** excludes RAD (both show diminished social reciprocity, but the ASD child has restricted, repetitive behaviours and a history without required neglect), whereas ASD does not exclude DSED. DSED must also be more than the behavioural impulsivity of **ADHD**, which may co-occur; the DSED behaviour is specifically social disinhibition toward strangers, not general impulsivity. The table alongside sets out the split.

| AXIS | REACTIVE ATTACHMENT DISORDER (RAD) | DISINHIBITED SOCIAL ENGAGEMENT DISORDER (DSED) |
|---|---|---|
| **Core behaviour** | **Emotionally withdrawn, inhibited toward caregivers** | **Indiscriminate over-familiarity with unfamiliar adults** |
| **Comfort** | Minimal seeking and minimal response to comfort | Reduced checking-back with the caregiver |
| **Required threshold** | Full withdrawn pattern plus social/emotional disturbance | **At least 2 disinhibited behaviours** |
| **Developmental age / onset** | At least 9 months; before age 5 | At least 9 months; before age 5 |
| **Autism spectrum disorder** | **Excludes the diagnosis** | **Does NOT exclude the diagnosis** |
| **Response to good care** | Signs typically recede within weeks of adequate caregiving | **May persist even after a stable attachment forms** |

Same neglectful root, opposite behavioural expression; the autism exclusion and the response to remediation are the two most-tested discriminators.

### 28.5 Course: why the two diverge under good care

**RAD tends to remit, DSED can stick.** This is the highest-yield prognostic point. In children moved from institutional to adequate caregiving (the Bucharest Early Intervention Project randomised foster care against continued institutional care), RAD signs typically recede within weeks of an adequate caregiving environment. DSED behaviours improve but can persist in a minority even after the child forms a selective attachment to a foster or adoptive parent, which is why some argue the core deficit in DSED is not simply the absence of an attachment relationship. Earlier placement helps: signs of DSED are not reported when deprivation begins only after age 2, and attachment disorders are very rare in children adopted before **6 months** of age.

### 28.6 Management: the intervention is the caregiving environment

**Change the care, not the child, and never a drug.** There is no established pharmacotherapy for RAD or DSED; the treatment is a stable, sensitive, individualised caregiving relationship, delivered by placing the child with a committed caregiver (foster or adoptive) and coaching that caregiver to respond to the child's distress as if it had been requested, so the child learns comfort is available. The clinician also protects the child (DSED carries real danger from going off with strangers). Critically, the coercive "attachment therapies", holding, restraint and rebirthing, are unproven and dangerous, having caused child deaths, and are formally discredited (AACAP practice parameter). This is the one point where the benzodiazepine over-prescription theme becomes a pure trap: there is nothing to prescribe here.

**RULE** **No neglect, no diagnosis.** A documented history of grossly insufficient care is a criterion for both RAD and DSED, not background colour; without it, look elsewhere.

**TRAP** **Do not reach for medication.** There is no drug treatment for RAD or DSED; prescribing a sedative or antipsychotic for the "difficult" neglected child treats the clinician's discomfort, not the disorder, and misses that the intervention is the caregiving environment.

**WORKED EXAMPLE**

A boy of nearly four, adopted at 30 months from an orphanage with rotating caregivers, is brought for "hyperactivity." He approaches and chats with complete strangers, has more than once followed a stranger willingly, tolerates injury with an oddly high pain threshold, and his warmth toward adults feels shallow rather than selective. Teachers find him charming yet attention-seeking. The history of institutional early neglect plus at least two disinhibited-approach behaviours, beyond ADHD-type impulsivity, gives disinhibited social engagement disorder. Management is protective and relational, focused on curbing self-endangering approach and rewarding age-appropriate social hesitance; his approach to strangers persists at a reduced, more manageable level.

**INDIA**

Institutional care of orphaned, abandoned and maltreated young children remains widely used across Asia, India included, which is exactly the pathogenic-care setting that produces RAD and DSED; child-protection and alternative-care law (the Juvenile Justice framework and CARA-regulated foster care and adoption) is the practical lever, since the treatment is the caregiving environment, not a prescription. This is also the sharpest place to name the **benzodiazepine over-prescription** problem by its absence: the temptation to sedate a "hyperactive" or "withdrawn" neglected child must be resisted, because no anxiolytic or antipsychotic treats an attachment disorder, and the wider Indian pattern of handing out clonazepam or alprazolam against the IPS Clinical Practice Guidelines has no foothold here at all.

| 9 mo | before 5 yr | 2 | 12 mo |
|---|---|---|---|
| MINIMUM DEVELOPMENTAL AGE FOR EITHER DIAGNOSIS | REQUIRED AGE OF ONSET | DISINHIBITED BEHAVIOURS NEEDED FOR DSED | PERSISTENCE FOR THE "PERSISTENT" SPECIFIER |

**MNEMONIC**

### IN versus OUT

**RAD turns IN** (withdrawn, inhibited, minimal comfort-seeking, and autism is excluded); **DSED turns OUT** (over-familiar, approaches strangers, checks back less, and autism is NOT excluded). Same neglect at the root, opposite direction of travel.

**PEARL**

The clean way to remember the prognosis split: RAD, being about the missing relationship, largely repairs when a good relationship arrives; DSED, being about missing stranger-wariness and inhibitory control, can outlast the arrival of a secure attachment.

**GOOD TO REMEMBER**

- **Reactive attachment disorder (RAD):** defining criterion is a consistent emotionally withdrawn, inhibited pattern toward caregivers, minimal comfort-seeking and minimal response to comfort, on a history of grossly insufficient care, developmental age at least 9 months and onset before 5 years; autism spectrum disorder excludes it, and the first step is to confirm the neglect history and check the child is not autistic.
- **Disinhibited social engagement disorder (DSED):** defining criterion is at least two disinhibited behaviours toward unfamiliar adults (over-familiarity, reduced reticence, diminished checking-back, willingness to go off with a stranger) beyond ADHD impulsivity, on the same neglect history and same age window; autism does NOT exclude it, and the discriminator from RAD is the outward, over-social direction of behaviour.
- **Caregiving intervention (the "therapy"):** the principle is that a stable, sensitive, individualised caregiving relationship is itself the treatment; there is no established medication, and coercive holding or "attachment" therapies are dangerous and discredited, so the first-line move is protective placement with a committed caregiver plus coaching of responsive care.

**HOLD THIS**
RAD (withdrawn, autism excluded) and DSED (over-familiar, autism not excluded) are the two trauma-related disorders of early neglect, both needing a developmental age of at least 9 months and onset before 5, and both treated by fixing the caregiving environment, never with a drug.

For the wider childhood picture and the child-protection and services detail, see the Child behavioural, emotional and other disorders notes and the Child psychiatry: assessment, treatment, services and protection notes; for the amygdala and fear-circuit anatomy touched on in the brain-imaging work, the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes.

## Apply and consolidate

### 29 · Worked cases

The anxiety, OCD and trauma syllabus rewards a candidate who can *reason* a case from the door to the discharge letter, not one who recites lists. This note is the arc closer: four **worked cases**, each taken as a **vignette** and reasoned aloud, so that the diagnostic discriminator, the criterion that clinches it, and the guideline-anchored management fall out in the right order. Read each as the examiner reads it, the story first, then "which disorder, and is it even a psychiatric disorder at all?", then "what, in what order, at what dose, for how long?".

**Why it matters, and how to use this note.** Every fact here is developed at depth elsewhere in the chapter; this note only assembles them into four reasoned walk-throughs and ends each named syndrome and each named drug or psychotherapy with an anchored Good-to-remember box. The Clark cognitive loop behind the first case lives in the Clark cognitive model note; the OCD algorithm behind the second in the OCD management note; the PTSD algorithm behind the third in the PTSD treatment note; and the organic-mimic grid behind the fourth in the anxiety differential note. The deep antidepressant pharmacology (SSRIs, SNRIs, clomipramine, doses, interactions) sits in the Mood disorders: pharmacotherapy notes; the anxiolytic and benzodiazepine pharmacology in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; the general CBT and exposure principle in the Psychotherapies, clinical assessment, MSE and nosology notes; and the deep brain stimulation option for refractory OCD in the ECT and neurostimulation notes. Each case refers to a *case:* line so you can find it fast on revision.

#### 29.1 Case one, the presentation: panic disorder with agoraphobia

**case: the vignette.** A 28-year-old teacher gives a several-month history of episodes that begin from a calm state with a pounding heart, breathlessness and a lurching certainty that she is having a heart attack; the surge crests within about **10 minutes** and subsides. Two cardiology work-ups have been normal. She now avoids crowded markets, buses and being far from home alone, in case an attack strikes where she "cannot get out or get help", and she carries an alprazolam tablet "just in case". **The reasoning.** The fear is of the *attacks themselves* (anticipatory

anxiety about the next surge), which names panic disorder; the fear of situations where escape is difficult or help unavailable names the comorbid agoraphobia. Normal cardiac work-up and the out-of-the-blue onset from calm exclude an arrhythmia driving it.

### 29.2 Case one, the criterion that clinches panic disorder and agoraphobia

**Panic disorder.** The defining criterion is *recurrent unexpected panic attacks* not restricted to a particular stimulus or situation, followed by persistent concern about further attacks or a maladaptive change in behaviour to avoid them (ICD-11 6B01; DSM-5-TR 2022). The word that does the work is *unexpected*: the attack arrives from a calm state, which is exactly what separates panic disorder from the cued surges of a phobia. **Agoraphobia.** The defining criterion is marked fear or anxiety about *multiple* situations where escape might be difficult or help unavailable should a panic-like or incapacitating symptom occur (crowds, public transport, open or enclosed spaces, being alone outside the home), consistently avoided or endured with dread (ICD-11 6B02; DSM-5-TR 2022). The focus is on escape and rescue, not on the place itself.

**GOOD TO REMEMBER**

- **Panic disorder (the syndrome):** defining criterion is *recurrent unexpected* panic attacks (from a calm state, not cued) plus persistent concern about further attacks or behavioural change to avoid them; the discriminator is the "out-of-the-blue" onset, and the first step is to exclude an organic mimic (arrhythmia, thyrotoxicosis) before the label (ICD-11 6B01; DSM-5-TR 2022).
- **Agoraphobia (the syndrome):** defining criterion is fear of *multiple* situations where escape is difficult or help unavailable (crowds, transport, open or enclosed spaces, being alone out), avoided or endured with dread; the discriminator is that the fear is of being unable to escape or get help, not of the place itself (ICD-11 6B02; DSM-5-TR 2022).

### 29.3 Case one, the formulation: Clark's cognitive model applied

**The loop, drawn on this patient.** Clark's model formulates her disorder as the *catastrophic misinterpretation of benign bodily sensations*: a benign internal trigger (a fleeting palpitation) is read as an imminent catastrophe ("heart attack"), which produces fear, which drives more autonomic arousal, which is again misinterpreted, tightening a self-amplifying vicious cycle until a full attack crests (the accompanying figure lays out the loop). Her carried alprazolam and her habit of sitting down and gripping a companion's arm are safety behaviours: they feel protective but prevent the belief from ever being *disconfirmed*, because she attributes surviving each attack to the prop rather than to the harmlessness of the sensation. That is the engine of chronicity and of the widening agoraphobic avoidance.

**GOOD TO REMEMBER**

- **Clark cognitive model (the formulation):** the mechanism is catastrophic misinterpretation of benign bodily sensations feeding a vicious cycle; the signature principle is that *safety behaviours* (the pocketed benzodiazepine, sitting down, gripping) block disconfirmation and maintain the disorder; the first therapeutic move is to drop the safety behaviour and let the sensation prove itself harmless (Clark 1986).

### 29.4 Case one, the management: an SSRI plus CBT, and the activation warning

**What the guidelines say.** First-line is an SSRI licensed for panic (escitalopram, sertraline, paroxetine or citalopram) or venlafaxine, *with* CBT built on Clark's protocol; combination of CBT and an antidepressant has the best outcomes (IPS 2025; NICE 2011; BAP 2014). CBT for panic is delivered in the optimal range of **7 to 14 hours**, typically weekly over about **4 months** (NICE 2011), using cognitive restructuring, interoceptive exposure and the dropping of safety behaviours. **The activation caution to state.** An SSRI started for panic must carry an explicit warning about *early activation* and a transient worsening of anxiety in the first days, because in Clark's terms the initial jitteriness supplies fresh bodily sensations to misinterpret; start low and titrate. A responder continues the drug for at least six months (BAP 2014). **The benzodiazepine is not the answer.** NICE explicitly advises *against* benzodiazepines in panic disorder, associated with a worse long-term outcome; her carried alprazolam is a chemical safety behaviour to be withdrawn, not the treatment.

**RULE** **SSRI plus CBT, never a standing benzodiazepine.** First-line panic disorder is an SSRI licensed for panic (escitalopram, sertraline, paroxetine, citalopram) or venlafaxine combined with Clark-protocol CBT; benzodiazepines are advised against because they worsen long-term outcome and act as a safety behaviour (NICE 2011; BAP 2014).

**TRAP** **Starting the SSRI without the activation warning.** Beginning an SSRI in panic disorder without warning of early activation invites the patient to misread the first-week jitteriness as "the tablets are giving me attacks", abandon treatment, and reach back for the benzodiazepine; start low, titrate, and forewarn from the outset.

**GOOD TO REMEMBER**

- **SSRI (first-line, panic disorder):** serotonin re-uptake inhibitor; the one first-line indication is an SSRI licensed for panic (escitalopram Nexito, sertraline Daxid/Zosert, paroxetine Paxidep, citalopram) with CBT; the signature caution is early activation, so start low, titrate, and warn from the outset; continue at least six months in a responder (BAP 2014; NICE 2011).
- **CBT for panic (first-line psychotherapy):** the mechanism is breaking the Clark vicious cycle by cognitive restructuring, interoceptive exposure and dropping safety behaviours; the one dose to quote is 7 to 14 hours over about 4 months; it is co-equal first-line with an SSRI and combination gives the best outcomes (NICE 2011; IPS 2025).

### 29.5 Case two, the presentation: OCD worked up the management algorithm

**case: the vignette.** A 26-year-old man has intrusive contamination thoughts he recognises as his own and as excessive, and washes his hands until they crack, spending four hours a day on rituals; his baseline Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) is 28, in the *severe* band. His wife has taken over "clean" tasks to keep the peace (family accommodation). **The reasoning.** Ego-dystonic, recognised-as-own intrusive obsessions plus repetitive neutralising compulsions that consume more than an hour a day and impair function name OCD; the Y-BOCS objectifies severity and will govern every rung of the algorithm. This is a *worked example* of the four-rung ladder.

GOOD TO REMEMBER

- **Obsessive-compulsive disorder (the syndrome):** defining criterion is persistent *obsessions* (intrusive, unwanted, recognised as one's own) and/or *compulsions* (repetitive behaviours or mental acts the person feels driven to perform) that are time-consuming (conventionally more than an hour a day) or cause marked distress or impairment; the discriminator from a psychotic disorder is preserved insight that the thoughts are excessive, and the first step is to rate the Y-BOCS (ICD-11 6B20; DSM-5-TR 2022).

### 29.6 Case two, rung one: a high-dose SSRI for a long trial, plus ERP

**The governing principle.** OCD is *under-treated by default* because the doses are timid and the trials cut short; the first rung is a high-dose SSRI for a long trial, together with CBT incorporating exposure and response prevention (ERP) (BAP 2014; IPS 2025). **Applied to him.** He is started on fluoxetine (Fludac, Prodep) and escalated to **60 mg** over about five to six weeks, the OCD target being roughly the 60 to 80 mg fluoxetine-equivalent, higher than the depression dose (IPS 2025; Maudsley). It is held at the maximum tolerated dose for a *long trial*: continue for at least **12 weeks** (about 10 to 12 weeks at ceiling, with at least 6 of those weeks at the maximum) before judging efficacy (IPS 2025; New Oxford Textbook). Alongside, therapist-guided ERP works up a graded hierarchy, exposing him to a "contaminated" object and preventing the washing ritual so anxiety habituates and the belief that the ritual averts catastrophe is disconfirmed; his wife's accommodation is assessed and reduced, since high accommodation predicts poor outcome (IPS 2025).

RULE **High dose, long trial, with ERP.** First-line OCD is an SSRI pushed to the maximum tolerated dose above the depression range (fluoxetine 60 mg equivalent) for about 10 to 12 weeks, combined with CBT incorporating exposure and response prevention; this is unanimous across BAP, IPS, NICE, CANMAT and WFSBP.

TRAP **Calling the SSRI a failure too early or at too low a dose.** An OCD trial is not adequate until roughly 10 to 12 weeks at the maximum tolerated dose, with at least 6 weeks at the ceiling; switching or augmenting on a depression-range dose stopped at six weeks mislabels a slow-but-real responder as treatment-resistant.

GOOD TO REMEMBER

- **Y-BOCS (the scale):** clinician-rated 10-item scale, each item 0 to 4, total **0 to 40**; bands 0-7 subclinical, 8-15 mild, 16-23 moderate, 24-31 severe, 32-40 extreme; response is a 25 to 35% reduction and remission a score of 12 or below; it is rated at baseline and every 4 weeks and governs every rung of the OCD algorithm (Goodman 1989; IPS 2025).
- **CBT with ERP (first-line psychotherapy, OCD):** the mechanism is graded exposure to the feared stimulus with prevention of the neutralising ritual, driving habituation and belief disconfirmation; the signature principle is that exposure and response prevention must be delivered *together* (either alone is inferior, Foa); the first-line indication is mild-to-moderate OCD as monotherapy and severe OCD combined with a high-dose SSRI (IPS 2025; CANMAT 2014).

### 29.7 Case two, the next rungs: switch, augment, then refer

**Reading the trial.** At 12 weeks his Y-BOCS is 25, under a 25% reduction, so despite an adequate high-dose long trial this is *non-response*. The dose is optimised toward 80 mg for a further period;

he remains a partial responder. **Rung two, switch.** The next step is to switch, either to a second SSRI or to clomipramine (Anafranil), the one tricyclic with genuine anti-obsessional efficacy and historically the first drug shown to work in OCD, at **150 to 225 mg/day** under ECG monitoring (IPS 2025; BAP 2014). Clomipramine sits at the margins of superiority over the SSRIs but is *less well tolerated* (anticholinergic burden, cardiac conduction effects, seizure risk in overdose), which is exactly why an SSRI is tried first and clomipramine held in reserve. **Rung three, augment.** On continued partial response, low-dose antipsychotic augmentation is added to the continuing serotonergic drug: risperidone **1 to 3 mg/day** (strongest augmentation evidence) or low-dose aripiprazole, continued for at least 8 weeks, with intensive ERP throughout (IPS 2025; BAP 2014). Antipsychotics are augmentation only, never monotherapy for OCD (NICE 2005). **Rung four, refer.** Only after this whole sequence, with each trial genuinely adequate, is he refractory; the refractory rung is intensive therapist-led ERP and, at a specialist centre such as NIMHANS, IV clomipramine then deep brain stimulation or ablative neurosurgery (cross-referenced to the ECT and neurostimulation notes).

| RUNG | STEP FOR THIS PATIENT | DOSE / DURATION |
|---|---|---|
| **1 First-line** | **High-dose SSRI (fluoxetine) + ERP** | 60 (up to 80) mg for at least 12 weeks at ceiling |
| **2 Switch** | Optimise dose, then another SSRI or clomipramine | Clomipramine 150 to 225, ECG-monitored |
| **3 Augment** | **Add low-dose risperidone or aripiprazole to the SRI** | Risperidone 1 to 3, at least 8 weeks |
| **4 Refer** | Intensive ERP, then IV clomipramine, DBS, neurosurgery | Specialist centre only |

The OCD management algorithm worked through case two; doses are bare numbers in the cells with the unit in the header (BAP 2014; IPS 2025; NICE 2005). The full ladder is drawn in the accompanying figure.

**GOOD TO REMEMBER**

- **Clomipramine (Anafranil):** the most serotonergic tricyclic and the original anti-obsessional drug; mechanism is potent serotonin (and noradrenaline) re-uptake inhibition; the one dose is 150 to 225 mg/day in OCD; the signature caution is poorer tolerability than an SSRI (anticholinergic, cardiac conduction, seizure in overdose) requiring ECG monitoring, so it is the switch after an adequate SSRI trial fails, not the first drug (BAP 2014; IPS 2025).
- **Antipsychotic augmentation (risperidone / aripiprazole, OCD):** low-dose dopamine blockade *added* to a continuing SSRI or clomipramine for treatment-resistant OCD; the one dose is risperidone 1 to 3 mg/day for at least 8 weeks (strongest evidence risperidone); the signature caution is that antipsychotics are augmentation only and never monotherapy for OCD (IPS 2025; NICE 2005).

### 29.8 Case three, the presentation: PTSD, and choosing psychotherapy first

**case: the vignette.** A 40-year-old lorry driver, six months after a fatal highway collision he survived, has nightly nightmares of the crash that feel as if it is happening again, cannot drive past the accident stretch, startles violently at horns and is convinced the world is now lethally dangerous. He asks for "something to calm the nerves". **The reasoning.** A qualifying trauma

(directly experiencing a life-threatening event, Criterion A) followed by re-experiencing *in the present*, avoidance of reminders and a persistent sense of current threat, all lasting more than one month, names PTSD (ICD-11 6B40; DSM-5-TR 2022). The pathognomonic feature is that the crash is re-lived here-and-now, not merely remembered, which is the line that separates PTSD from ordinary painful recollection.

**GOOD TO REMEMBER**

- **Post-traumatic stress disorder (the syndrome):** defining criterion is a qualifying trauma (Criterion A) followed by the ICD-11 triad, *re-experiencing in the present*, deliberate avoidance of reminders, and a persistent sense of current threat, lasting more than one month; the single discriminator is that the event is re-lived here-and-now (flashbacks, nightmares that feel current), not simply recalled; the first management step is to offer a trauma-focused psychotherapy, not a drug (ICD-11 6B40; DSM-5-TR 2022).

### 29.9 Case three, the management: trauma-focused therapy first, SSRI and prazosin considered

**The governing rule.** The single most examinable statement in PTSD management is that a trauma-focused psychotherapy, not a drug, is first-line: trauma-focused CBT (cognitive processing therapy, cognitive therapy for PTSD, narrative exposure, prolonged exposure) or EMDR, typically over **8 to 12** sessions, offered in preference to medication (NICE 2018; BAP 2014; VA/DoD 2023). Drugs are second, offered when psychotherapy is declined, unavailable or insufficient. **Applied to him.** He is told that trauma-focused psychotherapy would be the first choice; because he presses for medication, a licensed SSRI, sertraline (Daxid, Zosert) or paroxetine (Paxidep), or the SNRI venlafaxine, is appropriate, titrated to the maximum tolerated dose, with the warning that a therapeutic effect may take up to **12 weeks** to assess and a responder continues for at least **12 months** (BAP 2014; NICE 2018). Because trauma nightmares are prominent, prazosin (Minipress, Prazopress), a selective alpha-1 blocker, is *considered* as a targeted add-on for the nightmares, started at 1 mg nocte and titrated to **2 to 15 mg** nocte to avoid first-dose hypotension (CANMAT 2014; Maudsley). **The hard negative.** The "something to calm the nerves" he wants must *not* be a benzodiazepine: it treats none of the PTSD clusters, blunts the emotional engagement trauma-focused therapy needs, and may *increase* PTSD risk if given early after trauma (VA/DoD 2023; CANMAT 2014).

**RULE** **Trauma-focused therapy first; never a benzodiazepine.** In PTSD the first-line treatment is a manualised trauma-focused psychotherapy (TF-CBT or EMDR); where a drug is used it is a licensed SSRI (sertraline, paroxetine) or venlafaxine, with prazosin for nightmares; the benzodiazepine is excluded at every step (NICE 2018; VA/DoD 2023).

**GOOD TO REMEMBER**

- **Trauma-focused psychotherapy (first-line, PTSD):** the mechanism is deliberate processing of the traumatic memory so the event is refiled as past rather than relived as present (TF-CBT or EMDR); the one indication is that it is offered *before* drugs, typically over 8 to 12 sessions; the signature principle is that generic supportive counselling does not qualify, only memory-processing therapies (NICE 2018; VA/DoD 2023).
- **Sertraline / paroxetine (SSRI, PTSD):** the two SSRIs licensed for PTSD and the first-line drug once psychotherapy is set aside; mechanism is serotonin re-uptake inhibition; the signature caution is that a full trial takes up to 12 weeks and early activation can transiently worsen symptoms; continuation after response is at least 12 months (BAP 2014; NICE 2018).
- **Prazosin (Minipress / Prazopress, alpha-1 blocker):** the targeted add-on for PTSD trauma nightmares and sleep disturbance; mechanism is alpha-1 blockade lowering central noradrenergic tone; the one dose is 1 mg nocte titrated to 2 to 15 mg; the signature caution is first-dose hypotension, and it augments, never replaces, first-line psychotherapy or an SSRI/SNRI (CANMAT 2014; Maudsley).

### 29.10 Case four, the presentation: anxiety versus a medical mimic

**case: the vignette.** A 45-year-old man is referred with "treatment-resistant panic": episodic palpitations, pounding headache, drenching sweats and a surge of dread, several times a week, unresponsive to an SSRI. He drinks six cups of coffee a day and takes an over-the-counter decongestant. **The reasoning, in the right order.** Before any anxiety label, ask "is this organic?". The *tempo* is the pivot: these are *episodic, paroxysmal* autonomic storms, which mimic panic rather than GAD, and the pounding headache plus recorded blood-pressure spikes point away from a primary disorder. The work-up must clear the body first, and only then, if all negative, settle on a primary anxiety disorder. This is a *worked example* of "exclude the body before you name the disorder".

### 29.11 Case four, excluding the three mimics: hyperthyroidism, phaeochromocytoma, substance

**Hyperthyroidism, the sustained mimic of GAD.** Thyrotoxicosis reproduces the somatic tail of anxiety (irritability, tremor, tachycardia), but because the thyroid drive is *continuous* it mimics *sustained* GAD, not his episodic picture; still, it is the commonest organic mimic and is screened with thyroid function (a suppressed TSH with raised free T4), looking for goitre, exophthalmos, atrial fibrillation and weight loss with a preserved appetite (Shorter Oxford Textbook). **Phaeochromocytoma, the paroxysmal mimic of panic.** This catecholamine-secreting tumour releases adrenaline and noradrenaline in *bursts*, producing exactly his paroxysmal palpitations, pounding headache and sweating; because the secretion is episodic it mimics panic. The pointing feature is *paroxysmal hypertension*, blood pressure spiking with each attack, and the screening test is plasma or 24-hour urinary metanephrines and catecholamines; the discriminator is the recorded hypertensive surge and the headache, which panic disorder does not produce (Shorter Oxford Textbook; New Oxford Textbook). **The substance cause.** His six coffees and the sympathomimetic decongestant are a substance mimic: DSM defines caffeine intoxication as recent intake usually over **250 mg** with **five or more** autonomic and neuropsychiatric symptoms, and the discriminator is that the anxiety settles on cutting the load (DSM-5-TR 2022). Only with thyroid function, metanephrines and the substance history all clear would a primary panic disorder be diagnosed, and the SSRI failure re-read as a mislabelled organic case.

| MIMIC IN THIS CASE | POINTING FEATURE | SCREENING TEST |
|---|---|---|
| **Hyperthyroidism** | Goitre, exophthalmos, AF, weight loss with appetite (sustained, mimics GAD) | Thyroid function (TSH, free T4) |
| **Phaeochromocytoma** | **Paroxysmal hypertension, pounding headache (episodic, mimics panic)** | Plasma/urinary metanephrines, catecholamines |
| **Substance (caffeine / sympathomimetic)** | High caffeine load, decongestant, intoxication signs | Substance history; caffeine over 250 mg + 5 or more symptoms |

Case four, the three mimics excluded before settling on a primary anxiety disorder; the tempo (episodic vs sustained) tells you which physical test to send (Shorter Oxford Textbook; New Oxford Textbook; DSM-5-TR 2022).

**COMMON TRAP**

The exam-fatal error in case four is to accept "treatment-resistant panic" at face value and add a second psychotropic while an organic lesion sits in plain sight. Episodic autonomic storms with paroxysmal hypertension and a pounding headache are a phaeochromocytoma until proven otherwise; a high caffeine and decongestant load is a substance mimic; each is treated at its source, not with another SSRI or, worse, a benzodiazepine.

**GOOD TO REMEMBER**

- **Phaeochromocytoma (the mimic):** a catecholamine-secreting tumour producing paroxysmal palpitations, pounding headache and sweating that mimic panic; the discriminator is paroxysmal hypertension with a recorded blood-pressure surge in the attack; the screening test is plasma or urinary metanephrines and catecholamines; the first step is that episodic autonomic storms mean "exclude phaeochromocytoma" before a panic label (Shorter Oxford Textbook; New Oxford Textbook).
- **Hyperthyroidism (the mimic):** the sustained organic mimic of GAD (tremor, tachycardia, restlessness); the discriminator is the physical thyroid signs (goitre, exophthalmos, weight loss with appetite) and resolution on treating the thyroid; the screening test is thyroid function (suppressed TSH, raised free T4) (Shorter Oxford Textbook).

**INDIA**

All four cases sit inside the same Indian reality: a wide anxiety, OCD and PTSD treatment gap (these disorders are common, heavily somatised as palpitations, tremor and "gas", and under-recognised), and an entrenched benzodiazepine over-prescription problem, typically alprazolam, prescribed long-term in primary care against every guideline. Case one's carried alprazolam, case three's request to "calm the nerves", and case four's referral as "treatment-resistant panic" are each the habit in miniature; a large number of Indian patients labelled "anxiety" are actually in daily alprazolam micro-withdrawal, itself an iatrogenic mimic. The Indian Psychiatric Society clinical practice guidelines correct all of this, exclude the organic causes, treat the primary disorder with an SSRI or SNRI and a structured psychotherapy first, and keep the benzodiazepine a short, time-limited bridge rather than a standing prescription. Indian brands are available across the board, fluoxetine (Fludac, Prodep), sertraline (Daxid, Zosert), escitalopram (Nexito), paroxetine (Paxidep), clomipramine (Anafranil), prazosin (Minipress, Prazopress) and propranolol (Ciplar, Inderal), so the limiting factor is recognition and adequate dosing, not access.

| 10 to 12 weeks | 28 | 2 to 15 | 250 mg |
|---|---|---|---|
| ADEQUATE OCD SSRI TRIAL AT MAXIMUM TOLERATED DOSE | Y-BOCS OF CASE TWO (SEVERE BAND, 24 TO 31) | PRAZOSIN NOCTE RANGE, MG (START 1, TITRATE) | CAFFEINE INTOXICATION THRESHOLD (PLUS 5 OR MORE SYMPTOMS) |

MNEMONIC

### READ THE TEMPO, THEN THE FOCUS, THEN THE LADDER

Across the four cases: **READ THE TEMPO** (episodic storms mean exclude phaeochromocytoma, hypoglycaemia, a substance; sustained tremor with weight loss means thyrotoxicosis) → **THEN THE FOCUS** of the fear names the anxiety disorder (the next attack = panic; escape/rescue = agoraphobia; re-lived here-and-now = PTSD; ego-dystonic obsessions = OCD) → **THEN THE LADDER** (SSRI + CBT for panic; high-dose SSRI + ERP then switch/augment/refer for OCD; trauma-focused therapy first then SSRI + prazosin for PTSD), never a standing benzodiazepine.

PEARL

Every one of these four vignettes has the same wrong answer hiding in it, the benzodiazepine: the pocketed alprazolam in case one, the "calm the nerves" request in case three, the reflex add-on in case four's mislabelled panic. Name why it is wrong in each (safety behaviour in panic, harmful in PTSD, masking an organic lesion in the mimic case) and you have shown the examiner the single thread that runs through the whole anxiety, OCD and trauma management syllabus.

HOLD THIS

Work every anxiety, OCD or trauma case in the same order, exclude the organic mimic by tempo first (episodic storms = phaeochromocytoma/hypoglycaemia/substance, sustained = thyrotoxicosis), then name the disorder by the focus of the fear, then climb the guideline ladder (SSRI plus CBT for panic; high-dose SSRI plus ERP then clomipramine then risperidone/aripiprazole augmentation for OCD; trauma-focused psychotherapy first then a licensed SSRI plus prazosin for PTSD), and at no step reach for a standing benzodiazepine.

## 30 · Consolidation

The whole chapter now sits in front of you, and this closing topic is not new content but the machinery that turns reading into recall. The examination does not reward the candidate who has *read* the anxiety, OCD and trauma block; it rewards the one who can, cold and under pressure, state the discriminating criterion for each syndrome, the one first-line agent for each disorder, the OCD ladder in order, the scale bands to the number, and the PTSD algorithm in a single line. Those retrievals are what this topic drills. The single most robust finding in the science of learning is that **active recall** (pulling an answer out of an empty page) and **retrieval practice** (repeatedly testing yourself rather than re-reading) beat every passive method for durable memory, so the design here is deliberately answer-sparse: prompts first, and the answers held elsewhere in the chapter so you must fetch them.

**Why it matters, and what this note owns.** This note owns three things: the **mnemonic** that ties the chapter together, paid off from where it was seeded at the generalised-anxiety-disorder opener; a set of answer-free prompt lists for **active recall** across the anxiety disorders and their

first-line agents, the OCD ladder, the Y-BOCS bands, the PTSD algorithm and the trauma-disorder discriminators; and a whole-chapter **synthesis map** you are meant to **redraw from memory**. It re-derives no criteria, doses or bands of its own; every number quoted here is a pointer back to the topic that verified it. The deep pharmacology behind the drug names lives in the Mood disorders: pharmacotherapy notes and the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; the fear-circuit substrate in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; the general CBT and exposure model in the Psychotherapies, clinical assessment, MSE and nosology notes. The synthesis map that carries the whole block is drawn in the accompanying figure, framed cover-the-branches so it can be used as a retrieval test.

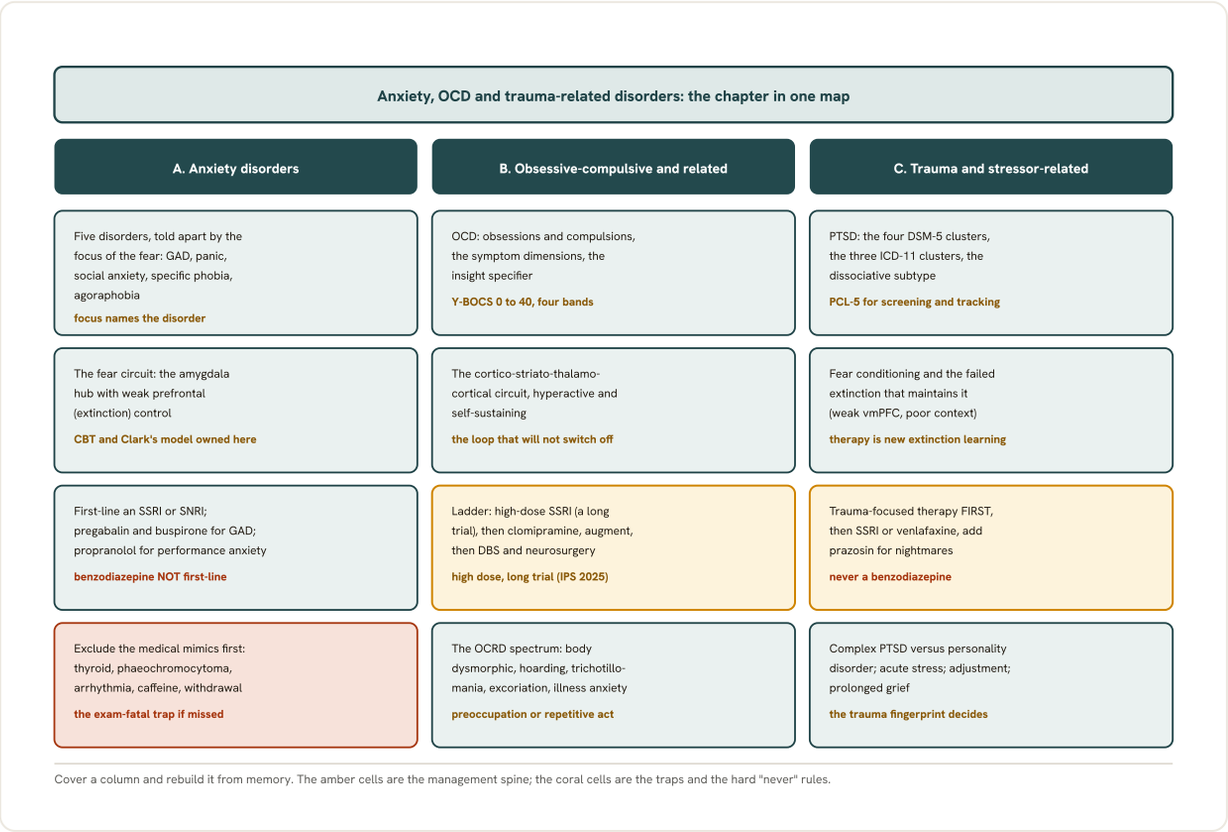



**Fig 14.20** The whole chapter in one synthesis map, built for retrieval. Three columns, one per band: the anxiety disorders (told apart by the focus of the fear, treated with an SSRI or SNRI, the medical mimics excluded first); the obsessive-compulsive and related disorders (OCD and its cortico-striato-thalamo-cortical circuit, the high-dose long-trial ladder, the OCRD spectrum); and the trauma and stressor-related disorders (PTSD and its clusters, the fear-conditioning neurobiology, the trauma-focused-therapy-first algorithm, and the complex-PTSD discrimination). The amber cells are the management spine and the coral cells the traps and the hard rules. Cover a column and redraw it from memory, then check it against the note.

## 30.1 The master mnemonic, paid off: WORRIED FEARS

**Seeded at the opener, closed here.** The chapter opened by seeding the master mnemonic **"WORRIED FEARS"** at generalised anxiety disorder, and this is where it is paid off, because its two halves are not really about GAD alone: they encode the *template* that repeats across the whole anxiety block. **WORRIED** is the anxiety *picture* (worry that is free-floating and uncontrollable, on-edge restlessness, rumination and poor concentration, relaxation made impossible by muscle tension, irritability, easy fatigue, disturbed sleep). **FEARS** is the anxiety *management* template:

**F**irst-line SSRI or SNRI, **E**valuate for a long enough trial before judging failure, **A**lternatives such as pregabalin and buspirone, **R**efer to CBT as a co-equal first-line, and **S**hun the benzodiazepine as long-term monotherapy. If you can expand both halves without looking, you hold the spine of the entire block.

MNEMONIC

### WORRIED FEARS

The chapter master mnemonic, paid off. **WORRIED** = the picture: **W**orry (free-floating, uncontrollable), **O**n edge/restless, **R**umination/poor concentration, **R**elaxation impossible (muscle tension), **I**rritability, **E**asily fatigued, **D**isturbed sleep. **FEARS** = the management template that repeats across the anxiety disorders: **F**irst-line SSRI/SNRI, **E**valuate over a long enough trial, **A**lternatives pregabalin and buspirone, **R**efer/CBT co-equal first-line, **S**hun the benzodiazepine long-term. Two further chapter mnemonics ride alongside it: **TIDRC** for the five Y-BOCS parameters, and **PTSD + A S R** for complex PTSD (the PTSD triad plus affect dysregulation, negative self-concept, relationship difficulty).

RULE **One template, many disorders.** For every anxiety disorder the default answer is SSRI (or SNRI) first-line plus CBT/exposure, with the benzodiazepine never the long-term monotherapy; learn the template once and vary only the twist (propranolol for performance, prazosin for nightmares, high-dose-long-trial for OCD).

## 30.2 Active-recall prompt list: the anxiety disorders and their first-line agents

**Cover the answers and pull each one.** This is a **retrieval practice** list, not a summary. For each disorder below, say aloud (or write) the one discriminating criterion and the first-line treatment before you check it against the topic that verified it. The point is the effort of retrieval: the harder the pull, the stronger the trace.

- **Generalised anxiety disorder.** Prompt: the discriminator from normal worry? The two numbers in DSM-5-TR? First-line drug and the two options? (Answers in the GAD topic: uncontrollable free-floating worry; **6 months**, 3 of 6; SSRI/SNRI first-line, pregabalin and buspirone as options.)
- **Panic disorder.** Prompt: what makes an attack diagnostic, and the one prescribing warning? (Recurrent *unexpected* attacks peaking within minutes plus a month of anticipatory anxiety; SSRI first-line started *low and slow* with a warning about early activation.)
- **Agoraphobia.** Prompt: the cognitive core, and is it tied to panic? (Fear that escape is difficult or help unavailable in at least two of five situations; a stand-alone disorder now, *irrespective* of panic disorder; exposure-based CBT core, SSRI for the panic component.)
- **Social anxiety disorder.** Prompt: the object of the fear, the one specifier, and the twist in treatment? (Fear of negative evaluation; the *performance-only* specifier; SSRI plus CBT for the generalised form, a situational beta-blocker for discrete performance anxiety.)
- **Specific phobia.** Prompt: the treatment of choice, and the drug trap? (Graded in-vivo exposure; a benzodiazepine is *not* first-line and blunts the extinction learning exposure

depends on.)

- **Separation anxiety disorder.** Prompt: the fear, and how it is easily missed? (Fear of separation from an attachment figure; presents as school refusal in a child or is sedated rather than diagnosed in an adult; exposure-based CBT, SSRI if needed.)

**GOOD TO REMEMBER**

- **The anxiety first-line map:** every one of GAD, panic disorder, agoraphobia, social anxiety and separation anxiety is SSRI (or SNRI) plus CBT/exposure first-line; specific phobia is *exposure-first* with drugs not first-line; the two disorder-specific twists are the situational beta-blocker (propranolol) for performance anxiety and starting the SSRI low-and-slow in panic; the benzodiazepine is never the long-term answer for any of them.

### 30.3 Active-recall prompt list: the OCD ladder and the trial rules

**Recite the staircase in order.** OCD management is a four-rung ladder governed by one idea, that OCD is under-treated by default because doses are timid and trials are cut short. Retrieve the rungs and the numbers before checking them against the OCD management topic.

- **Rung one.** Prompt: the first-line, and what makes it different from depression prescribing? (A *high-dose* SSRI for a *long* trial of about **10 to 12 weeks** plus CBT with exposure and response prevention; target roughly the 60 to 80 mg fluoxetine-equivalent.)
- **Rung two.** Prompt: what to do on partial or no response, and the classic contrast? (Optimise the dose, then switch SSRI or to clomipramine; the clomipramine-versus-SSRI point is that clomipramine sits at the margins of superiority but is *less tolerated*, so it is held in reserve, under ECG monitoring.)
- **Rung three.** Prompt: the augmentation step? (Low-dose antipsychotic augmentation, aripiprazole or risperidone, added to the continuing serotonergic drug for at least 8 weeks; augmentation only, never monotherapy.)
- **Rung four.** Prompt: the refractory options? (Intensive therapist-led ERP and, at a specialist centre only, intravenous clomipramine, then deep brain stimulation and ablative neurosurgery, cross-referenced to the ECT and neurostimulation notes.)
- **The judging rule.** Prompt: how is failure judged, and over what window? (By a less-than-25-to-35% fall on the Y-BOCS after a full high-dose trial of at least **12 weeks**, not by the patient's gestalt.)

**TRAP** **Calling an OCD trial a failure too early.** The commonest error is switching or augmenting before a high-dose SSRI has run about 10 to 12 weeks at the maximum tolerated dose, or judging on a depression-range dose; a slow-but-real responder is then mislabelled treatment-resistant and the ladder is climbed prematurely.

**GOOD TO REMEMBER**

- **The OCD ladder:** high-dose SSRI (about 60 to 80 mg fluoxetine-equivalent) for a long trial of roughly 10 to 12 weeks with ERP, then clomipramine (Anafranil) on failure, then aripiprazole or risperidone augmentation, then intensive ERP and specialist neurosurgery/DBS; judge response by a 25 to 35% Y-BOCS fall, continue a responder for at least 1 to 2 years, and the benzodiazepine has no place at any rung.

### 30.4 Active-recall prompt list: the Y-BOCS bands and the other scales

**The bands are pure memorisation, so drill them.** The scale numbers are the ones examiners ask for verbatim, so they reward the tightest retrieval practice. Cover the answers, recite each scale's structure and cut-off, and only then check against the scale topics. The instrument forms themselves are typeset centrally, so work from the accompanying scale plate rather than any pasted questionnaire.

| SCALE | WHAT IT MEASURES | STRUCTURE AND THE NUMBER TO HOLD |
|---|---|---|
| **Y-BOCS** | **OCD *severity* (not diagnosis)** | 10 items, each 0 to 4, total 0 to 40; bands subclinical 0 to 7, mild 8 to 15, moderate 16 to 23, severe 24 to 31, extreme 32 to 40; **16** is the trial-entry cut-off (floor of moderate); five parameters = TIDRC |
| **GAD-7** | GAD screen and severity | **7 items, each 0 to 3, total 0 to 21; bands 5/10/15; clinical cut-off 10; GAD-2 is the rapid screen** |
| **HAM-A** | Anxiety severity (clinician-rated) | 14 items, each 0 to 4, max **56**; psychic and somatic factors; the GAD drug-trial outcome measure |
| **PCL-5** | PTSD screen and monitor | **20 items, each 0 to 4, total 0 to 80; provisional cut-off 31 to 33 for probable PTSD; not a diagnosis (CAPS-5/SCID is)** |
| **LSAS** | Social anxiety severity | 24 items rating fear and avoidance, each 0 to 3; the social-anxiety trial outcome measure; Mini-SPIN is the rapid screen |

The five chapter scales and the verified numbers to recall cold; the Y-BOCS bands and the PCL-5 cut-off are the two most-asked, and each instrument's form is set out on the accompanying scale plate (verified against the individual scale topics).

| | | | |
|---|---|---|---|
| **0 to 40** | **10** | **31 to 33** | **10 to 12 weeks** |
| Y-BOCS TOTAL; MODERATE BAND FLOOR AT 16 | GAD-7 CLINICAL CUT-OFF (BANDS 5/10/15) | PCL-5 PROVISIONAL CUT-OFF FOR PROBABLE PTSD | ADEQUATE HIGH-DOSE SSRI TRIAL IN OCD |

**GOOD TO REMEMBER**

- **The five scales:** Y-BOCS (10 items, 0 to 40, cut-off 16, bands 0-7/8-15/16-23/24-31/32-40); GAD-7 (7 items, 0 to 21, cut-off 10); HAM-A (14 items, max 56, clinician-rated); PCL-5 (20 items, 0 to 80, cut-off 31 to 33); LSAS (24 items, fear and avoidance) are the block's instruments; the defining fact each time is that a scale *grades severity or screens*, it never makes the diagnosis.

### 30.5 Active-recall prompt list: the PTSD algorithm and the two hard rules

**State the ladder as one ordered line.** PTSD management is one of the cleanest algorithms in the syllabus, and it hangs off two rules that should be the first sentence of any answer: *trauma-focused psychotherapy is first-line, and a benzodiazepine is never used*. Retrieve the sequence before checking it against the PTSD treatment topic.

- **First-line.** Prompt: which treatment, and which therapies qualify? (A manualised **trauma-focused psychotherapy**, either TF-CBT (cognitive processing therapy, cognitive therapy for PTSD, narrative exposure, prolonged exposure) or EMDR, over about 8 to 12 sessions, ahead of any drug.)
- **First-line drug.** Prompt: when a drug is used, which agents? (The two licensed SSRIs sertraline and paroxetine, or the SNRI venlafaxine; allow up to **12 weeks** for a trial.)
- **The symptom-targeted add-on.** Prompt: for nightmares and disturbed sleep? (Prazosin (Minipress, Prazopress), an alpha-1 blocker, started at **1 mg nocte** and titrated to 2 to 15 mg, as an add-on, never monotherapy.)
- **Non-response.** Prompt: the sequence? (Raise the dose, then switch, then augment with an antipsychotic (risperidone, olanzapine) or combine drug plus psychotherapy and refer.)
- **The exclusion.** Prompt: the one drug class ruled out? (The *benzodiazepine*, at every step, ineffective for the core clusters and possibly raising PTSD risk if given acutely.)

TRAP **A benzodiazepine in PTSD, or as monotherapy for any anxiety disorder.** Benzodiazepines touch none of the PTSD clusters and blunt the emotional engagement trauma-focused therapy needs; the exam-fatal error is to offer one for PTSD, or to write one up as the long-term treatment for GAD, panic or phobia.

**GOOD TO REMEMBER**

- **The PTSD algorithm:** trauma-focused psychotherapy (TF-CBT or EMDR) first everywhere; if a drug is used, a licensed SSRI (sertraline, paroxetine) or venlafaxine over a trial of up to 12 weeks; prazosin (Minipress, Prazopress) added for nightmares; on non-response raise, switch, then augment with an antipsychotic; continue a responder at least 12 months; and never a benzodiazepine at any step.
- **EMDR / trauma-focused CBT (the psychotherapies):** both process the traumatic memory directly and are the co-equal first-line for PTSD and complex PTSD over about 8 to 12 sessions; the principle is that generic supportive counselling does *not* qualify, and complex PTSD often needs a phased approach (stabilisation before trauma processing).

### 30.6 Active-recall prompt list: the trauma-disorder discriminators

**The trauma cluster is discriminated by time and by structure.** The stressor-related disorders are separated mainly by *duration* and by the *shape* of the reaction, and examiners test the boundaries, not the lists. Retrieve each discriminator before checking it against the relevant topic.

| BOUNDARY | THE DISCRIMINATOR TO STATE |
|---|---|
| **Acute stress reaction vs acute stress disorder** | **ICD-11 acute stress reaction is *not a disorder* (QE84, a health-status factor, transient, hours to 1 to 3 days); DSM-5-TR acute stress disorder *is* a diagnosis (9 of 14 symptoms, 3 days to 1 month)** |
| **Acute stress disorder vs PTSD** | Purely the time window: acute stress disorder under a month; PTSD over a month |
| **PTSD vs complex PTSD** | **Complex PTSD = the full PTSD triad *plus* the three disturbances in self-organisation (affect dysregulation, negative self-concept, relationship difficulty)** |
| **Complex PTSD vs personality disorder** | **Trauma-reminder avoidance and current threat (absent in PD); a *stable* negative self-view (vs unstable); disconnection (vs abandonment-driven instability); less frequent but higher-lethality suicidality** |
| **Prolonged grief disorder** | Persistent yearning exceeding the cultural norm; at least 6 months (ICD-11) or at least 12 months in adults (DSM-5-TR); do not pathologise normal grief too early |
| **Adjustment disorder** | Maladaptive reaction to an identifiable stressor (ICD-11 core: preoccupation plus failure to adapt); onset within 1 month (ICD-11) or 3 months (DSM-5-TR); resolves within 6 months of the stressor ending; trumped by any full disorder |

The trauma-disorder discriminator grid: the block is separated mainly by duration and by whether the reaction is a normal response, a diagnosable disorder, or a personality pattern (verified against the acute-stress, PTSD, complex-PTSD, prolonged-grief and adjustment-disorder topics).

**GOOD TO REMEMBER**

- **Complex PTSD versus personality disorder (the discrimination that unlocks treatment):** both share affective dysregulation, but complex PTSD carries the trauma fingerprint, reminder avoidance and a persistent sense of current threat, a *stable* negative self-view, relationships marked by disconnection (not fear of abandonment), and higher-lethality suicidality; getting the label right is what preserves a survivor's access to trauma-focused therapy.

### 30.7 The whole-chapter synthesis map, framed cover-the-branches

**Build the tree, then redraw it blind.** The strongest single revision act for this block is to **redraw from memory** a **synthesis map** that hangs every disorder off its trunk. The map has three trunks: the *anxiety disorders* (carved by the object of the fear), *OCD and the related disorders* (the obsessive-compulsive spectrum), and the *stressor-related disorders* (carved by time and structure). From each trunk hang the disorders, and from each disorder hang its one discriminator, its scale

and its first-line treatment. The accompanying figure draws this map; use it as a retrieval test by covering all the branches and regenerating them, then uncovering to score yourself, exactly the cover-the-branches method that makes **retrieval practice** work.

**How to use it.** First pass: cover everything but the three trunks and name the disorders under each. Second pass: for each disorder, cover the discriminator and pull it. Third pass: cover the treatment column and recite the first-line agent and the one twist. A branch you cannot regenerate is a branch to re-read in its own topic; a branch you can is one you can leave. Repeated over spaced sittings, the map converts the chapter from something you recognise into something you can produce.

**PEARL**

The chapter collapses to three trunks and one template. Trunk one, the *anxiety disorders*, are carved by the object of the fear (diffuse worry, unexpected attacks, escape impossible, negative evaluation, a cued object, separation) and all answer to SSRI/SNRI plus CBT. Trunk two, *OCD and its spectrum*, run the high-dose-long-trial ladder governed by the Y-BOCS. Trunk three, the *stressor-related disorders*, are carved by time (reaction, acute stress disorder, PTSD) and by structure (complex PTSD, prolonged grief, adjustment), with trauma-focused psychotherapy first for PTSD. Cover the branches, redraw the tree, and you own the block.

**INDIA**

The Indian thread runs through every trunk and is worth carrying as one line at consolidation: a very wide **anxiety, OCD and PTSD treatment gap** (most sufferers never reach formal care, distress is somatised and under-recognised), into which has flowed the entrenched **benzodiazepine over-prescription problem** (typically long-term alprazolam or clonazepam given first-line against every guideline). The corrective is the same across the block and is exactly what the **Indian Psychiatric Society CPGs** say: an SSRI or SNRI first-line with CBT or exposure, the high-dose-long-trial rule enforced in OCD, trauma-focused psychotherapy first in PTSD, and the benzodiazepine reserved and time-limited, never the maintenance treatment. The first-line agents are cheap and familiar as Indian brands, escitalopram (Nexito, Rexipra), sertraline, fluoxetine and fluvoxamine, with clomipramine (Anafranil), propranolol (Ciplar, Inderal) for performance anxiety, and prazosin (Minipress, Prazopress) for trauma nightmares.

**HOLD THIS**

Redraw the three-trunk synthesis map from memory: anxiety disorders (carved by the object of the fear, all SSRI/SNRI plus CBT), OCD and its spectrum (high-dose SSRI for a long trial with ERP, then clomipramine, then antipsychotic augmentation, governed by the Y-BOCS), and the stressor-related disorders (carved by time and structure, trauma-focused psychotherapy first for PTSD, prazosin for nightmares), with the benzodiazepine excluded from long-term use across the whole block.

## Previously asked

This territory is examined at two levels at once: the syndrome (its clinical features, its differential diagnosis and its course) and the treatment (which first-line agent, the obsessive-compulsive-disorder ladder, the trauma-focused algorithm). The reliable pattern is that obsessive-compulsive disorder and panic disorder carry the most weight in the anxiety and related section, that post-traumatic stress disorder is the dominant trauma essay, and that a single disorder or a single treatment can be asked as a standalone note. Every long essay ends in a guideline-anchored management plan, so the first-line recommendation and the reason for it must be exam-ready. The genuine questions on record are grouped by band below. The anxiolytic and antidepressant pharmacology these disorders are treated with is developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes and the Mood disorders: pharmacotherapy notes; the fear-circuit anatomy is developed in the Neurochemistry, neuroendocrinology, neuroimaging and basic psychopharmacology notes; and the wider cognitive behaviour therapy technique is cross-referenced to the Psychotherapies, clinical assessment, MSE and nosology notes, while exposure and response prevention, trauma-focused therapy and Clark's model are owned here.

### HOW THIS TERRITORY IS ACTUALLY TESTED

- **The anxiety disorders, with panic and generalised anxiety leading.** "Describe various anxiety disorders. Discuss the clinical features and management of generalized anxiety disorder" has come as a full 25-mark essay, and panic disorder recurs at every length: "Discuss etiopathology, clinical features, differential diagnosis and management of panic disorder" as a 25-mark essay, "Differential diagnosis, clinical features and course of Panic disorders" as a 10-mark note, and "Panic disorder" and "Panic disorders" as standalone notes across several sittings. Prepare the anxiety-disorders differential (generalised anxiety versus panic versus social anxiety versus the phobias), the medical mimics that must be excluded, and the guideline-anchored first-line management for each. essay and short note, recurring

- **Obsessive-compulsive disorder, its management and its recent advances.** This is the most heavily examined disorder in the band: "Discuss etiopathogenesis, clinical features and management of obsessive compulsive disorders", "Discuss the etiology, clinical features and management of obsessive compulsive disorder", "Discuss the recent advances in the treatment of obsessive compulsive disorder" and "Discuss the nosological shift of OCD in DSM-5 and its spectrum" have all come as 25-mark essays, and "Management of OCD" recurs as a 10-mark note. Prepare the full management ladder (the high-dose serotonin reuptake inhibitor for a long trial, clomipramine, exposure and response prevention, antipsychotic augmentation and the refractory options), the cortico-striato-thalamo-cortical model and the obsessive-compulsive related spectrum. essay and short note, recurring

- **The obsessive-compulsive related disorders.** "Trichotillomania" has come as a standalone 10-mark note, and the obsessive-compulsive related disorders are drawn into the wider "impulse control disorders" essay. Prepare body dysmorphic disorder, hoarding, trichotillomania and excoriation as a compact spectrum, each recognised by its defining repetitive behaviour or preoccupation. short note, recurring

- **Post-traumatic stress disorder, its features and its evidence-based treatment.** "Describe the core symptoms and risk factors in PTSD. Discuss management strategies of PTSD" and "Discuss the etiopathogenesis, clinical features and management of post-traumatic stress disorder" have both come as 25-mark essays, and "Post traumatic stress disorder" recurs as a 10-mark note. Prepare the symptom clusters (the DSM-5 four and the ICD-11 three), the trauma-focused-psychotherapy-first algorithm with

the pharmacotherapy and prazosin behind it, and the rule that a benzodiazepine is not used. **essay and short note, recurring**

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Q **Acute stress, grief and the other trauma and stressor disorders.** "Acute Stress Disorder" (paired with chronic fatigue syndrome in one sitting), "Complicated grief" and "Grief" have each come as 10-mark notes across several years, and eye movement desensitisation and reprocessing ("EMDR") as a standalone note. Prepare the acute-stress-reaction-versus-disorder distinction, complex post-traumatic stress disorder and its separation from a personality disorder, prolonged grief disorder against normal grief, and EMDR as a trauma-focused therapy. **short note, recurring**

Prepare this material to be diagnosed and treated, not just recited: the anxiety-disorders and medical-mimic differentials, the scale cut-offs (the GAD-7, HAM-A, Y-BOCS and PCL-5 bands), the dose-anchored good-to-remember boxes, the obsessive-compulsive-disorder ladder and the trauma-focused algorithm in these notes are built to the way the block is examined. The anxiolytic and hypnotic pharmacology lives in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; the deep antidepressant pharmacology in the Mood disorders: pharmacotherapy notes; the fear-circuit anatomy in the Neurochemistry, neuroendocrinology, neuroimaging and basic psychopharmacology notes; deep brain stimulation and neurosurgery for refractory obsessive-compulsive disorder in the ECT and neurostimulation notes; and the somatic-symptom and illness-anxiety boundary in the Somatic-symptom, sexual, impulse-control disorders and suicide notes.

## Quick review

| THE ANXIETY DISORDERS: THE FOCUS OF THE FEAR, THE FIRST-LINE, AND THE SCALES | |
|---|---|
| THE DISCRIMINATOR | The anxiety symptoms are shared; the disorders separate by the **focus of the fear**. GAD = free-floating worry about everything; panic = the next attack; social anxiety = negative evaluation; specific phobia = one circumscribed object; agoraphobia = no escape or help. |
| GENERALISED ANXIETY DISORDER | Chronic uncontrollable worry across domains. First-line an **SSRI or SNRI**; also **pregabalin** and **buspirone**; a benzodiazepine is **not** first-line and not for long-term use. |
| PANIC DISORDER AND CLARK'S MODEL | Recurrent unexpected panic; the vicious cycle is the **catastrophic misinterpretation** of benign body sensations, maintained by safety behaviours. First-line SSRI (start low, warn about early activation) and CBT. |
| SOCIAL ANXIETY, PHOBIAS, AGORAPHOBIA | Social anxiety: SSRI and CBT, with **propranolol** for discrete performance anxiety. Specific phobia: graded exposure and systematic desensitisation (blood-injection-injury is diphasic vasovagal, use applied tension). Agoraphobia: exposure, now diagnosable independent of panic. |
| THE SCALES | **GAD-7**: 7 items 0 to 3, total **0 to 21**; 5, 10 and 15 mark mild, moderate and severe, and **10 or more** is the clinical cut-off. **HAM-A**: 14 clinician-rated items 0 to 4, total **0 to 56**. |
| EXCLUDE THE BODY FIRST | Before naming an anxiety disorder, exclude the mimics by tempo: **sustained** thyrotoxicosis (TSH) mimics GAD; **episodic** phaeochromocytoma (metanephrines) and hypoglycaemia mimic panic; the sign-first arrhythmia (ECG) and asthma; **worst on waking** is alcohol or benzodiazepine withdrawal; caffeine over **250 mg**. Missing a mimic is the exam-fatal error. |

| OBSESSIVE-COMPULSIVE AND RELATED DISORDERS: FEATURES, THE CIRCUIT, THE LADDER | |
|---|---|
| OCD FEATURES | Obsessions (intrusive, unwanted, ego-dystonic) and compulsions (neutralising acts); symptom dimensions (contamination/washing, symmetry/ordering, forbidden thoughts, harm/checking); the **insight specifier** (good or fair, poor, absent or delusional) and the tic-related specifier. |
| Y-BOCS | 10 clinician-rated items (5 obsession, 5 compulsion), each 0 to 4, total **0 to 40**. Bands: **0 to 7 subclinical, 8 to 15 mild, 16 to 23 moderate, 24 to 31 severe, 32 to 40 extreme**. |

| OBSESSIVE-COMPULSIVE AND RELATED DISORDERS: FEATURES, THE CIRCUIT, THE LADDER | |
|---|---|
| THE CSTC CIRCUIT | Orbitofrontal cortex and anterior cingulate to caudate to pallidum interna and substantia nigra reticulata to thalamus and back to cortex; the direct pathway disinhibits the thalamus, the indirect pathway brakes it. In OCD the direct loop is **overactive**, so the loop is hyperactive and self-sustaining. |
| THE MANAGEMENT LADDER | First-line a **high-dose SSRI** (fluoxetine 60, up to 80; sertraline 150 to 200; paroxetine 40 to 60; fluvoxamine to 300; escitalopram 20 to 30 mg) for a **long 10 to 12 week trial** plus CBT with ERP; then **clomipramine 150 to 225 mg** (ECG); then augment with **risperidone 1 to 3 mg** or aripiprazole for at least 8 weeks; then refractory options (DBS, neurosurgery). Continue a responder **1 to 2 years**. |
| CLOMIPRAMINE VERSUS SSRI, AND ERP | Clomipramine is marginally more efficacious on meta-analysis but less tolerable (cardiotoxicity, seizures), so an SSRI is first-line and clomipramine the switch. **Exposure and response prevention** (the exposure hierarchy plus prevention of the ritual, so anxiety habituates) is the core psychological treatment. |
| THE OCRD SPECTRUM | Body dysmorphic disorder (a perceived defect), hoarding, trichotillomania (hair-pulling) and excoriation (skin-picking); illness anxiety sits at the boundary with the somatic-symptom disorders. Each shares a repetitive act or a fixed preoccupation and responds in part to an SSRI and to behavioural therapy. |
| **TRAUMA AND STRESSOR-RELATED DISORDERS: PTSD, ITS TREATMENT, AND THE REST** | |
| PTSD CLUSTERS AND THE SCALE | DSM-5-TR requires four clusters (intrusion, avoidance, negative alterations in cognition and mood, arousal and reactivity); ICD-11 keeps three (re-experiencing in the here and now, avoidance, a persistent sense of current threat) and drops the negative-cognitions cluster. DSM-5 adds a dissociative subtype. **PCL-5**: 20 items 0 to 4, total **0 to 80**; provisional cut-off about **31 to 33**. |
| THE PTSD ALGORITHM | Two hard rules: **trauma-focused psychotherapy is first-line** (trauma-focused CBT or EMDR, 8 to 12 sessions), and a **benzodiazepine is never used**. If a drug is used: a licensed SSRI (**sertraline or paroxetine**) or **venlafaxine 37.5 to 300 mg**, trialled up to 12 weeks; add **prazosin 1 to 15 mg nocte** for nightmares; augment with risperidone or olanzapine; continue a responder at least **12 months**. |

| TRAUMA AND STRESSOR-RELATED DISORDERS: PTSD, ITS TREATMENT, AND THE REST | |
|---|---|
| COMPLEX PTSD | ICD-11 6B41 = the PTSD triad **plus** three disturbances in self-organisation (affect dysregulation, a persistent negative self-concept, interpersonal difficulty). Versus borderline personality disorder: complex PTSD shows trauma-reminder avoidance and current threat (absent in the PD), a **stable** negative self-view (not unstable identity), relationships of avoidance and disconnection (not abandonment), and suicidality that is less impulsive but of higher lethality. |
| ACUTE STRESS AND ADJUSTMENT | ICD-11 **acute stress reaction** is a transient normal response, classified outside the disorders; the DSM-5 **acute stress disorder** is a diagnosable disorder in the first month. Adjustment disorder is a maladaptive reaction to an identifiable stressor (ICD-11: preoccupation with the stressor plus failure to adapt); do not over-diagnose a normal reaction. |
| PROLONGED GRIEF; THE ATTACHMENT DISORDERS | Prolonged grief disorder (ICD-11 and DSM-5-TR): persistent, pervasive grief and yearning beyond the expected social norm and time; not normal grief. Reactive attachment and disinhibited social engagement disorders are the trauma disorders of early severe neglect (child home). |

| THE GUIDELINE SPINE AND THE HARD RULES | |
|---|---|
| THE GUIDELINE ORDER | Guidelines-first on the where-to-treat, target-by-phase, modes-of-treatment spine: **BAP leads**, then the **IPS CPG** (the 2025 guideline for OCD), NICE, CANMAT and WFSBP; state the BAP position first, then what the others recommend. |
| ANXIETY | **SSRI or SNRI first-line** for the anxiety disorders; the benzodiazepine is not first-line and not for long-term use; pregabalin and buspirone for GAD, propranolol for performance anxiety. |
| OCD | The anchoring rule is **high dose, long trial**: OCD needs a higher SSRI dose and a longer (10 to 12 week) trial than depression before it is called a failure; then clomipramine, then augmentation. A wrong first-line or an under-dose is critical. |
| PTSD | **Trauma-focused psychotherapy first**; then a licensed SSRI or venlafaxine with prazosin for nightmares; and **never a benzodiazepine**, which is not a treatment and may increase PTSD risk given early. |
| THE FIVE TRAPS | Missing a medical mimic of anxiety; under-dosing or stopping the OCD SSRI trial early; a benzodiazepine for PTSD; complex PTSD mislabelled as a personality disorder; and treating normal grief as prolonged grief disorder too soon. |

## References

The anxiety, obsessive-compulsive and trauma content is grounded in the standard psychiatry and psychopharmacology sources (the source hierarchy below), with Kaplan and Sadock and the Maudsley Prescribing Guidelines as the depth bar for the phenomenology, the neurobiology, the scale cut-offs and the doses, and Gelder's Shorter Oxford Textbook for the medical mimics of anxiety. The management is guideline-first, led by the British Association for Psychopharmacology, with the Indian Psychiatric Society (the 2025 clinical practice guideline for obsessive-compulsive disorder), NICE, CANMAT, the WFSBP, the United States VA/DoD and WHO mhGAP. Every scale cut-off, criterion, dose and first-line recommendation was checked against these sources and the adversarial content pass; anything that could not be confirmed was omitted and flagged rather than guessed. Each journal PMID below was verified against PubMed this build and matched on title, first author and year; identifiers that resolved to the wrong paper were corrected or dropped. Books and guideline documents are cited by author, title and year without a PMID.

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PART VIII

# VIII

# Dissociative, Conversion and Anxiolytic-Hypnotic Pharmacology

*The dissociative and conversion disorders, and the pharmacology of the anxiolytics and hypnotics.*


01 Dissociative and conversion disorders

02 Anxiolytic and hypnotic pharmacology

03 Apply and consolidate

PAPER II

# Dissociative, conversion and anxiolytic-hypnotic pharmacology

Two sections . one complete notes document

This material gathers the dissociative and conversion disorders and the anxiolytic and hypnotic drugs into one document, by syndrome and by mechanism. The first band is **the dissociative and conversion disorders**: the concept and classification of dissociation, the pathogenesis across the psychodynamic, trauma and neurobiological models, dissociative identity disorder, conversion or dissociative neurological symptom disorder with its positive rule-in signs, depersonalisation-derealisation, dissociative amnesia and fugue, and the possession and trance states. The second band is **the anxiolytic and hypnotic pharmacology**: the benzodiazepines in full, buspirone and the azapirones, the z-drugs, the dual orexin receptor antagonists, melatonin and ramelteon, etifoxine and the other non-benzodiazepine anxiolytics, and the beta-blockers, antihistamines and gabapentinoids used for anxiety. The examinable skill is twofold: the safe discrimination of the dissociative presentations from one another and, above all, of a conversion disorder from an organic disease by its positive signs; and the guideline-anchored selection of an anxiolytic or hypnotic, with the benzodiazepine mechanism, its half-life classes, its dependence and taper, and the short-course rule at the centre.

**HOW TO USE THESE NOTES**

Read the two bands as one account of a shared theme: the boundary between a functional and an organic disturbance, and the drugs that act on the same GABA-A receptor. The **dissociative and conversion** band builds the frame: the classification of the dissociative disorders, the models of how dissociation arises, each named syndrome discriminated from the others, and conversion or functional neurological disorder taught in full as a diagnosis made on POSITIVE signs (Hoover's sign, the give-way weakness, tremor entrainment, the tubular field) rather than by exclusion. The **anxiolytic and hypnotic** band teaches the drugs by mechanism: the benzodiazepines as positive allosteric modulators at the GABA-A receptor, their half-life-based classes, their dependence and the withdrawal-and-taper, and flumazenil as the antagonist, then buspirone, the z-drugs, the orexin and melatonergic hypnotics and the adjunctive anxiolytics. Every management recommendation is guideline-anchored, led by the BAP guidelines with the NICE, Maudsley and Indian Psychiatric Society positions stated, on the where-to-treat, target-by-phase, modes-of-treatment spine, and the benzodiazepine-sparing short-course rule is the anchor. Every named syndrome ends with a **Good to remember** box giving the criterion, the discriminator and the first step, and every named drug ends with one giving the mechanism or class, the signature caution and the one dose or half-life. Figures declare one axis and code it in colour: **petrol** marks structure and the neutral case, **coral** marks the trap or the danger, and **marigold** highlights the number to hold; the dissociation rating scales are reproduced as the actual instruments where the licence permits, credited to their sources, or typeset from their structure where it does not. Several boundaries are stated up front and cross-referenced, not re-taught: the GABA-A receptor and the wider receptor systems in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; the sedative and benzodiazepine use disorder as an addiction in the Substance use disorders notes; the lorazepam management of catatonia in the Other psychotic disorders, delusional syndromes and catatonia notes; the anxiety disorders where these anxiolytics are used in the Anxiety, OCD and trauma-related disorders notes; the SSRI and SNRI first-line anxiolytics in the Mood disorders: pharmacotherapy notes; clinical insomnia and CBT for insomnia in the Eating and sleep-wake disorders notes; the video-EEG work-up of dissociative convulsions in the Epilepsy and psychiatry notes; and the possession-trance states in the transcultural frame in the Consultation-liaison, geriatric and transcultural psychiatry notes. Conversion or dissociative neurological symptom disorder and the whole anxiolytic and hypnotic pharmacology are owned and fully taught here. This is doctor-facing revision, so the full technical vocabulary is used, brand names lead with the Indian brand, and every dose, half-life, scale cut-off, positive sign and first-line recommendation is verified against a source or omitted and flagged.

## Dissociative and conversion disorders

### 1 . Dissociative disorders: classification and clinical types

**Why this leads the chapter.** This topic is the orienting map for everything that follows. Dissociation is the **disruption of the normal integration** of consciousness, memory, identity, emotion, perception, body representation, motor control and behaviour (DSM-5-TR; ICD-11). Once you can name the components that can come apart, the whole family of **dissociative disorders** falls into place as a small number of **clinical types**, each defined by *which* integrative

function has failed. The examiner reward here is a clean **disorder-class map**: the ICD-11 grouping, the DSM-5-TR grouping, and the single organising split that separates the disorders of mind from the disorder of body. Get the map right and every individual criterion set hangs off it; get it wrong and you will confuse a depersonalisation state for psychosis, a functional seizure for epilepsy, or a possession trance for delusional identification.

This fragment first defines dissociation and its positive and negative symptoms, then draws the **classification of dissociative disorders** as a decision-tree, then walks each named clinical type with its defining criterion, and closes on how dissociation is measured. The accompanying figure sets the same content out as a classification tree (a decision-tree, or criteria map) to be memorised as one image. The wider anxiety context and the dissociative subtype of PTSD live in the Anxiety, OCD & trauma-related disorders notes; the video-EEG work-up of dissociative (non-epileptic) convulsions is developed in the Epilepsy & psychiatry notes; the transcultural reading of possession and trance is expanded in the Consultation-liaison, geriatric & transcultural psychiatry notes; and the somatic-symptom, functional and factitious boundary sits in the Somatic-symptom, sexual, impulse-control disorders & suicide notes.

**Fig 15.1** The classification of dissociative disorders as a decision tree: dissociation splits into disorders of mind and a single disorder of body, with ICD-11 keeping conversion inside the family and DSM-5 filing it elsewhere.

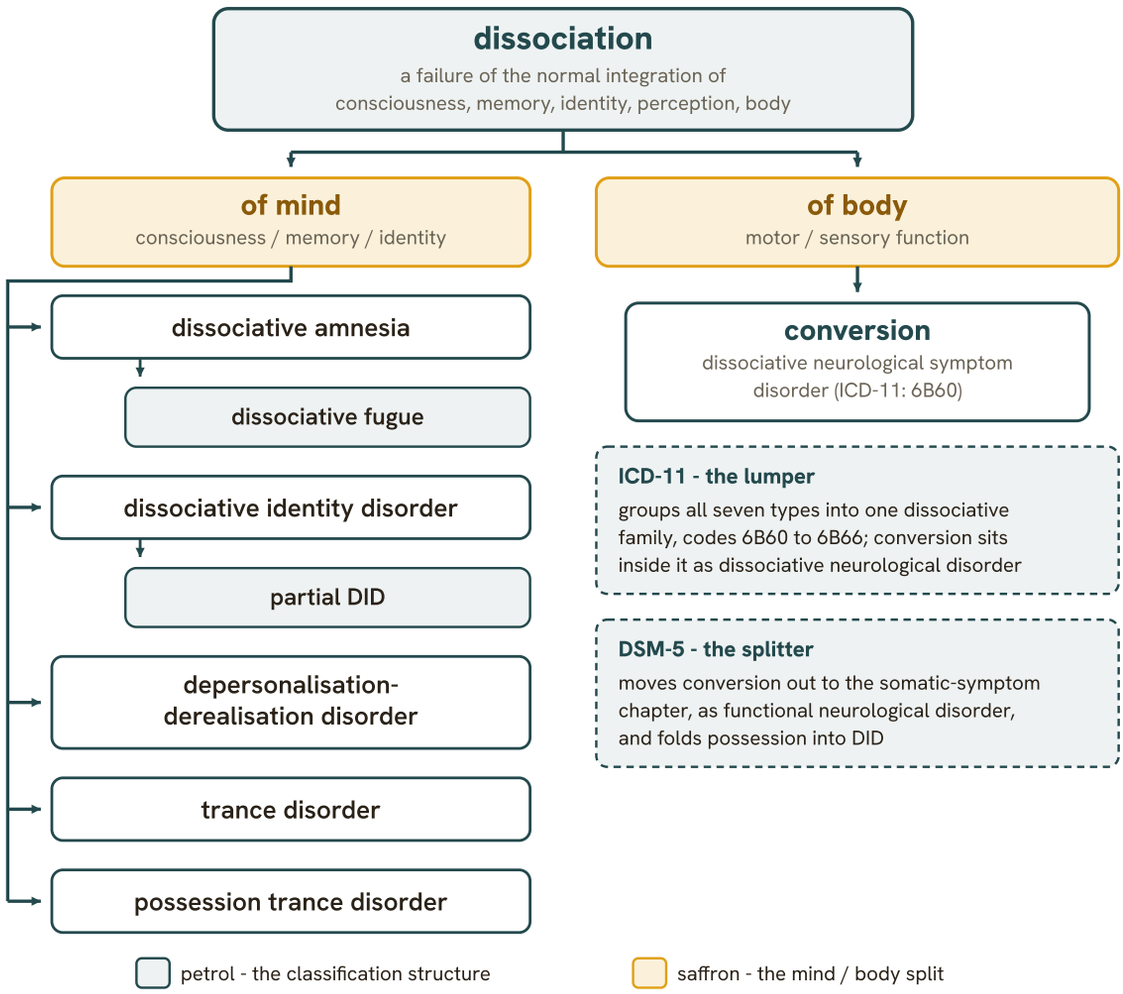


**Fig 15.2** The Dissociative Experiences Scale (DES-II) reproduced in full: the 28-item self-report screen for dissociative experiences, scored as the mean of all items.

## Dissociative Experiences Scale (DES-II)

**Instructions.** This questionnaire asks about experiences that you may have in your daily life. We are interested in how often you have these experiences. It is important that your answers show how often these experiences happen to you when you are not under the influence of alcohol or drugs. For each item, mark the percentage of the time the experience applies to you.

0% (never) · 10 · 20 · 30 · 40 · 50 · 60 · 70 · 80 · 90 · 100% (always)

1. Some people have the experience of driving or riding in a car or bus or subway and suddenly realizing that they don't remember what has happened during all or part of the trip.
2. Some people find that sometimes they are listening to someone talk and they suddenly realize that they did not hear part or all of what was said.
3. Some people have the experience of finding themselves in a place and have no idea how they got there.
4. Some people have the experience of finding themselves dressed in clothes that they don't remember putting on.
5. Some people have the experience of finding new things among their belongings that they do not remember buying.
6. Some people sometimes find that they are approached by people that they do not know, who call them by another name or insist that they have met them before.
7. Some people sometimes have the experience of feeling as though they are standing next to themselves or watching themselves do something and they actually see themselves as if they were looking at another person.
8. Some people are told that they sometimes do not recognize friends or family members.
9. Some people find that they have no memory for some important events in their lives (for example, a wedding or graduation).
10. Some people have the experience of being accused of lying when they do not think that they have lied.
11. Some people have the experience of looking in a mirror and not recognizing themselves.
12. Some people have the experience of feeling that other people, objects, and the world around them are not real.
13. Some people have the experience of feeling that their body does not seem to belong to them.
14. Some people have the experience of sometimes remembering a past event so vividly that they feel as if they were reliving that event.
15. Some people have the experience of not being sure whether things that they remember happening really did happen or whether they just dreamed them.
16. Some people have the experience of being in a familiar place but finding it strange and unfamiliar.
17. Some people find that when they are watching television or a movie they become so absorbed in the story that they are unaware of other events happening around them.
18. Some people find that they become so involved in a fantasy or daydream that it feels as though it were really happening to them.
19. Some people find that they sometimes are able to ignore pain.
20. Some people find that they sometimes sit staring off into space, thinking of nothing, and are not aware of the passage of time.
21. Some people sometimes find that when they are alone they talk out loud to themselves.
22. Some people find that in one situation they may act so differently compared with another situation that they feel almost as if they were two different people.
23. Some people sometimes find that in certain situations they are able to do things with amazing ease and spontaneity that would usually be difficult for them (for example, sports, work, social situations, etc.).
24. Some people sometimes find that they cannot remember whether they have done something or have just thought about doing that thing (for example, not knowing whether they have just mailed a letter or have just thought about mailing it).
25. Some people find evidence that they have done things that they do not remember doing.
26. Some people sometimes find writings, drawings, or notes among their belongings that they must have done but cannot remember doing.
27. Some people sometimes find that they hear voices inside their head that tell them to do things or comment on things that they are doing.

28. Some people sometimes feel as if they are looking at the world through a fog, so that people and objects appear far away or unclear.

**Scoring and interpretation.** Score each item 0 to 100. The DES-II score is the **mean** of all 28 items (range 0 to 100). A mean of about **30 or more** is a positive screen that warrants a full clinical interview for a dissociative disorder (a threshold of 20 to 30 is used across studies; only about 1 percent of people with dissociative identity disorder score below 30). The 8-item **DES-Taxon** (items 3, 5, 7, 8, 12, 13, 22, 27) estimates the probability of pathological dissociation. The DES is a screen, not a diagnosis.

Dissociative Experiences Scale (DES-II). Bernstein EM, Putnam FW (1986); Carlson EB, Putnam FW (1993), Dissociation 6(1):16-27. The copyright belongs to the authors, who have given permission for the scale to be copied, distributed or reproduced for clinical, research and educational use. Reproduced here for free, non-commercial educational use with acknowledgement.

**Fig 15.3** The Somatoform Dissociation Questionnaire (SDQ-20): structure and cut-offs, typeset from the published instrument (not reproduced verbatim).

### Somatoform Dissociation Questionnaire (SDQ-20)

A 20-item self-report measure of **somatoform (bodily) dissociation**: the disruption of the normal integration of sensory, motor and pain functions. It complements the DES, which measures the psychological forms of dissociation. Each item is rated on a five-point scale from 1 (this applies to me not at all) to 5 (this applies to me extremely).

**Negative somatoform symptoms (losses)**

Analgesia and kinaesthetic anaesthesia, blurred or tunnel vision, difficulty swallowing or passing urine, motor inhibition or functional paralysis, and loss of taste or smell.

**Positive somatoform symptoms**

Localised bodily pain and unusual, hard-to-explain bodily sensations.

**Scoring and interpretation.** Sum the 20 items (range **20 to 100**); a higher score indicates more somatoform dissociation. A total above about **30** is suggestive of a somatoform dissociative disorder. A derived five-item screener, the **SDQ-5**, uses a cut-off of **8 or more** to flag a probable dissociative disorder. The questionnaire is a screen, not a diagnosis.

Somatoform Dissociation Questionnaire (SDQ-20). Nijenhuis ERS, Spinhoven P, Van Dyck R, Van der Hart O, Vanderlinden J (1996), J Nerv Ment Dis. Structure and cut-offs typeset here in our own words with acknowledgement; the full item set is not reproduced.

## 1.1 What dissociation actually is

**An integration failure, not a content abnormality.** Normal mental life binds perception, memory, affect, identity and the sense of a body-in-control into one continuous stream owned by one self. In **dissociation** that binding fails, so functions that are normally seamless become detached, inaccessible or intrusive (DSM-5-TR; Kaplan 2024). DSM-5-TR frames the symptoms in two directions worth reproducing verbatim: **positive** dissociative symptoms are unbidden intrusions with loss of continuity in subjective experience (division of identity, depersonalisation, derealisation), while **negative** dissociative symptoms are an inability to access information or control mental functions that are normally available (amnesia). The historical spine runs from Pierre Janet, who described *desagregation*, the disaggregation of functions that should be integrated (Tasman 2024). Crucially, dissociation is best understood as a *transdiagnostic* process: a large meta-analysis of 219 studies across 19 diagnostic categories found dissociation cutting across most disorders and predicting poorer treatment response, which is why it should be assessed routinely rather than only when a dissociative disorder is suspected (Kaplan 2024).

**RULE** **Ask which function came apart.** Every dissociative disorder is named by the integrative function that has failed: memory (amnesia), identity (identity disorder), self and surroundings (depersonalisation-derealisation), awareness (trance), or motor and sensory control (conversion). Localise the failure and the diagnosis names itself.

### 1.2 The organising split: mind versus body

**Psychoform against somatoform dissociation.** The single most useful cut across the family is between dissociation of the *mind* (consciousness, memory and identity) and dissociation of the *body* (voluntary motor and sensory function). The first branch holds dissociative amnesia, dissociative identity disorder and its partial form, depersonalisation-derealisation disorder, and the trance and possession-trance disorders. The second branch is a single entity, conversion, which ICD-11 places *inside* the dissociative disorders as dissociative neurological symptom disorder, whereas DSM-5-TR keeps it under the somatic symptom disorders as functional neurological symptom disorder (Kaplan 2024). This is the heart of the **conversion versus dissociation** question: they are the same underlying process expressed in different currencies, one in the medium of consciousness and memory, the other in the medium of the body. Holding this split is what lets you place any new presentation on the **disorder-class map** in one step.

MNEMONIC

#### MIND SPLITS, BODY SPEAKS, GABA SLEEPS

The master hook for the whole chapter. **MIND SPLITS** is the psychoform half, the dissociative disorders of consciousness, memory and identity (amnesia, dissociative identity disorder and partial dissociative identity disorder, depersonalisation-derealisation, trance and possession trance). **BODY SPEAKS** is the somatoform half, conversion or dissociative neurological symptom disorder, the mind speaking through the body in motor and sensory symptoms. **GABA SLEEPS** hands over to the second half of the chapter, the anxiolytic and hypnotic pharmacology that acts on the GABA-A receptor. Carry these three beats and the whole chapter reassembles at consolidation from one line.

### 1.3 The ICD-11 dissociative grouping

**The seven named types, in code order.** ICD-11 defines dissociative disorders by "involuntary disruption or discontinuity in the normal integration of one or more of the following: identity, sensations, perceptions, affects, thoughts, memories, control over bodily movements, or behaviour" (ICD-11 CDDR). It then lists seven specified entities plus two residual codes. Reproduce the block as one table; the ICD-11 dissociative grouping is the version most examiners now expect first because it keeps conversion within the family.

| ICD-11 CODE | DISORDER | INTEGRATIVE FUNCTION DISRUPTED |
|---|---|---|
| 6B60 | **Dissociative neurological symptom disorder** | Motor and sensory control (conversion) |

| ICD-11 CODE | DISORDER | INTEGRATIVE FUNCTION DISRUPTED |
|---|---|---|
| **6B61** | Dissociative amnesia (specify with or without dissociative fugue) | Autobiographical memory |
| **6B62** | Trance disorder | Awareness of surroundings, narrowed consciousness |
| **6B63** | Possession trance disorder | Identity replaced by an external agent |
| **6B64** | Dissociative identity disorder | Identity split into distinct personality states |
| **6B65** | Partial dissociative identity disorder | Identity intruded on, without executive takeover |
| **6B66** | Depersonalisation-derealisation disorder | Sense of self and of reality (with intact reality testing) |
| **6B6Y / 6B6Z** | Other specified / unspecified dissociative disorder | Residual |

The ICD-11 dissociative-disorders grouping, ordered by code; conversion (6B60) sits inside the family.

### 1.4 The DSM-5-TR grouping and where conversion sits

**The one deliberate difference to memorise.** The DSM-5-TR dissociative disorders are dissociative identity disorder, dissociative amnesia (with dissociative fugue as a specifier), depersonalisation-derealisation disorder, other specified and unspecified dissociative disorder (Kaplan 2024). The load-bearing contrast is placement of conversion: ICD-11 keeps it as a dissociative disorder, DSM-5-TR classifies it under the somatic symptom disorders as functional neurological symptom disorder. DSM-5-TR also folds pathologic possession *into* dissociative identity disorder rather than giving it a separate code, and has no standalone trance or partial dissociative identity disorder categories, handling those subtler presentations under other specified dissociative disorder. The comparison, read side by side, is the examinable core of the **classification of dissociative disorders**.

| ENTITY | ICD-11 | DSM-5-TR |
|---|---|---|
| **Amnesia** | Dissociative amnesia (6B61) | Dissociative amnesia (fugue = specifier) |
| **Identity fragmentation** | Dissociative identity disorder (6B64) | **Dissociative identity disorder** |
| **Sub-threshold identity intrusion** | Partial dissociative identity disorder (6B65) | Other specified dissociative disorder |
| **Detachment from self / world** | Depersonalisation-derealisation (6B66) | Depersonalisation-derealisation disorder |
| **Narrowed awareness** | Trance disorder (6B62) | Other specified (dissociative trance) |

| ENTITY | ICD-11 | DSM-5-TR |
|---|---|---|
| **Possession** | Possession trance disorder (6B63) | Folded into dissociative identity disorder |
| **Conversion** | **Dissociative neurological symptom disorder (6B60)** | Functional neurological symptom disorder (under somatic symptom disorders) |

ICD-11 versus DSM-5-TR: same clinical types, two different filing decisions, conversion the pivotal one.

**PEARL**

The two manuals disagree in exactly one direction. ICD-11 is the "lumper" that keeps conversion, trance, possession and partial identity states inside the dissociative family; DSM-5-TR is the "splitter" that exports conversion to the somatic disorders and absorbs possession into dissociative identity disorder. Learn the ICD-11 list of seven, then learn what DSM-5-TR *moves*.

### 1.5 Dissociative amnesia (and dissociative fugue)

**Retrograde loss of autobiographical memory.** Dissociative amnesia is an inability to recall important autobiographical information, usually of a traumatic or stressful nature, that is inconsistent with ordinary forgetting (DSM-5-TR). The deficit is predominantly *retrograde* and the new-learning (anterograde) capacity is preserved, which is the discriminator from organic amnesia. It takes recognised patterns: **localised** (a circumscribed event or period), **selective** (some aspects of an event), **generalised** (whole life history and identity, and rare), and the older descriptors **continuous** and **systematised** (Kaplan 2024). Dissociative fugue, purposeful travel or bewildered wandering with amnesia for identity, is a specifier of dissociative amnesia rather than a free-standing disorder. Most patients are initially unaware of the gap, noticing it only when confronted with evidence of events they cannot recall.

**TRAP** **Calling organic amnesia dissociative.** Dissociative amnesia is retrograde and autobiographical with intact new learning; a dense *anterograde* deficit, clouding, or a focal neurological picture points to delirium, a seizure, transient global amnesia, Wernicke-Korsakoff or a head injury. Exclude the organic cause before you reach for a dissociative label.

**GOOD TO REMEMBER**

- **Dissociative amnesia:** defining criterion is an inability to recall important autobiographical information (usually stressful) inconsistent with normal forgetting; deficit is *retrograde*, new learning intact; subtypes localised / selective / generalised (continuous, systematised); fugue is a *specifier*, not a separate disorder. First step: rule out an organic amnestic cause.

### 1.6 Dissociative identity disorder and partial dissociative identity disorder

**Two or more identity states, plus amnesia.** Dissociative identity disorder is defined by (a) two or more distinct personality states, or an experience of possession, producing marked discontinuity in the sense of self and agency, and (b) recurrent episodes of *dissociative amnesia* (DSM-5-TR). The amnesia criterion is not optional: it is what separates dissociative identity disorder from non-pathological possession or from a purely intrusive picture. DSM-5 deliberately dropped the older requirement that states visibly "take control", because phenomenological studies showed most

cases are subtle, with overlapping and interfering states rather than dramatic switching; amnesia was endorsed by essentially all such patients. ICD-11 adds partial dissociative identity disorder for the common presentation in which one dominant state runs daily life but is intruded on by non-dominant states that take executive control only briefly; in DSM-5-TR most of these fall under other specified dissociative disorder because they lack the full amnesia picture.

**The trauma model against the sociocognitive model.** The dominant *trauma (posttraumatic) model*, formalised as structural dissociation theory, holds that the personality fails to integrate under severe, repeated early-childhood trauma, and cites consistently high verified trauma rates across cultures. The competing *sociocognitive (fantasy) model* argues the disorder is shaped by suggestion, media exposure and inadvertent therapist cueing in highly hypnotisable, fantasy-prone individuals. The examinable position is that the evidence robustly links dissociation to early trauma, while structural dissociation theory itself remains contested even among dissociation experts (Kaplan 2024).

**GOOD TO REMEMBER**

- **Dissociative identity disorder:** defining criteria are two or more distinct personality states (or possession) *plus* recurrent dissociative amnesia; DSM-5 removed the "takes control" requirement. Discriminator: the *amnesia* criterion. Debate: trauma / structural-dissociation model versus sociocognitive (fantasy) model.
- **Partial dissociative identity disorder (ICD-11):** one dominant state that functions in daily life, intruded on by non-dominant states that seize executive control only transiently; sits under other specified dissociative disorder in DSM-5-TR.

### 1.7 Depersonalisation-derealisation disorder

**Detachment with the reality check intact.** Depersonalisation-derealisation disorder is persistent or recurrent depersonalisation (unreality or detachment from one's own mind, self or body) and/or derealisation (unreality or detachment from surroundings), during which *reality testing remains intact* (DSM-5-TR criterion B). That intact reality testing, and the "as if" quality of the experience without any delusional or dereistic explanation, is the defining differential-diagnostic point separating the disorder from a psychotic disorder (Kaplan 2024). The symptoms must cause distress or impairment, not be attributable to a substance or medical condition (notably seizures), and not be better explained by another disorder. Transient depersonalisation is extremely common in the general population and after cannabis, ketamine or hallucinogens; it becomes a disorder only when persistent and impairing.

**TRAP** **Reading detachment as psychosis.** The patient who says the world feels unreal, "as if behind glass", but *knows* it is not truly unreal has intact reality testing and depersonalisation-derealisation, not schizophrenia. A delusional, ideas-of-reference or Schneiderian colouring, or belief that the unreality is literal, moves you toward the psychotic spectrum.

GOOD TO REMEMBER

- **Depersonalisation-derealisation disorder:** defining criterion is persistent/recurrent depersonalisation and/or derealisation with *intact reality testing* and an "as if" quality; the intact reality check is the discriminator from psychosis. Scale anchor: a Cambridge Depersonalisation Scale score of **70 or more** is the conventional diagnostic threshold (Kaplan 2024).

### 1.8 Trance disorder and possession trance disorder

**Narrowed awareness, or replaced identity.** Trance disorder is an acute narrowing or complete loss of awareness of immediate surroundings, with profound unresponsiveness or insensitivity to stimuli, and often stereotyped movements experienced as outside one's control. Possession trance disorder adds the replacement of the usual sense of personal identity by an external "possessing" identity, typically attributed to a spirit, deity or other agent, with amnesia for the episode. ICD-11 gives these two their own rubric, distinct from dissociative identity disorder; DSM-5-TR instead folds pathological possession into dissociative identity disorder and leaves non-possession trance under other specified dissociative disorder. The decisive criterion for both is the cultural exclusion: the state must be *involuntary, unwanted and outside accepted collective cultural or religious practice*. A trance willingly entered by a healer within an accepted ritual, causing no distress, is not a disorder (Kaplan 2024).

INDIA

Trance and possession presentations are among the most frequent dissociative presentations in Indian practice; the Indian texts describe them directly as "trance and possession disorders (possession hysteria)" and list conversion and dissociative signs such as pseudo-seizures and possession states together. This is the genuine culture-bound edge of the topic: possession and mediumship are woven into accepted ritual across many Indian communities, so the same phenomenology may be normative in one setting and pathological in another. The reading turns entirely on the ICD-11 and DSM-5-TR cultural-exclusion criterion and a proper cultural formulation, not on the surface picture. The transcultural frame is expanded in the Consultation-liaison, geriatric & transcultural psychiatry notes.

GOOD TO REMEMBER

- **Trance disorder:** defining criterion is acute narrowing/loss of awareness of surroundings with unresponsiveness; identity is *not* replaced.
- **Possession trance disorder:** defining criterion is replacement of identity by an external possessing agent with amnesia; both require the state to be involuntary, distressing and *outside accepted cultural or religious practice*. First step: apply the cultural-exclusion criterion.

### 1.9 Conversion: dissociative neurological symptom disorder

**Diagnosed by positive signs, not by exclusion.** Dissociative neurological symptom disorder (ICD-11), the functional neurological symptom disorder of DSM-5-TR, is motor, sensory or seizure-like symptoms that are genuinely experienced but incompatible with recognised neurological disease. The modern rule, from Stone and Carson, is that this is *not* a diagnosis of exclusion: it requires **positive signs of internal inconsistency** demonstrating that the nervous

system can work normally when attention is diverted (New Oxford 2024). The cardinal example is Hoover's sign: hip-extension weakness that returns to normal strength during contralateral hip flexion against resistance. The tremor entrainment test shows a functional tremor changing frequency to, or stopping with, a copied rhythm in the other hand. A "give-way" or collapsing weakness, a tubular visual field of the same width at 1 m and 2 m, and astasia-abasia are further positive findings. DSM-5-TR also *removed* the old requirement for an identifiable psychological stressor, and la belle indifference and the mere presence of a stressor are unreliable and are not diagnostic. Because it can coexist with true neurological disease, the positive sign is what rules it in.

| POSITIVE SIGN | POSITIVE FINDING THAT RULES IN A FUNCTIONAL CAUSE |
|---|---|
| **Hoover's sign** | **Hip-extension weakness that normalises with contralateral hip flexion against resistance** |
| **Tremor entrainment test** | Unilateral tremor entrains to, stops with, or cannot copy a rhythm set by the other hand |
| **Give-way (collapsing) weakness** | Ratchety, inconsistent power that gives way rather than a fixed pyramidal pattern |
| **Tubular visual field** | A field defect of the same width at 1 m as at 2 m (physiologically it should widen) |
| **Astasia-abasia / dragging gait** | Bizarre, non-physiological gait, or a leg dragged with the forefoot kept on the floor |

Positive rule-in signs of functional (conversion) neurological symptoms (Stone and Carson, New Oxford 2024).

**RULE** **Rule in, do not rule out.** Conversion is diagnosed on a positive sign of internal inconsistency (Hoover's, entrainment, tubular field), not on the absence of a lesion or the presence of a stressor. The sign shows the system works when attention is elsewhere.

**WORKED EXAMPLE**

A patient reports a dense right-leg weakness after an argument. Direct right hip extension is flaccid. You then ask her to flex the *left* hip against your hand; as she does, involuntary right hip extension presses firmly into your other palm. That is a positive Hoover's sign: the "paralysed" extensors fire automatically when attention shifts to the good leg. You have ruled the diagnosis *in* at the bedside, without imaging and without invoking the stressor.

**COMMON TRAP**

The mirror error: an early multiple sclerosis, a spinal cord lesion, a stroke or a focal seizure labelled "conversion" because the presentation seemed emotional or a stressor was present. A positive functional sign coexists with real disease more often than expected, so a rule-in sign never removes the duty to screen for an organic lesion when the picture is atypical, progressive or accompanied by hard neurological findings.

**GOOD TO REMEMBER**

- **Dissociative / functional neurological symptom disorder:** defining criterion is a neurological symptom with a *positive* sign of internal inconsistency (Hoover's sign, tremor entrainment, tubular field); it is *not* a diagnosis of exclusion, no stressor is required (DSM-5-TR), and la belle indifference is unreliable. First step: elicit a positive sign.

### 1.10 Measuring dissociation: the DES and the two-tier screen

**Screen, then interview.** The Dissociative Experiences Scale (DES) is the workhorse self-report measure: subjects rate the percentage of time, from 0 to 100, that each dissociative experience occurs. Most epidemiology uses a two-tier approach, screening with the DES and then applying a structured interview (the SCID-D or the DDIS) to everyone above a cut-off "generally **20 to 30**", with **30** the conventional screening threshold (Kaplan 2024). Mean DES scores rank the family in a way worth memorising: dissociative identity disorder **48.7**, all dissociative disorders combined 38.9, PTSD 28.6, borderline personality disorder 27.9, functional neurological (conversion) disorder 25.6, against a non-clinical mean near 8. The eight-item *DES-Taxon* (DES-T) subscale isolates pathological dissociation and identifies a distinct high-scoring group; the population point-prevalence of this dissociative taxon is about **3.4%**. Somatoform (bodily) dissociation is captured separately by the Somatoform Dissociation Questionnaire (SDQ-20). The instrument forms themselves are reproduced on the accompanying scale plate; a high DES is a flag for further interview, never a diagnosis on its own.

| 30 | 8 | 48.7 | 3.4% |
|---|---|---|---|
| USUAL DES SCREENING CUT-OFF | ITEMS IN THE DES-T TAXON SUBSCALE | MEAN DES IN IDENTITY DISORDER | POPULATION DISSOCIATIVE TAXON |

**HOLD THIS**

The whole family divides once: dissociation of the mind (amnesia, identity, depersonalisation-derealisation, trance and possession) against dissociation of the body (conversion), with ICD-11 keeping conversion inside the dissociative disorders and DSM-5-TR exporting it to the somatic symptom disorders.

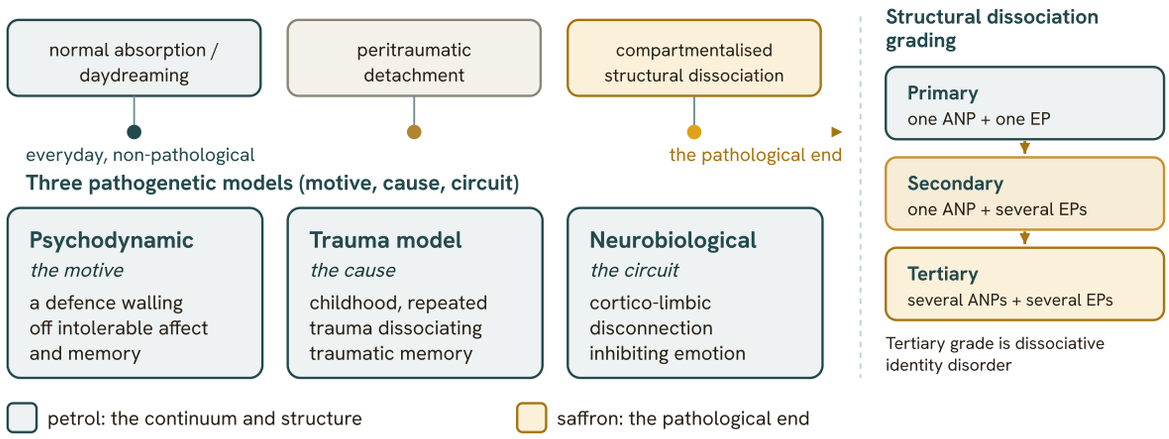
## 2 · The pathogenesis of dissociation

The **pathogenesis of dissociative disorders** is a favourite viva topic precisely because there is no single mechanism to recite; the examiner wants to hear that you can hold three complementary explanations at once and say what each adds. **The frame to open with.** Dissociation is a disruption of the normally integrated stream of consciousness, memory, identity, emotion, perception and body-sense; the question of pathogenesis asks how that integration comes to fail. The classical answer runs across a **psychodynamic model** (dissociation as a defence that walls off intolerable affect and memory), a **trauma model** (childhood and repeated trauma driving the dissociation of traumatic memory, formalised as the theory of structural dissociation of the personality), and a **neurobiological model** (a fronto-limbic, or cortico-limbic, disconnection with cortico-limbic inhibition visible on imaging). These are not

rival hypotheses to be scored against one another so much as three levels of the same process, and the candidate who names all three, and links them, has the marks.

**Why it matters clinically.** The three models are not academic; each tells you something the others cannot. The trauma model tells you to take a developmental and abuse history and to expect comorbid post-traumatic disorder. The psychodynamic model tells you why the patient cannot simply "remember" on command and why phased, safety-first therapy, not forced abreaction, is the treatment grammar. The neurobiological model tells you that reality testing stays intact (so this is not psychosis) and explains the numbing and detachment as emotional over-regulation rather than absence of feeling. Teach the **aetiology of dissociation** as one concept model with three faces; the accompanying figure sets it out as a dissociation continuum, from everyday absorption through peritraumatic detachment to the compartmentalised structural dissociation of the severe disorders. The wider dissociative subtype of post-traumatic stress disorder is developed in the Anxiety, OCD & trauma-related disorders notes; here we own the pathogenesis.

**Fig 15.4** Dissociation as one continuum with three complementary pathogenetic models, and the primary-to-tertiary grading of structural dissociation (ANP the apparently-normal part, EP the emotional part).




## 2.1 The three-model scaffold and the dissociation continuum

**The concept to fix first.** Dissociation runs along a continuum. At one end are normal, non-pathological experiences (absorption in a book, highway hypnosis, momentary "spacing out"); at the other are the compartmentalised amnesias and identity alterations of the severe disorders. Overlaid on that continuum is a genuine debate about whether pathological dissociation is merely the high end of a smooth dimension or a discontinuous *type*; taxonometric work (see the scale section) argues for a distinct pathological taxon rather than a pure continuum. Either way, the pathogenetic question is the same: what pushes a person from normative, transient dissociation into the persistent, involuntary, compartmentalising kind?

**Three answers, three levels.** The psychodynamic model answers at the level of *meaning and motive* (a defence against unbearable experience); the trauma model answers at the level of *developmental cause* (early, repeated, relationship-based trauma); and the neurobiological model

answers at the level of *mechanism and circuit* (a cortico-limbic disconnection that inhibits emotion). Read them as motive, cause and circuit and they stop competing and start cooperating. The accompanying figure draws this as the concept model to carry through the whole answer.

RULE **Dissociation is a failure to integrate, not a loss of contact with reality.** The three models describe one process at three levels, a psychodynamic defence that walls off intolerable affect and memory, a developmental response to early repeated trauma, and a cortico-limbic disconnection that inhibits emotion; reality testing stays intact throughout, which is what separates dissociation from psychosis.

MNEMONIC

### Three D's: Defence, Developmental trauma, Disconnection

Run the pathogenesis in three beats. **Defence** is the psychodynamic model (dissociation walls off intolerable affect and memory). **Developmental trauma** is the trauma model (childhood and repeated trauma, formalised as structural dissociation into an apparently-normal and an emotional part). **Disconnection** is the neurobiological model (cortico-limbic disconnection with prefrontal inhibition of the limbic system). Name the three D's, then link them, and the "aetiology of dissociation" answer writes itself.

## 2.2 The psychodynamic model: dissociation as a defence

**The founding schism.** The **psychodynamic model** descends from the late-nineteenth-century split between Pierre Janet and Sigmund Freud. Janet described *désagrégation*, a passive splitting-off of "fixed ideas" and traumatic memory from the integrated personality under the impact of overwhelming events; Freud, by contrast, moved toward *repression* and, later, "splitting" as the governing defences, and psychoanalysis broadened the word trauma to mean almost any developmental stressor. The consequence, as Kaplan & Sadock note, is that psychoanalytic theory has long had difficulty treating dissociation as a *specific* intrapsychic defence against awareness of trauma, tending instead to fold it into "splitting"; David Rapaport's Menninger group in the 1940s, and the older notion of "hypnoid states," were among the few attempts to reconcile the Freudian and Janetian lines.

**The defensive core, and the autohypnosis variant.** Stripped to its clinical claim, the psychodynamic model says dissociation protects the person by keeping intolerable affect, memory and self-knowledge out of integrated awareness. The most exam-relevant developmental version is the autohypnosis model (associated with Bliss and with Richard Kluft): a traumatised child recruits an innate hypnotic capacity to induce a self-protective dissociative state during overwhelming or repetitive abuse, and with continuous use that autohypnotic state becomes personified into self-states. It is supported by the observation that dissociative patients, especially those with dissociative identity disorder, are the most highly hypnotisable clinical group, though the model is criticised for reasoning backward from adults and for the fact that more systematic research has not confirmed that childhood trauma reliably raises hypnotisability. The grounding of grounding and hypnosis as psychotherapy technique sits in the Psychotherapies, clinical assessment, MSE & nosology notes.

### 2.3 The trauma model: childhood and repeated trauma

**Trauma as the necessary antecedent.** The **trauma model** is the dominant contemporary account of the **aetiology of dissociation**. Since the 1980s, several hundred studies across many cultures have found significantly higher dissociation, measured on the Dissociative Experiences Scale and comparable instruments, in traumatised groups than in non-traumatised clinical and general-population samples. **Childhood trauma** is the strongest correlate: childhood sexual and physical abuse, emotional abuse and neglect, and non-maltreatment traumas such as repeated painful medical procedures or wartime displacement, are all over-represented, and survivors of severe, chronic adult trauma (prisoners of war, torture survivors, trafficked and prostituted women, victims of intimate-partner violence) carry high dissociation scores. A meta-analysis by Vonderlin and colleagues of 65 DES studies (7,532 participants) found that a childhood history of abuse or neglect predicted significantly higher DES scores, with combined physical and sexual abuse, earlier onset, longer duration and parental (intrafamilial) abuse driving the highest scores.

**Two mechanisms the model adds.** First, *peritraumatic dissociation*, dissociation measured at or right after the event (depersonalisation, derealisation, time distortion, trance-like automatism, amnesia), predicts later post-traumatic stress disorder, and dissociation statistically *mediates* the relationship between trauma and subsequent psychopathology. Second, betrayal trauma theory (Jennifer Freyd) explains why abuse by a trusted, depended-upon caregiver is especially likely to be walled off: amnesia here is protective of the attachment the child cannot afford to lose, which is why intrafamilial childhood abuse produces more amnesia than, say, a natural disaster. This is the model that makes dissociation a target for early intervention after trauma.

### 2.4 Structural dissociation of the personality

**The formal trauma theory to name.** The trauma model's most cited theoretical elaboration is the theory of structural dissociation of the personality (Onno van der Hart, Ellert Nijenhuis and Kathy Steele), especially influential in Europe and the guiding force behind the ICD-11 criteria for dissociative identity disorder and partial dissociative identity disorder. It proposes that severe traumatic experience produces a "split" between an apparently normal personality (ANP), which is constricted and avoidant, focused on daily functioning, unaware of and phobic toward the traumatic material, and an emotional personality (EP), which carries the unintegrated, unprocessed traumatic memory and its raw affect. The degree of splitting is graded, which is the examinable part.

| LEVEL OF STRUCTURAL DISSOCIATION | CONFIGURATION | PROTOTYPICAL DISORDER |
|---|---|---|
| **Primary** | one ANP + one EP | **Post-traumatic stress disorder (and simple conversion presentations)** |
| **Secondary** | **one ANP + several EPs** | Complex / "less complex" dissociative disorders, complex PTSD |
| **Tertiary** | several ANPs + several EPs | **Dissociative identity disorder** |

Structural dissociation graded from primary to tertiary; the accompanying figure renders it as the compartmentalised end of the dissociation continuum. Grounded in Kaplan & Sadock CTP 2024 (van der Hart, Nijenhuis & Steele).

**Hold the critique too.** Examiners reward a candidate who can also say why the theory is contested: it over-reifies the mind as a "structure" made of "parts," it reasons backward from adult dissociative identity disorder rather than forward from child development, it posits adult-style psychological "structures" in infants (mapping the ANP/EP alternation onto Type-D attachment), and meta-analytic data fit dissociation better as a subtype or component of post-traumatic stress disorder than as the substrate of all of it. A cleaner contemporary framing treats these conditions as disorders of self-organisation, complex dynamic self-state systems, rather than a lesioned "personality."

### 2.5 The neurobiological model: cortico-limbic disconnection

**The core hypothesis.** The **neurobiological model** reframes dissociation as a hard-wired inhibitory response of the psychobiological survival system, one that damps anxiety and physiological hyperarousal under extreme threat and becomes pathological when it persists outside danger. Its anatomical statement is the corticolimbic disconnection hypothesis of Mauricio Sierra and German Berrios: in depersonalisation and derealisation there is a bilateral cortico-limbic disconnection with *prefrontal activation and limbic inhibition*, yielding hypoemotionality (the flat, detached, "unreal" quality) together with attentional difficulty. This is the fronto-limbic / cortico-limbic disconnection the question is pointing at.

**The imaging and the PTSD bridge.** The signature is captured on functional imaging: a positron-emission study using intravenous tetrahydrocannabinol found that cerebral blood-flow *increases* in right frontal and anterior cingulate cortex tracked depersonalisation/derealisation severity, while subcortical blood flow *fell* in the amygdala, hippocampus, basal ganglia and thalamus, exactly the "cortex up, limbic down" pattern. Ruth Lanius and colleagues extended this model to trauma: the dissociative subtype of post-traumatic stress disorder is a state of emotional *over*-modulation (prefrontal over-activation inhibiting the limbic system, producing hypoarousal, numbing and detachment), the mirror image of the emotional *under*-modulation (weak prefrontal restraint over a hyperaroused amygdala) seen in ordinary re-experiencing PTSD. The receptor systems and the wider fear circuitry are developed in the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes and in the Anxiety, OCD & trauma-related disorders notes; that transcranial magnetic stimulation of the dorsolateral and ventrolateral prefrontal cortex and the temporoparietal junction can *reduce* depersonalisation symptoms, while deep stimulation over medial prefrontal cortex can *induce* depersonalisation in healthy volunteers, is direct causal support for the circuit.

**PEARL**

One circuit, two directions. Non-dissociative, re-experiencing PTSD is emotional *under*-modulation, a weak prefrontal brake over a hyperaroused amygdala. The dissociative subtype, and depersonalisation-derealisation disorder, is the mirror image, emotional *over*-modulation, prefrontal over-activation that inhibits the limbic system and produces numbing, detachment and hypoarousal. Same cortico-limbic axis, opposite setting; that one sentence answers "what is the neurobiology of dissociation?"

**GOOD TO REMEMBER**

- **Depersonalisation-derealisation disorder (the criterion anchor):** persistent or recurrent depersonalisation (detachment from one's own mind or body, an "as-if-in-a-dream" unreality) and/or derealisation (unreality of surroundings) with *reality testing kept intact throughout*; the intact reality testing is the discriminator from psychosis, and the cortico-limbic disconnection signature (prefrontal over-activation inhibiting the limbic system) is its putative mechanism, so the first step when it is mislabelled "psychotic" is to confirm that the patient knows the experience is not real.

### 2.6 The trauma model versus the sociocognitive challenge

**The debate to state.** The pathogenesis of dissociative identity disorder is where the models are fought over hardest, and this trauma-model-versus-sociocognitive debate is the single most examinable controversy in the chapter. The trauma model holds that severe, early, repeated (often betrayal-laden) trauma drives the disorder (tertiary structural dissociation, or a developmental failure to consolidate one self across states). Against it stand a family of sceptical accounts, the iatrogenic model (states cued and shaped by suggestible therapy, including hypnosis and "recovered-memory" work), the sociocognitive model (a socially learned role reinforced by media and culture), and the fantasy model (dissociation as a trait of fantasy-proneness and suggestibility, with the trauma link an artefact of confabulation).

**Where the evidence sits.** A systematic comparison by Dalenberg and colleagues (2012) tested the trauma and fantasy models against multiple predictions and found the trauma model confirmed on the great majority, including the point that neurobiological research supports dissociation as a regulatory response to fear with measurable biological correlates. The sociocognitive and iatrogenic accounts, by contrast, rest largely on old, small, analogue studies and cannot account for the large, cross-cultural epidemiological samples ascertained with validated instruments in ordinary clinical settings. The honest examination answer is that trauma is the principal driver, while sociocognitive and cultural factors shape the *secondary structuring* (the number, form and content of self-states) rather than manufacturing the disorder wholesale.

**TRAP** **Diagnosing dissociative identity disorder without the dissociative-amnesia criterion.** Distinct "states" or role enactments are not enough; the diagnosis requires a disruption of identity with two or more distinct personality states *plus* recurrent dissociative amnesia (gaps in recall for everyday events, personal information or trauma). If the amnesia is absent, or the picture is florid and suggestion-shaped, go back to the sociocognitive and iatrogenic differentials before you commit.

**GOOD TO REMEMBER**

- **Dissociative identity disorder (the criterion anchor):** on the trauma model it is the tertiary structural dissociation of a child overwhelmed by early, repeated, betrayal-laden **childhood trauma**; the defining criterion is a disruption of identity with two or more distinct personality states *and* recurrent dissociative amnesia, with the presentation not a normal part of a broadly accepted cultural or religious practice; never diagnose it without the amnesia criterion, and weigh the sociocognitive and iatrogenic challenge before accepting a suggestion-shaped presentation.

### 2.7 Measuring the process: the DES and the dissociative taxon

**The instrument to name.** Pathogenesis research runs on measurement, and the workhorse is the Dissociative Experiences Scale (DES; Bernstein & Putnam 1986, with the DES-II by Carlson & Putnam 1993), a self-report screen for the frequency of dissociative experiences. It is used in a two-tier way: the DES screens a population, and those above a cut-off are taken to a structured diagnostic interview (the SCID-D-R or the DDIS). The commonly cited screen cut-off is a DES mean of the order of 20 to 30, with a score above 30 indicating severe dissociative psychopathology; the meta-analytic mean DES climbs with severity, from around 25 in conversion disorder and 28 in post-traumatic stress disorder to about 39 across the dissociative disorders and 49 in dissociative identity disorder. The actual instrument is shown in the accompanying scale plate rather than pasted here.

**Continuum or taxon.** The DES also feeds the continuum-versus-type debate: a taxonometric analysis (Waller, Putnam & Carlson) isolated a DES-Taxon (the DES-T), a subset of items that identifies a discrete subgroup of genuinely pathological dissociators rather than simply high scorers on a smooth dimension. A large Finnish general-population study put the point prevalence of that pathological taxon at about 3.4%. The taxon reading implies a different developmental trajectory (and different treatment) from the merely-high end of normal, and dovetails with the trauma and neurobiological models by pointing to a distinct, trauma-linked pathological process.

### 2.8 The culture-bound lens and the Indian setting

**Culture shapes the presentation.** Sociocognitive and cultural factors do not create dissociation, but they strongly shape how it is expressed. Conversion-like dissociative presentations are, on cross-cultural data, more common in India than in certain affluent Western settings, and possession and trance phenomena are a major idiom of dissociative distress in the Indian clinic. The nosologies diverge here: DSM-5 folds pathological possession into dissociative identity disorder, whereas ICD-11 keeps trance disorder and possession trance disorder as categories distinct from dissociative identity disorder. The transcultural framing of possession, trance and the culture-bound syndromes is developed in the Consultation-liaison, geriatric & transcultural psychiatry notes.

**The prescribing distortion.** Onto this genuinely trauma-and-culture-driven presentation the Indian system too often lays a pharmacological reflex. Benzodiazepines are well documented to be over-prescribed for long-term anxiety, and alprazolam in particular is the widely used, default anxiolytic, dispensed freely for the "tension," insomnia and dissociative or possession-flavoured distress that actually call for a trauma-informed, phased psychotherapy. This is the wrong tool on every one of the three models: it addresses neither the defence, nor the developmental trauma, nor the cortico-limbic circuit, and it breeds dependence. The sedative and benzodiazepine use disorder, intoxication and dependence in the addiction sense are taught in the Substance use disorders notes.

**TRAP** **Reading a possession or trance state as a dissociative disorder without the cultural formulation.** Possession trance that is a sanctioned, time-limited part of a collective religious or cultural practice is normative, not pathological; the diagnosis requires that the state be unwanted, involuntary, distressing or impairing and occur outside an accepted cultural or religious practice.

**INDIA**

Dissociation is arguably *more* visible in Indian practice than in the Western textbook: conversion-like and possession/trance presentations are common, culturally embedded and frequently the first idiom in which trauma is spoken. The pathogenesis lens gives two correctives. First, formulate possession and trance culturally before pathologising them (ICD-11's possession trance disorder is defined by the involuntary, distressing, out-of-context state, not by belief in possession itself). Second, resist the country's **benzodiazepine over-prescription** pattern: alprazolam (Alprax, Restyl) is the default anxiolytic and is handed out long-term for exactly these presentations, which suits neither the defence, the developmental trauma, nor the cortico-limbic circuit, and courts dependence. The trauma-informed answer is a phased psychotherapy that builds safety and grounding first, with any anxiolytic kept to a genuine short course.

**GOOD TO REMEMBER**

- **Alprazolam (Alprax or Restyl), the dose anchor and the anti-pattern:** a high-potency, short-to-intermediate-acting benzodiazepine and India's default anxiolytic, a positive allosteric modulator at the GABA-A receptor (the mechanism is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes); the licensed anxiolytic range is 1 to 4 mg per day (about 5 to 6 mg per day for panic), yet it does *not* treat dissociation. Its short action causes interdose rebound anxiety and its seizure risk is greatest in the first three days after abrupt discontinuation, so if used at all it is kept to the lowest effective dose for the shortest time (a course of the order of 4 weeks) with a slow taper; it is not a substitute for trauma-focused psychotherapy.

### 2.9 Putting the three models together

**One process, three faces.** The pathogenesis answer is strongest when the three models are stitched into a single developmental story: early, repeated, relationship-based **childhood trauma** (the trauma model) recruits a defensive walling-off of intolerable affect and memory (the psychodynamic model), and that defence is instantiated as a cortico-limbic disconnection in which the prefrontal cortex over-inhibits the limbic system (the neurobiological model), so the person is protected from unbearable feeling at the cost of an integrated, continuous sense of self. Structural dissociation names the endpoint of that process; the DES and its taxon measure it; culture shapes how it is expressed. Every effective treatment targets one of these levels, and none of them is a benzodiazepine.

| MODEL | CORE CLAIM / MECHANISM | KEY FIGURES | SIGNATURE EVIDENCE | TREATMENT IMPLICATION |
|---|---|---|---|---|
| **Psychodynamic** | **Dissociation is a defence walling off intolerable affect and memory** | Janet, Freud; Bliss, Kluft (autohypnosis) | High hypnotisability of dissociative patients; clinical case material | Phased, safety-first therapy; not forced abreaction |
| **Trauma (incl. structural dissociation)** | Early, repeated trauma drives dissociation of traumatic memory; ANP / EP split | **van der Hart, Nijenhuis, Steele; Freyd (betrayal); Putnam** | Raised DES in traumatised groups; Vonderlin & Dalenberg meta-analyses; peritraumatic dissociation predicts PTSD | Trauma history mandatory; trauma-focused, integration-oriented therapy |
| **Neurobiological** | Cortico-limbic disconnection: prefrontal overactivation inhibits the limbic system | Sierra & Berrios; Lanius | **PET (cortex up, amygdala/limbic down); over- vs under-modulation in PTSD; rTMS reverses/induces symptoms** | Reality testing intact (not psychosis); circuit-targeted and psychotherapeutic approaches |

The three complementary models of the pathogenesis of dissociative disorders, read as motive, cause and circuit; the accompanying figure carries the same three-model concept as a dissociation continuum. Grounded in Kaplan & Sadock CTP 2024.

### Dissociation

A disruption of the normally integrated functions of consciousness, memory, identity, emotion, perception and body-sense, with reality testing preserved.

### Structural dissociation

A trauma-driven "split" of the personality into an apparently-normal part and one or more emotional parts; graded primary, secondary and tertiary.

### Apparently normal personality (ANP)

The constricted, avoidant part focused on daily life, unaware of and phobic toward the traumatic material.

### Emotional personality (EP)

The part that holds the unintegrated, unprocessed traumatic memory and its raw affect.

### Peritraumatic dissociation

Dissociation occurring at or immediately after the traumatic event; a predictor of subsequent post-traumatic stress disorder.

### Cortico-limbic disconnection

The neurobiological model of dissociation: prefrontal (cortical) over-activation inhibiting limbic structures, producing hypoemotionality and detachment.

**WORKED EXAMPLE**

A woman with a history of chronic intrafamilial childhood sexual abuse presents with hours she cannot account for, a persistent sense that she is watching herself from outside her body, and two coworkers who describe her as "a different person" on some days. Read her through the three models at once. The *trauma model*: early, repeated, betrayal-laden abuse is the necessary antecedent, and her amnesia is the attachment-protective forgetting betrayal-trauma theory predicts. The *psychodynamic model*: the amnesia and the self-states are a defence that keeps unbearable memory and affect out of integrated awareness (an over-learned autohypnotic protection). The *neurobiological model*: her depersonalisation is the cortico-limbic disconnection, prefrontal over-modulation inhibiting the limbic system, which also tells you her reality testing is intact and this is not psychosis. No single model is complete; together they give the formulation, and the management is a phased trauma-focused psychotherapy, not a benzodiazepine.

| 30 | 48.7 | 3.4% | 3 |
|---|---|---|---|
| DES SCREEN CUT-OFF ABOVE WHICH SEVERE DISSOCIATIVE PSYCHOPATHOLOGY IS LIKELY (TWO-TIER THRESHOLD 20 TO 30) | MEAN DES IN DISSOCIATIVE IDENTITY DISORDER (META-ANALYSIS) | GENERAL-POPULATION PREVALENCE OF THE PATHOLOGICAL DISSOCIATIVE TAXON | LEVELS OF STRUCTURAL DISSOCIATION (PRIMARY, SECONDARY, TERTIARY) |

**HOLD THIS**

The pathogenesis of dissociation is one process told at three levels: early, repeated **childhood trauma** (the trauma model) recruits a defence that walls off intolerable affect and memory (the psychodynamic model), instantiated as a cortico-limbic disconnection in which the prefrontal cortex over-inhibits the limbic system (the neurobiological model); structural dissociation into an apparently-normal and an emotional part names the endpoint, reality testing stays intact (so it is not psychosis), and the treatment is a phased trauma-focused psychotherapy, never a long-term benzodiazepine.

## 3 · Dissociative identity disorder

**The examiner's whole target is one criterion.** **Dissociative identity disorder** is a low-volume topic that punches above its weight because it is decided by a single discriminating fact. The disorder is defined by two or more distinct personality (identity) states with a marked discontinuity in the sense of self and agency, layered on *recurrent gaps in autobiographical memory*. That memory criterion is the pivot: the **dissociative-amnesia criterion is required**, and its absence is the classic trap that turns a confident answer into a wrong one.

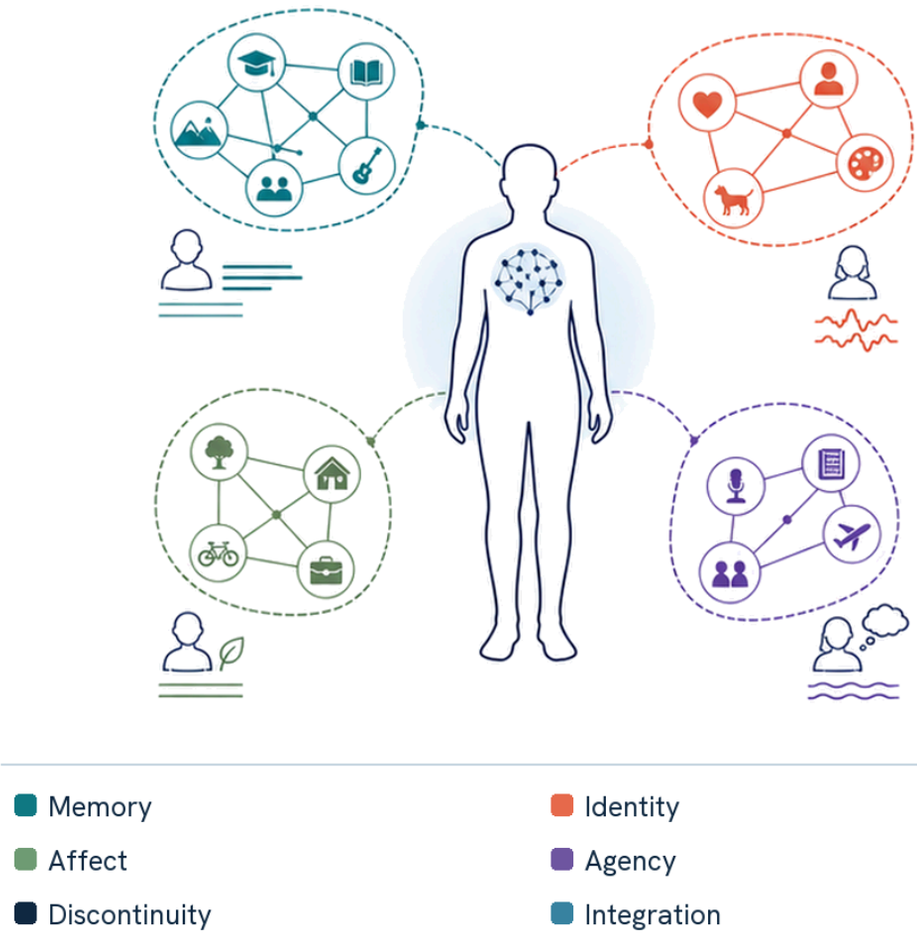



**Fig 15.5** Dissociative identity disturbance is represented as discontinuity in the integration of autobiographical memory, identity, affect and agency rather than as separate literal people.

Two things must travel together in every answer. First, the criterion set, because DID without recurrent amnesia is a different (residual) diagnosis. Second, the **controversy**, because the viva examiner reliably asks whether the disorder is a genuine post-traumatic condition or an artefact of therapy and culture, the **trauma model versus sociocognitive model** debate. The older name multiple personality disorder is worth knowing, as is the fact that the flamboyant, fully personified **alter identities** of popular culture are the exception, not the diagnostic rule.

### 3.1 Criterion A: the disruption of identity

**Two or more distinct states, discontinuity of self and agency.** DSM-5-TR Criterion A requires a disruption of identity characterised by two or more distinct personality states (which may be described in some cultures as an experience of possession), involving marked discontinuity in the sense of self and sense of agency, with related alterations in affect, behaviour, consciousness, memory, perception, cognition or sensory-motor functioning. Crucially, the elaboration of states with different names, wardrobes, handwriting and accents occurs in only a *minority* of cases and

is *not* essential to diagnosis; in most people the switching is subtle and inferred from sudden discontinuities in the sense of self, not from theatrical alter identities.

### 3.2 Criterion B: recurrent dissociative amnesia (the anchor)

**The required, most-tested criterion.** DSM-5-TR Criterion B demands **recurrent gaps** in the recall of everyday events, important personal information and/or traumatic events that are inconsistent with ordinary forgetting. This dissociative amnesia shows up as lost autobiographical memory (a wedding, all schooling before a certain age), lapses for well-learned skills (how to drive or cook), and the discovery of possessions or writings the person has no memory of acquiring. The remaining criteria fence the diagnosis: distress or impairment (C); the disturbance is *not* a normal part of a broadly accepted cultural or religious practice, and in children is not better explained by imaginary playmates or fantasy play (D); and it is not due to a substance (alcohol blackouts) or another medical condition such as complex partial seizures (E).

RULE **Identity disruption plus recurrent amnesia.** DID = two or more distinct identity states with discontinuity of self and agency AND recurrent dissociative amnesia; the amnesia is a required criterion, never an optional associated feature.

TRAP **Diagnosing DID without the amnesia.** An identity disturbance or an episode of possession with *no history of recurrent dissociative amnesia* is other specified dissociative disorder (OSDD, DSM-5-TR example 1), not DID. Do not upgrade it.

### 3.3 The ICD-11 framing and partial DID

**Executive control is the ICD-11 hinge.** ICD-11 codes DID at 6B64: two or more distinct personality states (dissociative identities) with marked discontinuities in the sense of self and agency, where at least two *recurrently take executive control* of consciousness and functioning. Its innovation is partial dissociative identity disorder (6B65): a single dominant personality state runs day-to-day life and is *intruded upon* by non-dominant states (intruding thoughts, affects, actions) that do *not* recurrently take executive control. The clean split to memorise is control (full DID: states seize control; partial DID: they only intrude).

### 3.4 Associated features, screening and the trauma load

**Intrusions that mimic psychosis, on a heavy trauma base.** People with DID report recurrent, ego-dystonic intrusions: voices (a child's voice, commenting or command voices), thoughts and impulses experienced as not their own, and made actions, phenomena that superficially resemble Schneiderian first-rank symptoms and drive the frequent misdiagnosis as schizophrenia. The overwhelming majority describe severe, chronic, early childhood trauma (abuse and neglect), and most meet criteria for comorbid PTSD (Kaplan and Sadock, CTP 2024). Screening and diagnosis use the Dissociative Experiences Scale (DES) as a self-report screen, with the structured SCID-D-R interview and the MID for diagnosis; a DES mean around **30** is the commonly cited screen threshold for a dissociative disorder (Carlson and Putnam 1993). The instrument itself is set out in the accompanying scale plate.

### 3.5 The discriminations that get tested

**Four fast separations.** Against *bipolar II with mixed features* (the commonest misdiagnosis): DID state shifts happen within minutes to hours, far too fast for even rapid-cycling bipolar disorder, and lack the classic reduced need for sleep. Against *schizophrenia*: the voices and passive-influence are dissociative intrusions in a person with intact reality testing and a trauma narrative, not a primary psychosis. Against *traumatic brain injury amnesia*: TBI carries loss of consciousness, disorientation and neurological signs, whereas dissociative amnesia sits with a discontinuity of self and agency. Against *conversion (functional neurological symptom) disorder*: conversion lacks the two-or-more distinct personality states. The factitious, imitative and malingered presentations (states built around a diagnosis, sometimes to evade responsibility) round out the differential.

### 3.6 The controversy: trauma model versus sociocognitive model

**The one debate to be able to argue both ways.** The *trauma (post-traumatic) model* holds that DID arises when severe, repeated, inescapable early-childhood trauma prevents the integration of a unified self, so identity develops in fragments. The rival *iatrogenic / sociocognitive / fantasy model* holds that DID is a socially constructed role, shaped by therapist suggestion (leading questions, hypnosis), cultural and media scripts, and high fantasy-proneness, rather than a naturally occurring post-traumatic disorder (Kaplan and Sadock, CTP 2024). This is the **trauma model versus sociocognitive model** question in one line. The examinable rejoinder in favour of the trauma model is that controlled neuroimaging and psychophysiology (the Reinders studies) distinguish genuine DID identity states from high-fantasy-prone individuals asked to simulate them, which a purely role-play account struggles to explain.

**WORKED EXAMPLE**

A woman in her late twenties is referred as "treatment-resistant bipolar II". She describes hearing a young child's voice narrating her actions, finds clothes and written notes she has no memory of buying or writing, and loses blocks of time at work. Her mood and behaviour shift within an hour, not over days, and she has a documented history of chronic childhood sexual abuse. The rapid within-hour switching, the recurrent amnesia (the notes and lost time), the discontinuity of self and agency, and the trauma history make this dissociative identity disorder, not bipolar II and not schizophrenia. Confirming recurrent dissociative amnesia is what secures the diagnosis; had it been absent, the correct label would be other specified dissociative disorder.

**INDIA**

The genuine India device here is culture, not pharmacology. Possession and trance presentations, in which an external spirit, deity or ancestor is felt to take over, are a common idiom of distress in Indian settings and can look like the possession form of DID. DSM-5-TR Criterion D and the cultural formulation are the safeguard: a possession or trance that is a *sanctioned, non-distressing part of a broadly accepted religious or cultural practice is not a disorder at all*. It becomes DID only when it is involuntary, unwanted, recurrent outside accepted ritual, and paired with recurrent amnesia and impairment. In practice such presentations are too often sedated (the reflex reach for a benzodiazepine such as alprazolam) or routed to faith healers rather than assessed with a proper cultural formulation and a trauma history. The transcultural framing of possession and trance is developed in the Consultation-liaison, geriatric & transcultural psychiatry notes.

| **2 or more** | **1.5%** | **30** |
|---|---|---|
| DISTINCT IDENTITY STATES, DSM-5-TR CRITERION A | 12-MONTH PREVALENCE (DSM-5-TR, SMALL US COMMUNITY SAMPLE) | DES SCREEN CUT-OFF FOR A DISSOCIATIVE DISORDER (CARLSON AND PUTNAM 1993) |

**PEARL**

The showy, fully named alter identities are the minority presentation. Most DID is quiet: subtle switching, chronic amnesia and passive-influence intrusions, which is exactly why it is missed or mislabelled as bipolar disorder or psychosis for years.

**GOOD TO REMEMBER**

- **Dissociative identity disorder (the syndrome):** the defining criteria are two or more distinct personality (identity) states with a marked discontinuity in the sense of self and agency (Criterion A) PLUS recurrent dissociative amnesia inconsistent with ordinary forgetting (Criterion B, required), causing impairment, not accounted for by an accepted cultural or religious practice, a substance or another medical condition; the discriminator from other specified dissociative disorder is the presence of the recurrent amnesia, and the first step is to confirm that amnesia criterion before ever applying the DID label.

**HOLD THIS**

DID needs BOTH halves: two or more distinct identity states with discontinuity of self and agency AND recurrent dissociative amnesia; drop the amnesia and it is other specified dissociative disorder, and the whole examinable debate is the trauma model versus sociocognitive model, with controlled neuroimaging that separates patients from simulators tilting the evidence toward trauma.

For the broader dissociative-disorders classification, the pathogenesis models and the conversion boundary, see the neighbouring topics; for the dissociative subtype of PTSD, the Anxiety, OCD & trauma-related disorders notes; for the somatic-symptom, functional and factitious boundary, the Somatic-symptom, sexual, impulse-control disorders & suicide notes; and for grounding and the place of hypnosis in dissociative work, the Psychotherapies, clinical assessment, MSE & nosology notes.

## 4 · Conversion (dissociative neurological symptom) disorder

**One idea rewrites this whole topic.** **Conversion disorder** is no longer a diagnosis of exclusion. The modern rule, from Stone and Carson, is that it is ruled *in* at the bedside by **positive signs** of internal inconsistency, findings that demonstrate the nervous system working normally when attention is diverted, and only secondarily by the absence of a lesion. Learn the signs and this becomes one of the most answerable clinical topics in the paper; miss the principle and you will either over-call a real neurological disease or, worse, sedate a functional presentation and never treat it.

**Dissociative neurological symptom disorder** is the ICD-11 name (code 6B60), which keeps the condition *inside* the dissociative family; DSM-5-TR files the same entity under the somatic symptom disorders as **functional neurological symptom disorder (conversion disorder)**, and the umbrella clinical term is **functional neurological disorder** (FND). Whatever the label, the examinable core is a small set of **rule-in signs** across four presentations: functional weakness and paralysis, functional movement and tremor, functional gait, and the sensory or special-sense forms, plus the **dissociative convulsions** that must be separated from true **epilepsy**. The two figures for this topic set the signs out as a positive-signs plate and a conversion-versus-**organic** discriminator grid to be memorised as images. The video-EEG work-up of the seizure form is developed in the Epilepsy & psychiatry notes; the wider somatic-symptom and factitious boundary in the Somatic-symptom, sexual, impulse-control disorders & suicide notes.

**Fig 15.6** The positive rule-in signs of a functional neurological disorder, grouped by domain - each one demonstrating internal inconsistency at the bedside.

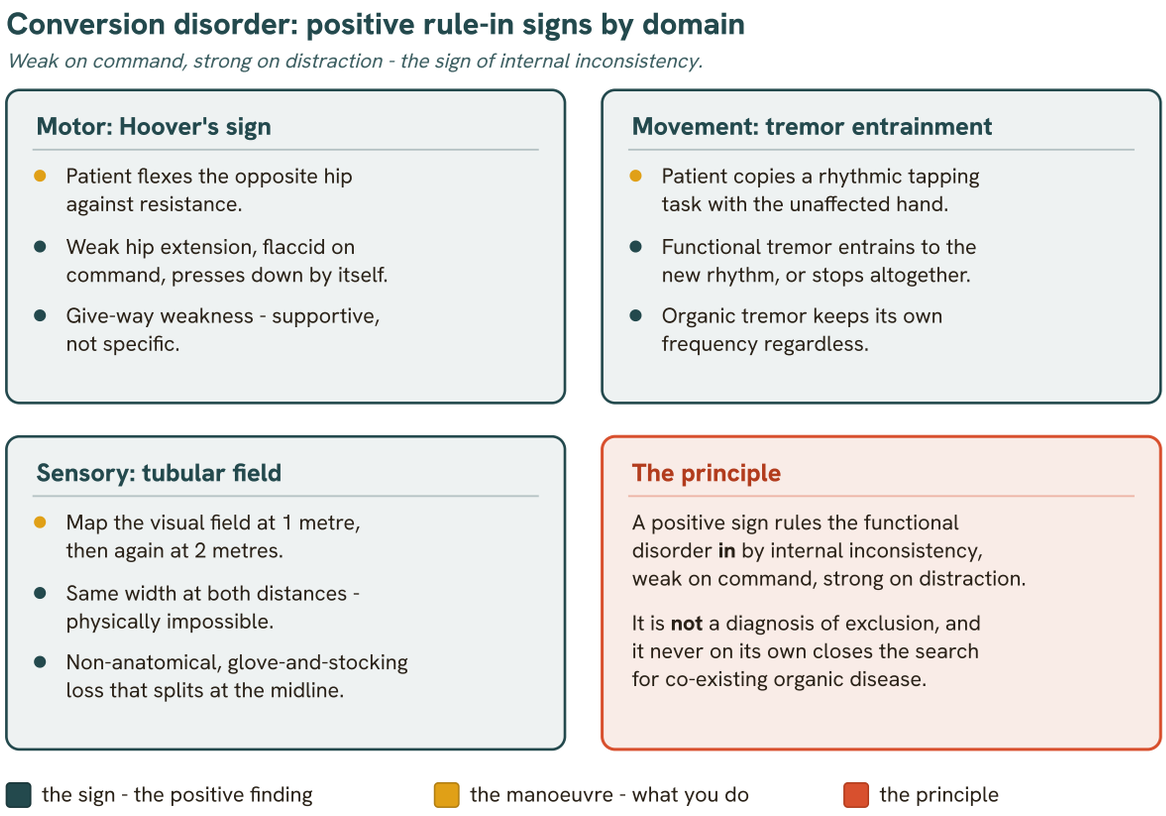


**Fig 15.7** How to tell a functional (conversion) presentation from organic disease across five features, and why a positive sign never closes the organic work-up.

| feature | functional (conversion) | organic disease |
|---|---|---|
| **diagnostic basis** | ruled in on a positive sign of internal inconsistency | a fixed anatomical or structural lesion |
| **consistency** | internally inconsistent, variable, distractible | consistent and reproducible on testing |
| **signs** | positive rule-in signs present: Hoover, entrainment, tubular field | hard neurological signs (reflexes, wasting, sensory level) |
| **course and onset** | often abrupt, may be linked to stress | progressive, vascular, or stepwise |
| **imaging and investigations** | investigations and imaging typically normal | structural or electrical abnormality (MRI, EEG) |

**Both can coexist.**
A positive functional sign never ends the organic work-up.

functional (conversion) organic disease

**Fig 15.8** Reading a convulsion: the reliable signs that separate a dissociative (non-epileptic) attack from an epileptic seizure, and the classic signs that do not.

| reliable discriminator | dissociative (non-epileptic) convulsion | epileptic seizure |
|---|---|---|
| awareness during bilateral shaking | often preserved; can recall or interrupt it | lost in a bilateral tonic-clonic seizure |
| duration and pattern | long and fluctuating, over 2 to 3 minutes | short and stereotyped, under 2 minutes |
| the eyes | closed, with active resistance to opening | typically open |
| head movement | side-to-side | not side-to-side |
| post-ictal prolactin (at 10 to 20 min) | no rise | rises to over twice baseline (generalised) |
| ictal video-EEG (gold standard) | normal during an apparent attack | epileptiform discharge |

**unreliable on their own**

occur in both and do not decide either way:
tongue-biting, urinary incontinence, injury, pelvic thrusting

dissociative convulsion · epileptic seizure · unreliable (occurs in both)

### 4.1 What conversion disorder is, and what it is not

**Genuine symptoms, incompatible with disease, ruled in by a sign.** Conversion disorder is one or more symptoms of altered voluntary motor or sensory function that are *genuinely experienced* (not feigned) yet demonstrably incompatible with recognised neurological or medical disease (DSM-5-TR Criterion B; ICD-11 6B60). DSM-5-TR deliberately *removed* the old requirement for an identifiable psychological stressor, so a stressor is neither necessary nor sufficient, and la belle indifference (bland unconcern) is unreliable and is *not* a diagnostic criterion. DSM-5-TR specifies the symptom type, which is the clinically useful map: with weakness or paralysis, with abnormal movement, with swallowing or speech symptoms, with attacks or seizures, with anaesthesia or sensory loss, with special-sensory symptoms, or mixed. The whole diagnosis now turns on Criterion B, the demonstration of internal inconsistency, not on Criterion D absence of disease.

RULE **Rule in, do not rule out.** Conversion disorder is a positive diagnosis made on a sign of internal inconsistency (Hoover, tremor entrainment, tubular field), never on the mere absence of a lesion or the presence of a stressor.

### 4.2 The positive-signs principle: internal inconsistency

**The system works when attention is elsewhere.** The unifying mechanism behind every rule-in sign is that the symptom depends on abnormally focused attention: divert attention and the "lost" function returns, revealing the pathways to be intact (Stone and Carson; New Oxford 2024). This is *internal inconsistency*, an incompatibility between the deficit shown on formal testing and the function that appears automatically or when the patient is distracted. It is a different logic from anatomical implausibility: a glove-and-stocking sensory loss is suggestive because it defies dermatomes, but a positive Hoover's sign is far stronger because it demonstrates the extensors *firing* a moment after they were "paralysed". The accompanying figure lays the signs out by domain as a single positive-signs plate. A skilled neurologist can make the positive diagnosis; the psychiatric assessment then secures a complete history and addresses the comorbid anxiety, panic, depression and PTSD that often travel with it (New Oxford 2024).

### 4.3 Functional weakness and paralysis: Hoover's sign

**The single most testable sign in psychiatry.** Hoover's sign exploits the automatic synergy of hip extension. With the patient supine, direct hip extension of the "weak" leg is flaccid; but when the patient flexes the *opposite* hip against resistance, the weak leg's extensors fire automatically and press down into the examiner's hand (Kaplan 2024). Weak on command, strong on distraction: that is a positive Hoover's sign, and it rules the diagnosis in at the bedside without imaging. Its sibling, the hip abductor sign, shows the same reversal in hip abduction. A give-way weakness (collapsing, ratchety power that builds then suddenly yields) and a global pattern of weakness affecting extensors and flexors equally are further pointers, but note the honest caveat: give-way weakness is *supportive, not specific*, because pain and poor effort in genuine organic disease can produce it too, whereas Hoover's sign is the discriminating one.

**WORKED EXAMPLE**

A woman develops a dense right-leg "paralysis" after a family argument. Direct right hip extension is flaccid. You cup your hand under the right heel and ask her to flex the *left* hip against your other hand; as she does, the right heel drives firmly down into your palm. The extensors she could not fire on command have fired automatically. That is a positive Hoover's sign, a rule-in for conversion disorder, elicited in seconds and without a scanner, and it doubles as the most persuasive thing you can later show her when you explain the diagnosis.

### 4.4 Functional movement disorder and tremor: the entrainment test

**A tremor that copies the other hand is functional.** The tremor entrainment test is the movement-domain equivalent of Hoover's sign. The patient with a unilateral tremor is asked to copy a rhythmical tapping movement with the *unaffected* limb; a functional tremor will *entrain* to the new rhythm (change its frequency to match), stop altogether, or the patient becomes unable to perform the simple copied rhythm at all (DSM-5-TR; New Oxford 2024). An organic tremor keeps its own frequency regardless. Other positive movement signs are a *fixed dystonic posture* (characteristically a flexed hand or a plantar-flexed, inverted ankle) and a typical functional facial overactivity (orbicularis or platysma over-contraction with jaw deviation) that masquerades as

facial weakness. The distractibility that defines these signs is the whole point: attention drives the symptom, so shifting attention exposes it.

### 4.5 Functional gait

**Bizarre, effortful, and better when unwatched.** The functional gaits are among the most recognisable presentations. A *dragging gait* keeps the forefoot in contact with the floor with the hip held externally or internally rotated, a pattern that fits no pyramidal or extrapyramidal disease. Astasia-abasia is a wide-based, staggering, dramatic and irregular gait with exaggerated near-falls yet a conspicuous absence of injury, the effort of not falling being greater than that of walking normally. The bedside rule-in is distraction: a patient with an apparently positive Romberg's test is asked to guess numbers written on the back or to carry out a complex task on a phone, and the balance improves markedly (New Oxford 2024). These motor and convulsive forms are especially common in Indian and other lower-income settings.

### 4.6 Sensory and special-sense presentations

**Boundaries that disobey anatomy and physics.** The classic sensory form is a glove-and-stocking anaesthesia: a loss of all modalities with a sharp boundary at the wrist or ankle that does not conform to any dermatome, and a splitting of vibration sense exactly at the midline, even though a tuning fork on the sternum or skull conducts through bone to both sides. In vision, the rule-in signs are the tubular field (also called tunnel vision): a visual-field defect of the *same* width at 1 metre as at 2 metres, which is physically impossible because a real field subtends a wider area with distance. The *fogging test* proves a claimed monocular blindness is functional, by progressively fogging the good eye with lenses while the patient reads an acuity chart; retained acuity at the end means the "blind" eye is doing the reading. Functional deafness and non-anatomical hemi-anaesthesia complete the group.

**GOOD TO REMEMBER**

- **Conversion / dissociative neurological symptom disorder (the syndrome):** defining criterion is a motor or sensory symptom, genuinely experienced, shown by a *positive* sign of internal inconsistency to be incompatible with neurological disease (Hoover's sign, tremor entrainment, tubular field); it is a *rule-in* diagnosis, *not* one of exclusion, no stressor is required (DSM-5-TR) and la belle indifference is non-diagnostic. First step: elicit a positive sign at the bedside.

### 4.7 Dissociative (non-epileptic) convulsions: the differentiation from epilepsy

**The seizure form, and how it parts from true epilepsy.** Dissociative convulsions (dissociative or psychogenic **non-epileptic** seizures, ICD-11 6B60.4 "with non-epileptic seizures") are paroxysmal episodes of altered movement, sensation or responsiveness that resemble an epileptic seizure but arise without epileptiform electrical discharge. The examinable skill is the **differentiation** from epilepsy on semiology (New Oxford 2024; Companion Box 13.12). Features that point to a dissociative convulsion are: preserved awareness during bilateral shaking, with the patient later able to recall being in the attack or even able to interrupt it; a long duration (often longer than 2 to 3 minutes, waxing and waning); closed eyes with active resistance to eye opening; side-to-side head movement and out-of-phase, asynchronous limb thrashing; and ictal or immediate post-ictal weeping. A post-ictal serum prolactin drawn 10 to 20 minutes after onset

that *rises to more than twice baseline* supports a true generalised or complex-partial seizure, so the *absence* of a prolactin rise is consistent with a dissociative convulsion, though the test has limited value (false positives and negatives) and a normal result never excludes epilepsy. The gold standard remains a normal *ictal* video-EEG captured during an apparently generalised attack (Companion), cross-referenced to the Epilepsy & psychiatry notes.

| FEATURE | POINTS TO A DISSOCIATIVE CONVULSION | POINTS TO EPILEPSY (OR UNHELPFUL) |
|---|---|---|
| **Awareness in bilateral shaking** | **Preserved; can recall the attack or interrupt it** | Lost in a generalised tonic-clonic seizure |
| **Duration** | Often longer than 2 to 3 minutes, fluctuating | Usually under 2 minutes, stereotyped |
| **Eyes** | **Closed, with resistance to eye opening** | Typically open |
| **Movements** | Side-to-side head movement, out-of-phase asynchronous thrashing | Rhythmic, synchronous, evolving clonic jerks |
| **Weeping** | Ictal or immediate post-ictal crying | Rare |
| **Post-ictal prolactin (10 to 20 min)** | No rise (consistent with, not proof of) | Rise to more than twice baseline (limited value) |
| **Ictal video-EEG** | **Normal during an apparent generalised attack** | Epileptiform discharge (gold-standard test) |
| **Tongue-biting, incontinence, pelvic thrust, injury** | Unhelpful, occur in both; do not use to decide either way (Companion) | |

Semiological differentiation of dissociative (non-epileptic) convulsions from epilepsy; the last row lists the classic unreliable discriminators.

**GOOD TO REMEMBER**

- **Dissociative convulsions (the seizure form):** defining criterion is a seizure-like attack with positive rule-in features (preserved awareness in bilateral shaking, duration over 2 to 3 minutes, closed and resisted eyes, no post-ictal prolactin rise) and a *normal ictal video-EEG*, the gold-standard confirmation; tongue-biting, incontinence and pelvic thrusting do NOT distinguish it from epilepsy. First step: capture the attack on video-EEG.

### 4.8 The conversion-versus-organic discrimination and its trap

**A positive sign never cancels the duty to screen.** The safe practice of this topic is a two-way **discrimination**: rule the functional disorder *in* on a positive sign, but keep screening for organic disease because the two coexist more often than expected. A sizable minority of patients with non-epileptic seizures *also* have epilepsy (Kaplan 2024), and functional signs are described alongside real disease. The accompanying discriminator grid contrasts the functional and organic pictures side by side. The classic misdiagnoses run in the mirror direction: early multiple sclerosis, a spinal cord lesion, frontal-lobe epilepsy (which can look bizarre and show a normal surface EEG), stiff-person syndrome, Wilson's disease and autoimmune encephalitis have all been

mislabelled conversion because the presentation seemed emotional or a stressor was present. So the positive sign rules the functional diagnosis in, but a progressive course, hard neurological findings, or an atypical picture reopens the organic search.

**TRAP** **Missing the organic cause behind a "conversion".** A stressor or a dramatic presentation is not evidence of a functional disorder; early MS, a cord lesion, frontal-lobe epilepsy or autoimmune encephalitis can all mimic it, and functional signs coexist with real disease, so never let a positive sign close the organic work-up in an atypical or progressive case.

### 4.9 Management: positive explanation, physiotherapy, CBT

**The explanation is the first treatment.** Management rests on three evidence-based pillars (New Oxford 2024). First, a clear *positive explanation* of the diagnosis: take the problem seriously, give it a positive diagnostic label rather than a list of what it is not, *show the patient their own positive sign* (the Hoover's sign, the tremor stopping on entrainment), use the software-versus-hardware metaphor to emphasise reversibility, and signpost written resources such as neurosymptoms.org. Second, *physiotherapy* is first-line for the functional motor forms (limb weakness and functional movement disorders), using distraction and retraining rather than generic stroke-style physiotherapy; a specific programme achieved a good outcome in about 72 percent despite years of symptoms. Third, *cognitive behavioural therapy*, generic tailored CBT for mixed functional symptoms and a dedicated CBT for dissociative seizures (number needed to treat around 5 for seizure remission). There is *no* specific drug for conversion disorder itself; medication is only for comorbid anxiety or depression, for which the SSRIs are the first-line agents taught in the Mood disorders: pharmacotherapy notes, and hypnosis and grounding sit in the Psychotherapies, clinical assessment, MSE & nosology notes.

| 2 min | 10 to 20 min | 2× |
|---|---|---|
| DURATION ABOVE WHICH A CONVULSION FAVOURS A DISSOCIATIVE ATTACK | POST-ICTAL PROLACTIN SAMPLING WINDOW AFTER SEIZURE ONSET | PROLACTIN RISE OVER BASELINE THAT SUPPORTS A TRUE SEIZURE |

**INDIA**

Two genuine India devices meet here. First, the culture-bound one: dissociative motor and convulsive presentations, and the possession and trance states, are among the most frequent dissociative presentations in Indian practice, and the convulsive form is recorded as "very common" in India and other developing countries. Read through a cultural formulation, not the surface picture: a possession or trance sanctioned within an accepted religious practice is not a disorder, a boundary developed in the Consultation-liaison, geriatric & transcultural psychiatry notes. Second, the pharmacological one: the reflex Indian response to a dramatic dissociative or convulsive presentation is too often a sedative, and India has a well-recognised benzodiazepine over-prescription problem with alprazolam (Alprax, Restyl) as the default anxiolytic. A benzodiazepine treats nothing in conversion disorder, risks dependence, and is exactly the wrong move; the Indian Psychiatric Society and the wider benzodiazepine-sparing position favour a positive explanation, physiotherapy and CBT instead. The sedative and benzodiazepine use disorder as an addiction is covered in the Substance use disorders notes.

**PEARL**

The most powerful therapeutic tool is the diagnostic sign itself. Demonstrating a patient's own Hoover's sign or entrainment, and explaining that a leg that moves automatically is a leg that *can* move, converts the bedside examination into the opening move of treatment: it proves the diagnosis, shows reversibility, and replaces “there is nothing wrong” with “here is what is wrong and here is why it can get better”.

**HOLD THIS**

Conversion disorder is ruled *in* on positive signs of internal inconsistency, weak on command but strong on distraction (Hoover's sign, tremor entrainment, tubular field), never out by exclusion; and its seizure form is separated from epilepsy by preserved awareness, closed and resisted eyes, a long fluctuating attack, no post-ictal prolactin rise, and above all a normal ictal video-EEG.

For the wider dissociative-disorders map and the pathogenesis models see the neighbouring topics; the somatic-symptom, functional and factitious boundary sits in the Somatic-symptom, sexual, impulse-control disorders & suicide notes; the video-EEG work-up of dissociative convulsions in the Epilepsy & psychiatry notes; and the transcultural reading of possession and trance in the Consultation-liaison, geriatric & transcultural psychiatry notes.

## 5 · Depersonalisation-derealisation disorder

**One discriminator carries this whole topic. Depersonalisation-derealisation disorder** is a low-volume entity that is decided by a single question: is reality testing intact? The patient describes the world and the self as strange, flat and unreal, yet *knows* that this is only how things feel, not how they truly are. That preserved reality check is what separates the disorder from a psychotic illness, and forgetting it is the classic error that turns detachment into a mistaken diagnosis of schizophrenia.

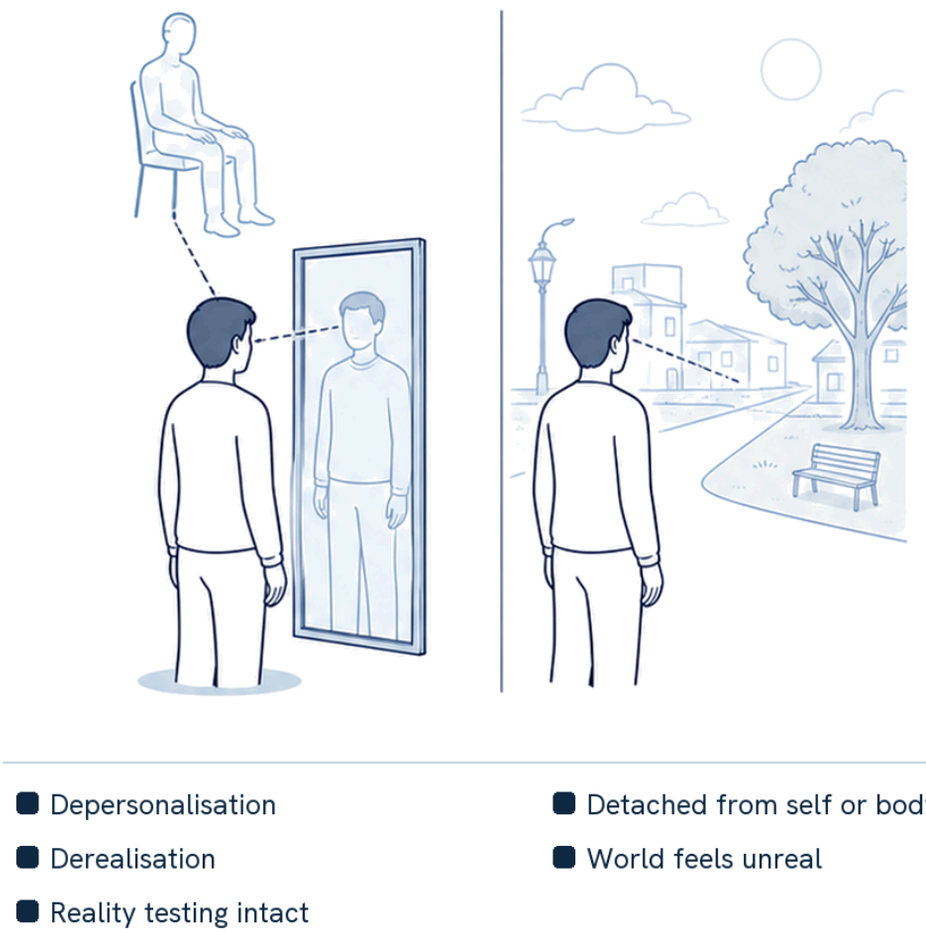



**Fig 15.9** Depersonalisation is detachment from one's self, mental processes or body; derealisation is an altered sense of the external world as unreal. In both, reality testing remains intact.

Two experiences travel together in the name. **Depersonalisation** is a sense of **unreality** or **detachment from self**, one's own mind, feelings, actions or body. **Derealisation** is the same unreal, detached quality applied to the surroundings, the world seen as if behind glass or like a stage set. The examiner reward is small and precise: the definition, the intact-reality-testing anchor, the fact that transient forms are extremely common and benign, and the awkward truth that *no drug has an established place* in treatment.

### 5.1 The two halves: depersonalisation and derealisation

**Detachment from the self, detachment from the world.** The disorder is built from two symptom clusters that may occur singly or together (DSM-5-TR; ICD-11 6B66).

#### Depersonalisation

Unreality, detachment or being an outside observer of one's own thoughts, feelings, body or actions; emotional or physical numbing, a distorted sense of time, an unreal or absent self.

#### Derealisation

Unreality or detachment applied to surroundings; people or objects felt as unreal, dreamlike, foggy, lifeless or visually distorted (as if behind glass).

#### The "as if" quality

The experiences are described in the conditional, "as if" I were not real, "as if" the world were a painting; the person never asserts that it is *literally* so.

### 5.2 The DSM-5-TR criteria and the intact-reality-testing anchor

**Persistent or recurrent, with the reality check preserved.** DSM-5-TR requires persistent or recurrent experiences of depersonalisation, derealisation or both (Criterion A); crucially, *during these experiences reality testing remains intact* (Criterion B). The symptoms cause clinically significant distress or impairment (C); they are not attributable to a substance or another medical condition, notably seizures (D); and they are not better explained by another mental disorder such as schizophrenia, panic disorder, major depression or another dissociative disorder (E). Because transient depersonalisation is extremely common in the general population and after cannabis, ketamine or hallucinogens, the label is reserved for presentations that are persistent, recurrent and impairing.

RULE **Intact reality testing is the diagnosis.** Persistent unreality of self and/or world with an "as if" quality and a preserved reality check is depersonalisation-derealisation disorder; the moment the patient believes the unreality is literally true, you have left the diagnosis for the psychotic spectrum.

TRAP **Mislabelling depersonalisation-derealisation as psychosis.** "The world feels unreal, like a film" is not a delusion. Historically these patients were read as psychotic-spectrum and started on antipsychotics, for which there is no evidence and which can deepen the very emotional deadening they complain of (Kaplan and Sadock, CTP 2024).

### 5.3 Onset, course and the trauma signature

**An adolescent onset with a heavy comorbidity load.** The mean age of onset is **16 years**, and the disorder can begin in early or middle childhood; onset for the first time in later life is unusual and should prompt a search for an organic or substance cause (DSM-5-TR). The course splits into rough thirds: about one in three run an episodic, relapsing-remitting course, one in three a chronic course, and one in three an episodic course that becomes chronic (Kaplan and Sadock, CTP 2024). Comorbidity is the rule rather than the exception, with major depressive disorder in around **73%** and an anxiety disorder in around **64%**, which is why most patients are first treated for the secondary mood and anxiety symptoms. The trauma signature is distinctive: unlike the sexual-abuse loading of dissociative identity disorder, it is *emotional (verbal) abuse* that most specifically predicts depersonalisation-derealisation scores.

### 5.4 Screening: the Cambridge Depersonalization Scale

**A dedicated instrument from the Maudsley unit.** The standard screen is the Cambridge Depersonalization Scale (CDS), developed by Sierra and Berrios at the Depersonalization Research Unit (Maudsley Hospital, London); the same unit and Daphne Simeon's group in the United States generated most of the modern clinical picture. A depersonalisation-derealisation

subscale of the broader Dissociative Experiences Scale (the DES-D) is also used. The instrument forms are set out centrally in the accompanying scale plate; carry the CDS as the named screen and read its cut-off off the reproduced plate rather than committing an unverified threshold to memory.

### 5.5 Management: no drug cures it

**Psychotherapy first, medication only for the comorbidity.** The single most examinable management fact is a negative one: extensive treatment histories show *no* established pharmacotherapy for the core disorder, with benzodiazepines, serotonin reuptake inhibitors and stimulants only partially helpful in some patients, and no evidence at all for antipsychotics (Kaplan and Sadock, CTP 2024). Antipsychotics are reserved strictly for overwhelming associated anxiety or mood disturbance and may worsen the emotional numbing. The mainstays are a range of psychotherapies (psychodynamic, cognitive, cognitive-behavioural, hypnotherapeutic and supportive, with no comparative data favouring one) plus definitive treatment of the comorbid depression or anxiety. Where a short course of an anxiolytic is used for a crisis, the benzodiazepine-sparing short-course rule still holds: it is a brief measure, not a long-term treatment for the unreality itself. The grounding techniques and the place of hypnosis are developed in the Psychotherapies, clinical assessment, MSE & nosology notes; the SSRIs and SNRIs as first-line anxiolytics for the comorbid anxiety are in the Mood disorders: pharmacotherapy notes and the Anxiety, OCD & trauma-related disorders notes.

**WORKED EXAMPLE**

A man in his early twenties is referred with a query psychotic illness. Since his late teens he has felt "outside" himself, watching his own actions from a step to one side; his voice sounds tinny and far away, his emotions muffled, the street "like a painting". He is frightened and desperate to "get back to me", but when asked he is clear that the world is not *actually* unreal, it only feels that way. The persistence since adolescence, the detachment from self and surroundings, the distress, and above all the intact reality testing make this depersonalisation-derealisation disorder, not schizophrenia. Confirming the preserved reality check is what secures it; starting an antipsychotic would be both unsupported and likely to deepen the emotional deadening.

| | | |
|---|---|---|
| **16 yrs** | **73%** | **1 in 3** |
| MEAN AGE OF ONSET (DSM-5-TR) | CARRY COMORBID MAJOR DEPRESSION (KAPLAN 2024) | FOLLOW A CHRONIC COURSE (KAPLAN 2024) |

**INDIA**

The genuine India device here is what *not* to prescribe. India carries a well-recognised benzodiazepine over-prescription problem, with alprazolam (Alprax or Restyl) functioning as the default anxiolytic reached for almost reflexively when a patient describes unreality, numbness or anxious detachment. Depersonalisation-derealisation disorder is precisely the presentation where that reflex fails: no benzodiazepine treats the core symptom, dependence and rebound follow long use, and the Indian Psychiatric Society position, like BAP and NICE, is benzodiazepine-sparing, a short course for crisis only. The safer move is to name the disorder, reassure about the intact reality check, treat the comorbid depression or anxiety, and refer for psychotherapy.

**PEARL**

Depersonalisation is best understood as a "hard-wired" protective response, an inhibitory dampening of overwhelming affect that is adaptive in acute terror or intoxication and pathological only when it persists. That is why transient unreality after trauma, a panic attack or cannabis is normal and self-limiting, and why the disorder sits at the anxious, not the psychotic, end of the spectrum.

**GOOD TO REMEMBER**

- **Depersonalisation-derealisation disorder (the syndrome):** the defining criterion is persistent or recurrent depersonalisation (unreality or detachment from self) and/or derealisation (unreality or detachment from the surroundings) during which *reality testing remains intact* (DSM-5-TR Criterion B), causing distress or impairment and not due to a substance, seizure or another disorder; the discriminator from psychosis is that intact reality check and the "as if" quality, and the first step is always to confirm reality testing is preserved before the label is applied.

**HOLD THIS**

Persistent unreality of self and world with an "as if" quality and INTACT reality testing is depersonalisation-derealisation disorder, onset around 16 years, screened with the Cambridge Depersonalization Scale, and it has no established drug treatment, so the answer is psychotherapy plus treating the comorbid mood or anxiety, never a reflex antipsychotic or a long-term benzodiazepine.

For the classification map and the pathogenesis models this disorder sits within, see the neighbouring dissociative-disorders topics; for the dissociative subtype of PTSD and the anxiolytics in anxiety, the Anxiety, OCD & trauma-related disorders notes; for possession and trance in the transcultural frame, the Consultation-liaison, geriatric & transcultural psychiatry notes; and for grounding and hypnosis, the Psychotherapies, clinical assessment, MSE & nosology notes.

## 6 · Dissociative amnesia and fugue

**One discrimination decides this topic. Dissociative amnesia** is an inability to recall important autobiographical information, usually of a traumatic or stressful nature, that is too extensive to be ordinary forgetting. The examiner is not really testing whether you can recite the criterion; he is

testing whether you can separate it from an *organic* amnesia at the bedside, because the two are worked up in opposite directions. The whole answer turns on one pattern: a retrograde loss of already-stored personal memory with an intact ability to lay down new memory, on a trauma trigger, and in the absence of a neurological or substance cause.




**Fig 15.10** Dissociative amnesia produces an autobiographical memory gap that exceeds ordinary forgetting. Fugue adds apparently purposeful travel or wandering with disruption of autobiographical identity.

The disorder was long called psychogenic amnesia (the ICD-10 and older term), and dissociative fugue is now folded into it as a specifier rather than standing as a separate disorder. Keep two moves ready: name the *type* of amnesia (the commonest is a **localised amnesia**), and rule *in* the dissociative picture against the organic mimics, transient global amnesia, the amnestic (Korsakoff) syndrome and complex partial seizures, by the direction of the memory loss and the fate of personal identity.

### 6.1 What dissociative amnesia is

**A reversible retrieval deficit for autobiographical memory.** DSM-5-TR Criterion A is an inability to recall important autobiographical information, usually of a traumatic or stressful

nature, that is inconsistent with ordinary forgetting. The memory has been successfully stored and would ordinarily be freely recollected, so the deficit is one of *retrieval* and is potentially reversible; that is precisely how dissociative amnesia differs from the amnesias of neurobiological damage or toxicity, which impair storage. Criterion B requires distress or impairment; Criterion C excludes a substance and a neurological or other medical condition (complex partial seizures, transient global amnesia, a closed head injury); Criterion D excludes dissociative identity disorder, PTSD, acute stress disorder, somatic symptom disorder and a neurocognitive disorder. In DSM-5-TR the deficit is primarily *retrograde* and, except in the rare continuous type, there is no ongoing amnesia for contemporary life events.

### 6.2 The five types (name the shape of the gap)

**Localised is the commonest.** DSM-5-TR and ICD-11 define the recognised types by the shape of the memory gap, and the examiner expects you to label the pattern.

#### Localised

inability to recall all events in a circumscribed period, usually the period around the stressor (the commonest form)

#### Selective

recall of some but not all of the events within that circumscribed period

#### Generalised

failure to recall one's entire life, including personal identity (rare, acute, and it usually brings the person to the police or emergency services)

#### Continuous

failure to recall each new event as it occurs; this is the *anterograde* form of dissociative amnesia, and it is rare

#### Systematised

loss of a specific category of memory, for example all memories of one person or of the family

### 6.3 Dissociative fugue (now a specifier)

**Purposeful travel plus amnesia for identity.** DSM-5-TR codes fugue as the specifier "with dissociative fugue" (F44.1): apparently **purposeful travel** or bewildered wandering that is associated with amnesia for identity or for other important autobiographical information. It is commonly associated with generalised dissociative amnesia, has a sudden onset under severe stress, and terminates abruptly, with recovery of the earlier life and a residual amnesia for the episode itself. Both DSM-5-TR and ICD-11 judge that there are insufficient data to keep fugue as a disorder distinct from dissociative amnesia; ICD-11 makes it a presence specifier of 6B61 (6B61.0 with, 6B61.1 without). Note the nosological shift that gets tested: the older criterion of "assumption of a new identity" during the episode would now meet criteria for dissociative identity disorder, so the classic full identity-swap fugue has been reallocated.

### 6.4 The critical discrimination: dissociative versus organic amnesia

**Direction of the loss, and the fate of identity.** This is the single most examinable separation on the topic, and it is a positive comparison, not a hunch. Dissociative amnesia is a retrograde **loss**

**of autobiographical memory** with new learning intact and personal identity frequently lost; the organic amnestic syndrome is the mirror image.

| FEATURE | DISSOCIATIVE AMNESIA | ORGANIC AMNESIA |
|---|---|---|
| **Predominant deficit** | **retrograde: loss of stored autobiographical memory** | anterograde: failure to form new memories, with a retrograde gradient |
| **New learning (anterograde)** | **intact** | impaired |
| **Personal identity** | **often lost (name, whole life history)** | preserved (the person knows who they are) |
| **Trigger** | acute psychological trauma or stress | a neurological insult or a substance |
| **Onset and recovery** | sudden onset, can recover suddenly and completely | tracks the lesion, recovery often incomplete |
| **Consciousness and signs** | clear sensorium, neurological examination normal | clouding or disorientation, focal signs may appear |

The bedside separation of dissociative from organic amnesia rests on the direction of the deficit and on whether personal identity is lost.

RULE **Retrograde loss of identity with intact new learning is dissociative.** Dissociative amnesia loses the stored past and often the personal identity while new memories still form; the organic amnestic syndrome does the reverse, failing to lay down new memory while identity is preserved.

### 6.5 Ruling in against the organic mimics

**Rule the picture in, do not just assume it.** The same positive-versus-organic logic that governs conversion applies here: dissociative amnesia and fugue are rule-in diagnoses that still require the organic mimics to be excluded (Criterion C). Transient global amnesia gives an abrupt, isolated anterograde amnesia with repetitive questioning, preserved personal identity, typically in an older adult, and it resolves within 24 hours. Complex partial seizures and temporal lobe epilepsy can produce an epileptic wandering with no assumption of a new identity, with confusion or disorientation during the episode, episodes not tied to a psychological stressor, and an abnormal EEG. The amnestic (Korsakoff) syndrome gives a dense anterograde amnesia with confabulation on a thiamine-deficiency base. Head injury, post-ictal states and a substance blackout complete the exclusions. Screening for dissociation itself uses the Dissociative Experiences Scale (DES), where a mean around 30 flags a probable dissociative disorder; the instrument is set out in the accompanying scale plate.

TRAP **Labelling a wandering, amnesic patient "dissociative" too soon.** Purposeful travel with amnesia can be an epileptic automatism, transient global amnesia or a post-ictal fugue; missing the organic cause is the classic error. Get an EEG, imaging and a substance history before the label sticks. The video-EEG work-up that separates epileptic from dissociative events is set out in the Epilepsy & psychiatry notes.

### 6.6 Course and the one management principle

**Recover the memory, do not sedate the patient.** Onset is typically sudden and under stress, and the localised form often recovers abruptly and completely, so the prognosis is generally good. The management principle is a safe, contained environment, removal from or resolution of the precipitating stressor, and a phased, psychologically informed recovery of the memory; a drug-assisted (abreaction) interview has historically been used but is not first-line, and grounding and the place of hypnosis are developed in the Psychotherapies, clinical assessment, MSE & nosology notes. What the disorder does *not* need is a benzodiazepine: there is no anxiolytic indication for dissociative amnesia, and reflexive sedation both masks the picture and courts dependence.

**WORKED EXAMPLE**

A man in his thirties is brought in by railway police having been found several hundred kilometres from home, unable to give his name, address or occupation, and calm rather than distressed. On examination the sensorium is clear, there are no focal neurological signs, and he can learn and recall a new list of three objects without difficulty. A relative later reports that he left home the morning after being served with a large debt notice. The intact new learning, the clear sensorium, the loss of personal identity and the clear psychological precipitant point to a dissociative fugue with generalised dissociative amnesia, not an organic amnesia. The immediate job is still to exclude a seizure, transient global amnesia and a substance cause before the label is fixed, and the treatment is safety, contact with family and gentle memory recovery, not a sedative.

**INDIA**

Two genuine India devices meet here. First, culture: possession and trance states, in which a deity, spirit or ancestor is felt to take over, are a common idiom of distress in Indian settings and can present with amnesia and purposeful travel that mimic dissociative amnesia and fugue. ICD-11 keeps these as separate categories (trance disorder 6B62, possession trance disorder 6B63), and a sanctioned, non-distressing ritual state is not a disorder at all; the cultural formulation is the safeguard. Second, the treatment reflex: such presentations, and dissociative amnesia generally, are too often met with a reflexive benzodiazepine rather than an assessment. Alprazolam (Alprax, Restyl) is the default anxiolytic of Indian practice and the emblem of the country's benzodiazepine over-prescription problem; the standard rule is that a benzodiazepine should not ordinarily run beyond 2 to 4 weeks. Dissociative amnesia has no benzodiazepine indication. The transcultural reading of possession and trance is developed in the Consultation-liaison, geriatric & transcultural psychiatry notes.

| 30 | 6 to 20 | 2 to 4 | 0.5 to 6 |
|---|---|---|---|
| DES SCREEN CUT-OFF FOR A DISSOCIATIVE DISORDER (CARLSON AND PUTNAM 1993) | ALPRAZOLAM HALF-LIFE IN HOURS | BENZODIAZEPINE SHORT-COURSE CEILING IN WEEKS | ALPRAZOLAM ANXIOLYTIC DOSE (MG PER DAY) |

**PEARL**

A patient who cannot say who they are is far more likely to have a dissociative (or a malingered or factitious) amnesia than an organic one, because the organic amnestic syndromes leave personal identity intact. Loss of the name is a psychogenic flag until proven otherwise, and the feigned or factitious version is covered in the Somatic-symptom, sexual, impulse-control disorders & suicide notes.

**GOOD TO REMEMBER**

- **Dissociative amnesia and fugue (the syndrome):** DSM-5-TR Criterion A is an inability to recall important autobiographical information, usually traumatic or stressful, that is inconsistent with ordinary forgetting (localised is the commonest type); fugue is the specifier "with dissociative fugue", apparently purposeful travel or bewildered wandering with amnesia for identity; the deficit is retrograde and reversible, and the first step is to exclude an organic cause (Criterion C) and to check whether personal identity is lost, which points away from an organic amnesia.
- **Alprazolam (Alprax, Restyl):** a short-acting benzodiazepine and the default Indian anxiolytic, half-life about 12 to 15 hours with no clinically important active metabolite, usual anxiolytic range 1 to 4 mg per day; it has no role in dissociative amnesia and should not ordinarily run beyond 2 to 4 weeks, the short-course rule that guards against the Indian over-prescription pattern.

**HOLD THIS**

Dissociative amnesia is a retrograde, reversible loss of autobiographical memory (localised is commonest) on a trauma trigger, with personal identity often lost but new learning intact; the organic amnestic syndrome is the mirror image (anterograde, identity preserved), so exclude a neurological or substance cause before you commit, and never sedate it with a benzodiazepine.

For the wider dissociative-disorders classification and dissociative identity disorder, see the neighbouring topics; the benzodiazepine mechanism, the half-life classes and the taper are developed in the neighbouring benzodiazepine topic; for the dissociative subtype of PTSD and the trauma-related disorders, the Anxiety, OCD & trauma-related disorders notes; and for the somatic-symptom, functional and factitious boundary, the Somatic-symptom, sexual, impulse-control disorders & suicide notes.

## 7 · Possession and trance states

**One question decides this whole topic:** is this a disorder at all, or a sanctioned cultural act? **Possession** and **trance** presentations are a genuinely **culture-bound** corner of the dissociative disorders, and the examinable skill is not the phenomenology but the boundary. A narrowing of awareness with stereotyped, seemingly uncontrolled behaviour, or the sense of being replaced by an external identity, is a *disorder* only when it is involuntary, unwanted, distressing or impairing, and lies *outside* an accepted collective cultural or religious practice. Read as an accepted ritual, the very same behaviour is not pathology at all.

The two systems diverge here, so carry both. **ICD-11 keeps two separate categories**, trance disorder (6B62) and possession trance disorder (6B63); **DSM-5-TR keeps neither as a standalone**, folding possession-form presentations into dissociative identity disorder and listing

dissociative trance only as an example under Other Specified Dissociative Disorder. In the Indian setting this is high-yield because possession presentations are common, and the clinician's first move is a cultural formulation, decoding the local **idioms of distress** before any label is applied.

### 7.1 The two ICD-11 entities: trance versus possession trance

**Narrowed awareness versus a replacing identity.** Both are trance states with a marked alteration of consciousness or a loss of the normal sense of personal identity; what separates them is whether an *external identity takes over* (ICD-11 CDDR).

#### Trance disorder (6B62)

A trance state with narrowing of awareness of the immediate surroundings (or unusually narrow, selective focus) *and* restriction of movements, postures and speech to a small repertoire experienced as outside one's control; crucially, it is *not* experienced as being replaced by an alternate identity.

#### Possession trance disorder (6B63)

A trance state in which the normal sense of personal identity is *replaced by an external "possessing" identity*, with behaviours or movements experienced as controlled by the possessing agent (a spirit, deity, demon or the dead); full or partial amnesia for the episode is usual.

#### Shared thresholds

Episodes are recurrent, or, if the diagnosis rests on a single episode, that episode has lasted at least several days; the state is involuntary and unwanted, is not a sanctioned collective practice, is not organic or substance-related, and causes distress or impairment.

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**RULE** **The sanctioned-practice line is the diagnosis.** An involuntary, unwanted, distressing or impairing trance or possession state that falls *outside* an accepted collective cultural or religious practice is a disorder; the identical experience embraced as ritual, without distress or impairment, is not.

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### 7.2 The critical boundary: accepted cultural practice is not a disorder

**Where possession is welcomed, it is not pathology.** ICD-11 states the exclusion twice over: the diagnosis is *not* applied to experiences accepted in the person's context as collective cultural phenomena or religious practice, and *not* applied where there is no significant distress or impairment. Single, transitory experiences lasting minutes to hours, and states cultivated within religious or cultural groups where possession is normative, are explicitly excluded, and people who gradually gain control and acceptance of the experience within such a group carry no excess of mental disorder over the general population. DSM-5-TR draws the same line in its own words: dissociative trance "is not a normal part of a broadly accepted collective cultural or religious practice."

### 7.3 ICD-11 versus DSM-5-TR versus the old ICD-10 label

**Same phenomenon, three homes.** The classification map is itself the examiner's favourite question here.

| SYSTEM | WHERE POSSESSION / TRANCE LIVES | KEY POINT |
|---|---|---|
| **ICD-11** | **Two categories: trance disorder (6B62), possession trance disorder (6B63)** | Possession trance = identity replaced by an *external* agent |
| **DSM-5-TR** | Possession folded into dissociative identity disorder; dissociative trance under Other Specified Dissociative Disorder | **No standalone possession/trance disorder** |
| **ICD-10** | F44.3 trance and possession disorder | A single combined rubric ("possession hysteria") |

The one entity ICD-11 splits in two, DSM-5-TR scatters across DID and OSDD, and ICD-10 merged under F44.3.

### 7.4 Reading the differential: identity, psychosis, PTSD and the organic mimics

**External agent, brief and stress-bound, normal EEG.** Four boundaries decide the case. Against dissociative identity disorder and partial DID, the possession identity is attributed to an *external* agent, whereas DID identities are internal and not so attributed; someone describing both internally and externally attributed identities is diagnosed with DID, not possession trance. Against schizophrenia, the intrusive voices, inserted thoughts and controlled behaviours occur *primarily during* the brief possession state, with no other positive or negative psychotic symptoms. Against PTSD, if trance-like states are confined to flashback re-experiencing, no additional trance diagnosis is made. Against the organic mimics, possession trance can resemble focal unaware (complex partial) seizures of temporal lobe epilepsy or delirium, but trance disorder shows a *normal EEG even during episodes* and lacks the global confusion of delirium.

TRAP **Reading a possession state without the cultural formulation, or missing an organic cause.** Two errors sink this presentation: labelling a sanctioned ritual possession as a disorder because the culture was never assessed, and stamping "possession trance" on what is actually temporal lobe epilepsy, delirium or a substance effect. Take the cultural formulation and clear the organic differential before the label.

### 7.5 The Indian frame: a common presentation, decoded through culture

**Common here, and best understood as an idiom of distress.** Possession trance presentations are very commonly seen in India and certain African countries, where the possessing agent characteristically matches a figure from the local religious tradition and the episode is often precipitated by domestic disharmony, interpersonal conflict or acculturative stress. The clinical task is not to sedate the phenomenon but to read it: apply the cultural formulation, decode the **idioms of distress** the family is using, and separate a sanctioned, welcomed ritual possession from an involuntary, distressing state that meets criteria. The wider transcultural framing and the neighbouring culture-bound syndrome concept sit in the Consultation-liaison, geriatric & transcultural psychiatry notes; the dissociative subtype of PTSD and the trauma-linked trance in the Anxiety, OCD & trauma-related disorders notes; and the video-EEG work-up that excludes non-epileptic (dissociative) convulsions in the Epilepsy & psychiatry notes.

| 20 to 25 | 6B62 · 6B63 | F44.3 |
|---|---|---|
| MEAN AGE AT ONSET (YRS), ICD-11 CDDR | ICD-11 TRANCE AND POSSESSION TRANCE CODES | ICD-10 TRANCE AND POSSESSION DISORDER RUBRIC |

**INDIA**

Possession is a mainstream idiom of distress across much of India, expressed through spirit intrusion, temple and dargah healing, mediumship and family-attended possession episodes, so the same behaviour can be devotional ritual in one setting and disorder in another. The examinable move is discipline: run the cultural formulation, ask whether the community accepts and welcomes the state, and reserve the diagnosis for the involuntary, unwanted, distressing or impairing form. The genuine local pitfall is the reflex to reach straight for a benzodiazepine, alprazolam (Alprax or Restyl) being the default anxiolytic in Indian practice, when the presentation calls first for cultural understanding and treatment of the underlying stress or comorbidity, not sedation of a culturally meaningful experience.

**PEARL**

The cleanest discriminator between the two ICD-11 entities is a single question: *who is acting?* If awareness narrows and behaviour shrinks to a small repertoire that feels outside control but the self is still the self, it is trance disorder; if the self is replaced by an external spirit, deity or demon that directs the behaviour, it is possession trance disorder. Replacement of identity by an external agent is the whole of the difference.

**GOOD TO REMEMBER**

- **Trance disorder (the syndrome):** the defining criterion is a trance state with a marked alteration of consciousness marked by *both* narrowing of awareness of the surroundings *and* restriction of movement, posture and speech to a small, seemingly uncontrolled repertoire, *without* replacement by an alternate identity; recurrent or a single episode of at least several days, involuntary and unwanted, outside sanctioned cultural practice, and distressing or impairing. The first step is always to check that it is not an accepted collective ritual and not organic (normal EEG even during the episode).
- **Possession trance disorder (the syndrome):** the defining criterion is a trance state in which the normal sense of identity is *replaced by an external "possessing" identity*, with behaviours experienced as controlled by that agent and usual full or partial amnesia; the discriminator from dissociative identity disorder is that the identity is attributed to an *external* agent, and the discriminator from all of them is that a sanctioned, non-distressing collective practice is not a disorder.

**HOLD THIS**

Trance disorder is narrowed awareness with a small uncontrolled behavioural repertoire and the self intact; possession trance disorder is the self replaced by an external agent; both are disorders ONLY when involuntary, unwanted, distressing or impairing and outside an accepted collective cultural or religious practice, which is why the cultural formulation, not sedation, is the first step in the Indian setting.

# Anxiolytic and hypnotic pharmacology

## 8 · Benzodiazepines

**This is the weightiest topic in the pharmacology block, and it rewards a single organising idea.** A **benzodiazepine** is a **positive allosteric modulator** at the **GABA-A receptor**: it does nothing on its own, but where GABA is already present it turns the inhibitory signal up. Hold that mechanism and three examinable strands fall out of it in order, the *receptor* story (how it works), the *kinetic* story (how long it works, which sorts the agents into short-acting and long-acting classes), and the *clinical* story (that it is a powerful short-term drug and a dangerous long-term one). The paper tests the last strand hardest: the single most answerable management statement on this material is that a benzodiazepine is **not** a first-line long-term anxiolytic or hypnotic.

The guideline spine is fixed. For anxiety and insomnia the British Association for Psychopharmacology (BAP) leads, the National Institute for Health and Care Excellence (NICE) sets the short-course limit, the Maudsley Prescribing Guidelines carry the receptor detail, the doses, the half-lives and the diazepam-equivalence table, and the Indian Psychiatric Society carries the Indian layer. The receptor mechanism and the GABA-A detail here are the same ones cross-referenced to the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes; the sedative and benzodiazepine *use* disorder as an addiction sits in the Substance use disorders notes; the lorazepam challenge in catatonia in the Other psychotic disorders, delusional syndromes & catatonia notes; and the SSRIs and SNRIs that outrank benzodiazepines as first-line anxiolytics in the Mood disorders: pharmacotherapy notes. The accompanying figures set out the receptor with its binding site, the half-life map, the withdrawal time-course, the class-and-mechanism map and the taper.

**Fig 15.11** The anxiolytic and hypnotic class-and-mechanism map: five mechanism classes and the agents that sit under each, so the whole band recalls from the receptor action outward.

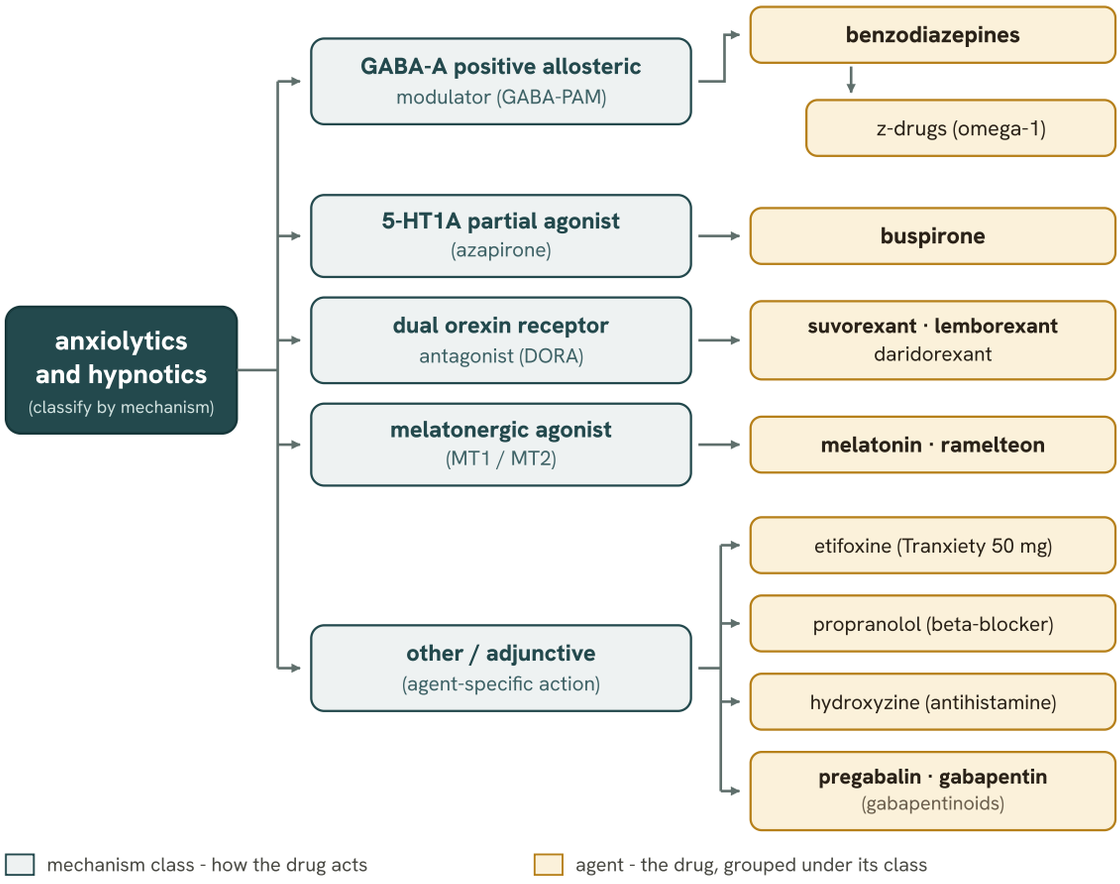



**Fig 15.12** The GABA-A receptor is a pentameric chloride channel; the benzodiazepine binds the allosteric site at the alpha/gamma interface and, only with GABA present, raises the frequency of channel opening.

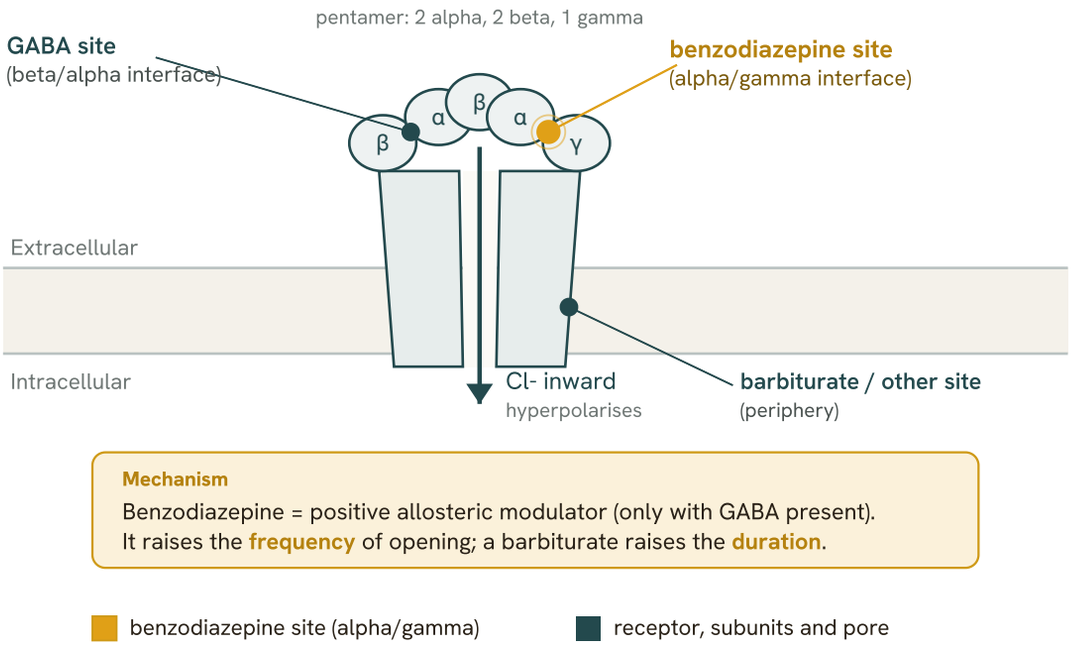


**Fig 15.13** The benzodiazepines sorted by elimination half-life into short-, intermediate- and long-acting classes, with the shared long-acting metabolite and the duration-to-use rule.

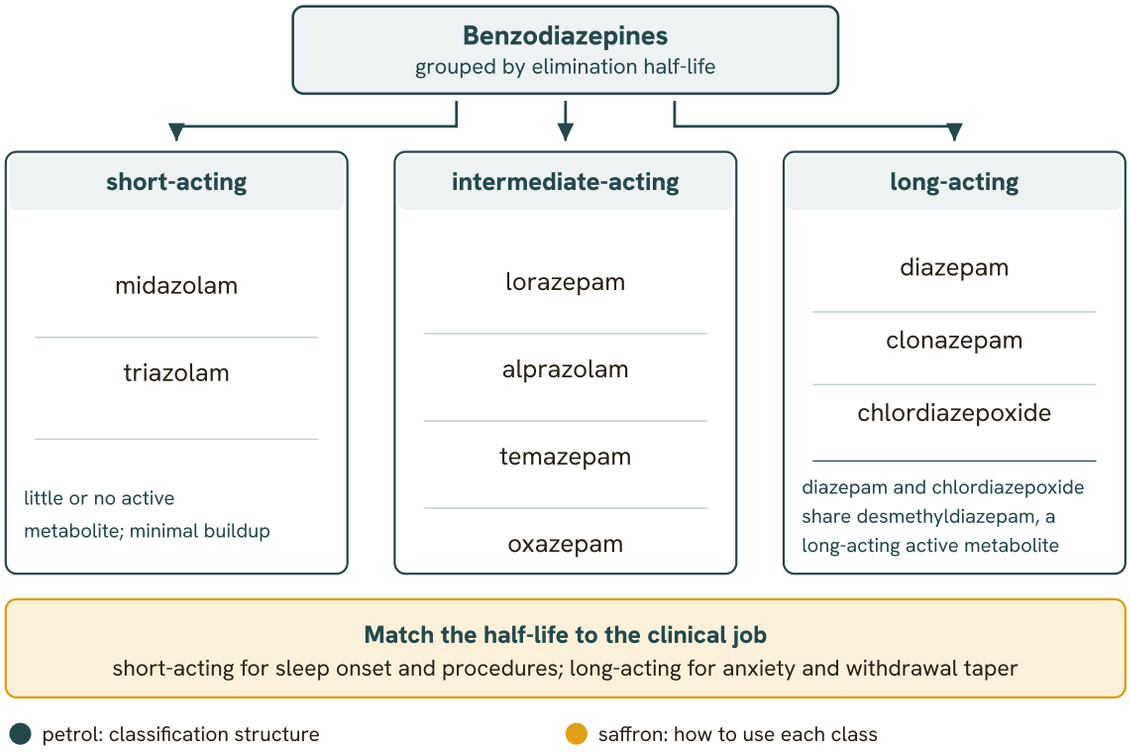
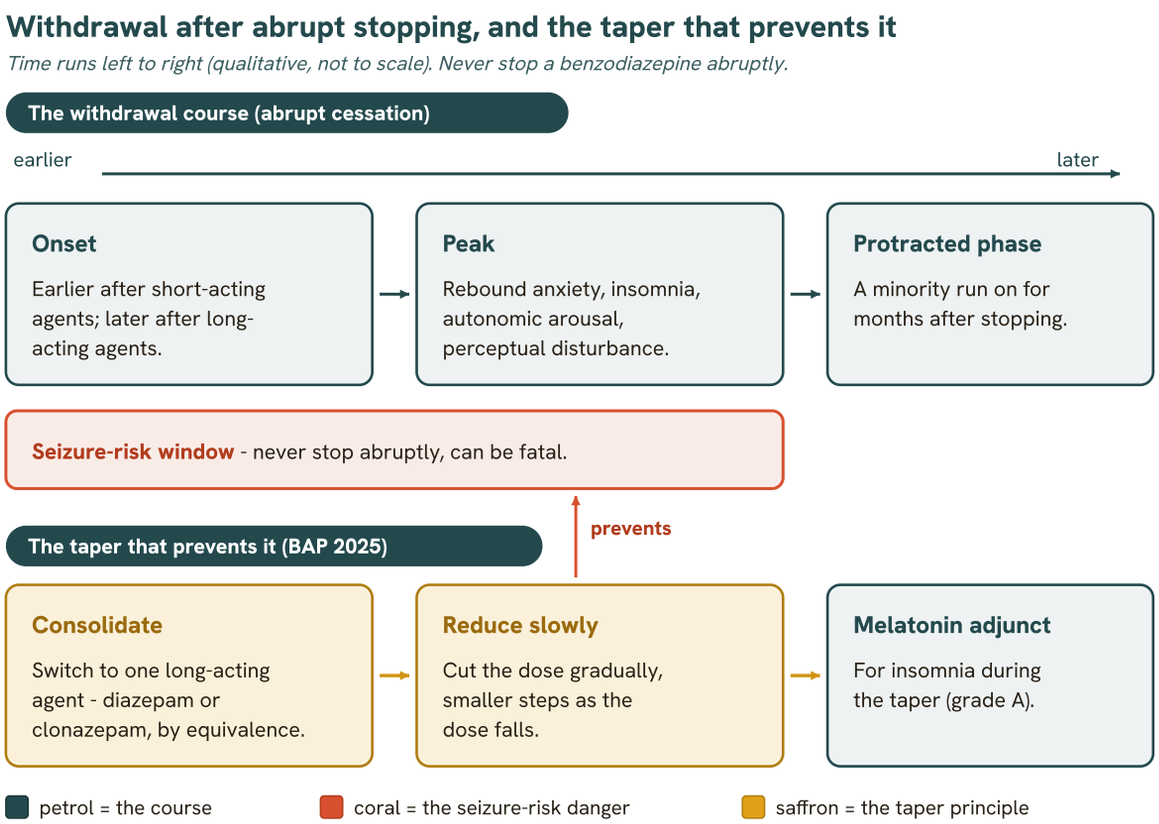



**Fig 15.14** The benzodiazepine withdrawal course after abrupt cessation, and the long-acting taper that prevents it.



### 8.1 Mechanism: a positive allosteric modulator that raises the frequency of channel opening

**Frequency, not duration, and only in the presence of GABA.** A **benzodiazepine** binds the **benzodiazepine binding site** on the GABA-A receptor and, by **allosteric modulation**, increases the affinity of the receptor for its own transmitter GABA (Stahl; KDT). The functional result is an increase in the *frequency* of **chloride channel** opening when GABA is bound, more chloride enters, the neurone hyperpolarises, and inhibition deepens (KDT). Two properties define the class from this one fact. First, the drug is a *modulator*, not a direct agonist: with no GABA present it opens no channel, which is the pharmacological reason benzodiazepines have a ceiling on respiratory depression and are relatively safe in isolated overdose. Second, the contrast with barbiturates is a favourite: barbiturates increase the *duration* (lifetime) of each chloride channel opening, and at high concentration open the channel directly without GABA, which removes the ceiling and makes them lethal in overdose (KDT). In Stahl's neuroscience-based nomenclature the class is written literally as a GABA positive allosteric modulator (GABA-PAM).

RULE **Frequency up, GABA required.** A benzodiazepine is a positive allosteric modulator that raises the *frequency* of chloride-channel opening only when GABA is present (contrast the barbiturate, which raises channel-opening *duration* and can open the channel alone), and that GABA-dependence is why it has a ceiling effect and is relatively safe in a pure overdose.

### 8.2 The GABA-A receptor and the benzodiazepine binding site

**GABA sits on the beta interface, the benzodiazepine between alpha and gamma.** The **GABA-A receptor** is a pentameric ligand-gated chloride channel, most commonly two alpha, two beta and one gamma subunit. GABA itself, the orthosteric ligand, binds at the interface involving the beta subunit and gates the channel; the **benzodiazepine binding site** is a separate, allosteric site at the interface between the *alpha and gamma* subunits (KDT; Stahl). Because occupancy of that site only modulates a channel GABA is already working on, agonists such as diazepam facilitate inhibition while inverse agonists at the same site (the beta-carbolines) hinder it and are anxiogenic and convulsant, a clean demonstration that this is genuine allosteric modulation in both directions. The subtype detail is worth a line for the paper: benzodiazepine action at alpha-1-containing receptors mediates sedation and the amnestic and anticonvulsant effects, while alpha-2 (and alpha-3) receptors carry the anxiolytic and muscle-relaxant effects (Stahl). This receptor-and-site anatomy is the shared substrate developed further in the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes, and the accompanying figure lays it out as the membrane-grammar plate.

GOOD TO REMEMBER

- **Benzodiazepine (the class):** a positive allosteric modulator at the GABA-A receptor, binding the benzodiazepine site at the *alpha-gamma* interface and increasing the *frequency* of chloride-channel opening in the presence of GABA; alpha-1 drives sedation, alpha-2 drives anxiolysis; the signature caution is dependence with long-term use and seizures on abrupt withdrawal.

### 8.3 The half-life classification and active metabolites

**The clinically useful sort is by half-life, because that predicts accumulation, hangover and withdrawal.** Agents split into **short-acting** or short-to-intermediate drugs, which clear quickly,

carry little or no active metabolite and so accumulate less, and **long-acting** drugs, which have long half-lives and long-lived active metabolites and therefore build up (a hazard in the elderly and in liver disease). The pivotal metabolite is desmethyldiazepam (nordiazepam), the long-lived active metabolite shared by diazepam and chlordiazepoxide, which is what makes those agents behave as long-acting even beyond their parent half-life (Stahl). The short-acting drugs with no clinically important active metabolite are the ones preferred when the liver is impaired, remembered as the LOT group. The half-lives below are the verified Stahl and Maudsley figures.

| AGENT | ELIMINATION HALF-LIFE (HOURS) | ACTIVE METABOLITE | CLASS |
|---|---|---|---|
| **lorazepam** | **10 to 20** | none | short-to-intermediate |
| **alprazolam** | **12 to 15** | none of note | short-to-intermediate, high-potency |
| **oxazepam / temazepam** | short (not tabulated here) | none | short-acting (the LOT group, liver-safe) |
| **chlordiazepoxide** | 24 to 48 | nordiazepam (long-lived) | long-acting |
| **clonazepam** | **30 to 40** | none of clinical note | long-acting, high-potency |
| **diazepam** | **20 to 50 (parent)** | nordiazepam, oxazepam, temazepam | long-acting |
| **zolpidem (z-drug, for contrast)** | about 2.5 | none | ultra-short |

Half-life classification with verified Stahl elimination half-lives; the long-acting agents owe their duration to the shared active metabolite nordiazepam (desmethyldiazepam). Oxazepam and temazepam half-lives were not individually verified here.

MNEMONIC

### LOT

**L**orazepam, **O**xazepam, **T**emazepam: the short-acting benzodiazepines with *no* active metabolite. They do not accumulate, so they are the preferred choice in hepatic impairment (lorazepam is explicitly named for this in Stahl) and the safer choice in the elderly.

### 8.4 Clinical uses and the short-course rule

**Powerful, fast, and reserved.** Benzodiazepines are genuinely useful for a small set of jobs, acute and severe anxiety or agitation, alcohol withdrawal, status epilepticus and acute seizures, catatonia (the lorazepam challenge, developed in the Other psychotic disorders, delusional syndromes & catatonia notes), pre-medication, and very short-term insomnia. What the examiner wants is the **short-course** discipline around chronic anxiety and insomnia. For generalised anxiety disorder an SSRI is the **first-line** pharmacological treatment (BAP 2014; NICE CG113 2011 names sertraline first), with an SNRI or pregabalin as the alternative; buspirone and hydroxyzine cover acute anxiety only. A benzodiazepine is not first-line and not for maintenance: BAP reserves it only after non-response to an SSRI, an SNRI, pregabalin *and* buspirone, for

persistent, severe, distressing, impairing anxiety, and NICE says do not offer a benzodiazepine for generalised anxiety disorder except as a short-term measure of no more than 2 to 4 weeks during a crisis (NICE 2011). For chronic insomnia CBT for insomnia is first-line (BAP 2019), with a benzodiazepine or a z-drug only as a short-term option, matching the half-life to the pattern. If a benzodiazepine is genuinely effective and continued, it is continued for a defined period and then tapered, never open-endedly.

TRAP **Starting a benzodiazepine for chronic anxiety or insomnia.** The reflex prescription for persistent worry or poor sleep is exactly the error the guidelines forbid: the first-line agents are the SSRI (anxiety) and CBT for insomnia, and the benzodiazepine is a 2 to 4 week crisis measure at most (NICE 2011), because tolerance, dependence and a withdrawal seizure risk follow long-term use.

### 8.5 Agent selection and the benzodiazepine-sparing principle

**Match the agent to the goal, and spare the benzodiazepine wherever a non-dependence option exists.** The practical skill of **agent selection** is deciding **which agent** for which target, and the organising rule is **benzodiazepine-sparing**: reach past the benzodiazepine to a drug that does the job without dependence whenever the clinical goal allows it. The class-and-mechanism map below (also drawn as the accompanying figure) sets out the Band B options with their Indian brands. For sustained anxiety, prefer the SSRI or SNRI, pregabalin or buspirone; for the somatic and performance-anxiety picture, propranolol; for short-term insomnia, a z-drug, melatonin or very-low-dose doxepin; and keep the benzodiazepine for the acute crisis, the seizure, the alcohol withdrawal and catatonia.

| CLASS | EXAMPLE AGENT (INDIAN BRAND) | MECHANISM | PLACE IN THERAPY |
|---|---|---|---|
| **Benzodiazepine** | diazepam (Valium, Calmpose) | GABA-A positive allosteric modulator, raises frequency of chloride-channel opening | **short-course / acute only** |
| **Z-drug** | zolpidem (Zolfresh, Nitrest) | GABA-A benzodiazepine-1 (omega-1) subtype selective | short-term hypnotic |
| **Azapirone** | buspirone (Buspin) | 5-HT1A partial agonist | non-dependence anxiolytic, delayed onset |
| **Gabapentinoid (alpha-2-delta ligand)** | pregabalin (Pregeb, Pregastar) | voltage-gated calcium channel alpha-2-delta binding | GAD alternative first-line |
| **Antihistamine** | hydroxyzine (Atarax) | H1 antagonist | acute anxiolytic |
| **Beta-blocker** | propranolol (Ciplar, Inderal) | peripheral beta-adrenergic blockade | somatic / performance anxiety |
| **Melatonergic** | melatonin (Meloset, Zytonin) | MT1 / MT2 receptor agonist | circadian / insomnia over 55 years |
| **Non-benzodiazepine GABA modulator** | etifoxine (Tranxiety 50 mg) | GABA-A modulation (non-benzodiazepine site) | anxiolytic (brand unverified) |
| **Orexin antagonist (DORA)** | suvorexant | dual orexin / hypocretin receptor block | insomnia |

The anxiolytic and hypnotic class-and-mechanism map; the SSRIs and SNRIs that outrank all of these as first-line anxiolytics are developed in the Mood disorders: pharmacotherapy notes.

### 8.6 Dependence, tolerance and the withdrawal syndrome

**Three syndromes, and one of them can kill.** Long-term use produces **tolerance** (a diminishing effect, so the dose is pushed up) and physical **dependence**. Kaplan separates three things that follow stopping. *Relapse* is the slow return of the original disorder over weeks to months. *Rebound* is a brief, more intense re-emergence of anxiety or insomnia, worse than baseline, lasting up to about 3 weeks; with a short-acting agent it appears as *interdose* rebound anxiety 3 to 4 hours after each dose (classically alprazolam, mistaken for craving). *Withdrawal* is the true discontinuation syndrome with new autonomic features. Its onset is kinetic: it begins within hours to a few days of stopping a **short-acting** benzodiazepine, but may not start for 1 to 2 weeks after abrupt cessation of a **long-acting** one (Kaplan). Minor symptoms are sweating, tachycardia, nausea, tremor, restlessness, and the characteristic *perceptual disturbance* (heightened sensitivity to light and sound, depersonalisation). The dangerous, major symptoms are *seizures*, psychosis and delirium, and seizures are the reason a benzodiazepine must never be stopped abruptly (Maudsley: abrupt cessation can cause seizures and can be fatal). Withdrawal is more severe with high-potency, short-half-life agents, typically lasts 2 to 4 weeks, and can occasionally become

protracted over many months. The accompanying figure lays this out as a withdrawal time-course timeline.

**3 to 4 h**
INTERVAL AT WHICH INTERDOSE REBOUND ANXIETY FOLLOWS A SHORT-ACTING AGENT

**1 to 2 wk**
DELAY BEFORE WITHDRAWAL STARTS AFTER A LONG-ACTING AGENT IS STOPPED ABRUPTLY

**2 to 4 wk**
USUAL DURATION OF THE WITHDRAWAL SYNDROME

**GOOD TO REMEMBER**

- **Benzodiazepine withdrawal syndrome (the syndrome):** the defining features are rebound anxiety and insomnia, perceptual disturbance and autonomic arousal, with the criterion-level dangers being *seizures*, psychosis and delirium; the discriminator from relapse is the timing (hours to days after a short-acting drug, up to 1 to 2 weeks after a long-acting one) and the new autonomic signs. First step: never stop abruptly, taper.

### 8.7 The taper: switch to a long-acting agent, then reduce slowly

**Consolidate, convert, then come down in proportions.** The evidence-based **taper** (BAP 2025; Maudsley) has a fixed shape. First *consolidate* the patient onto a single long-acting benzodiazepine, **diazepam** or **clonazepam**, chosen for their long half-life and dosing flexibility (use lorazepam or oxazepam instead where the liver is impaired). Converting to diazepam needs the diazepam-equivalence table, verified from the Maudsley Prescribing Guidelines below. Then *reduce gradually*: patients often tolerate reductions of about 10 percent of the last dose every 2 to 4 weeks, roughly 5 to 10 percent per month, with the steps becoming smaller as the dose falls (the dose-effect relationship is hyperbolic) and the final doses very small (often less than 1 mg of diazepam-equivalent). Melatonin is a useful adjunct for insomnia during the taper (BAP grade A). Two anchors for the paper: doses above 30 mg diazepam-equivalent daily may cause harm and are avoided (Maudsley), and the difficult final doses of alprazolam are often eased by substituting clonazepam (Kaplan).

| BENZODIAZEPINE | DOSE EQUAL TO DIAZEPAM 10 MG (MG) |
|---|---|
| **Diazepam** | 10 |
| **Chlordiazepoxide** | 25 |
| **Clonazepam** | **0.5** |
| **Lorazepam** | **1** |
| **Lormetazepam** | 1 to 2 |
| **Nitrazepam** | 10 |
| **Oxazepam** | 20 |
| **Temazepam** | 20 |

Approximate diazepam-equivalent doses, verified from the Maudsley Prescribing Guidelines (Table 3.29). Alprazolam is deliberately not tabulated there, so no alprazolam equivalence is asserted here (see gaps).

GOOD TO REMEMBER

- **Diazepam (Valium, Calmpose):** the long-acting anchor of the taper; oral 4 to 40 mg per day in divided doses; GABA-A positive allosteric modulator with the active metabolite nordiazepam; verified equivalence, diazepam 10 mg equals clonazepam 0.5 mg equals lorazepam 1 mg equals chlordiazepoxide 25 mg (Maudsley).
- **Clonazepam (Clonotril, Rivotril):** long-acting (half-life 30 to 40 hours), high-potency; panic 0.5 to 2 mg per day, seizures up to 20 mg per day; the other consolidation agent for the taper and the substitute that eases the final doses of alprazolam.
- **Alprazolam (Alprax, Restyl):** short-to-intermediate (half-life 12 to 15 hours), high-potency; anxiety 1 to 4 mg per day; the most toxic benzodiazepine in overdose (Maudsley) and the hardest to taper, manufacturer limit 2 to 4 weeks.
- **Lorazepam (Ativan, Loraz):** short-to-intermediate (half-life 10 to 20 hours), *no* active metabolite so liver-safe; oral 2 to 6 mg per day; the catatonia challenge agent at 1 to 2 mg.

### 8.8 Flumazenil: the antagonist and its seizure caution

**The antidote that can precipitate the seizure it looks like it should treat.** Flumazenil is the benzodiazepine-receptor *antagonist*, a competitive blocker at the benzodiazepine binding site used to reverse benzodiazepine sedation and respiratory depression (Stahl; Maudsley). Two facts matter clinically. Its half-life is short, only 41 to 79 minutes, much shorter than most benzodiazepines it reverses, so re-sedation follows a single dose and repeat dosing is often needed. And it carries a real *seizure* hazard: it can precipitate seizures in a patient who is tolerant to or dependent on benzodiazepines, in a mixed overdose (particularly with cyclic antidepressants), or where the benzodiazepine was suppressing seizures, so it is used cautiously and at the minimally effective dose, not reflexively in every benzodiazepine overdose. Because uncomplicated benzodiazepine overdose is usually benign with supportive care, the risk of flumazenil often outweighs the benefit.

GOOD TO REMEMBER

- **Flumazenil (the antagonist):** a competitive benzodiazepine-receptor antagonist with a short half-life of 41 to 79 minutes (so re-sedation and repeat dosing); the signature caution is that it can *precipitate seizures* in benzodiazepine dependence and in mixed or cyclic-antidepressant overdose, so it is used selectively at the lowest effective dose, not routinely.

### 8.9 Buspirone and the z-drugs: the non-benzodiazepine comparators

**Two agents the paper contrasts against the benzodiazepine. Buspirone** is an azapirone anxiolytic, a *5-HT1A partial agonist* (not a GABA drug at all), with a short half-life of 2 to 3 hours and a usual dose of 20 to 30 mg per day (up to 60 mg per day). Its defining contrast with the benzodiazepine is a *delayed* onset of 2 weeks or more and *no* dependence, sedation, anticonvulsant effect or interaction with alcohol, which is exactly why it is a benzodiazepine-sparing choice for sustained anxiety but useless for an acute crisis. The *z-drugs* (zolpidem, zopiclone, eszopiclone) act at the benzodiazepine-1 (omega-1) subtype of the GABA-A receptor, so they are hypnotic with less anxiolytic and muscle-relaxant effect; zolpidem is dosed 10 mg at bedtime for 7 to 10 days (short elimination half-life about 2.5 hours), and their signature warning is complex sleep behaviours (sleep-driving, sleep-eating with amnesia), plus dependence and

next-day impairment, so tolerance and rebound insomnia are real despite the earlier marketing that they were not.

**GOOD TO REMEMBER**

- **Buspirone (Buspin):** a 5-HT1A partial agonist, 20 to 30 mg per day; the mechanism-and-caution signature is a *delayed* onset (2 weeks or more) with *no* dependence, sedation or anticonvulsant effect, so it spares the benzodiazepine in sustained anxiety but is no use acutely.
- **Zolpidem (Zolfresh, Nitrest):** a z-drug at the benzodiazepine-1 (omega-1) GABA-A subtype, 10 mg at bedtime for 7 to 10 days; the signature caution is complex sleep behaviours (sleep-driving) with dependence and next-day impairment.

**PEARL**

Every hard fact on this topic is a corollary of one sentence: a benzodiazepine only turns up an inhibition that GABA is already producing. That is why it has a ceiling and is safe in a pure overdose (mechanism), why the receptor adapts and rebounds when it is removed (dependence and withdrawal), why the antidote flumazenil unmasks that rebound as a seizure, and why the long-term answer is a slow taper rather than an abrupt stop.

**INDIA**

India carries a genuine benzodiazepine over-prescription problem, and **alprazolam** (Alprax, Restyl) is the default anxiolytic reached for in general and psychiatric practice alike; benzodiazepines are among the most frequently prescribed drugs. Two consequences follow. First, alprazolam is the *most toxic* benzodiazepine in overdose (Maudsley) and, being short-acting and high-potency, the hardest to taper, so it is a poor default. Second, the Indian Psychiatric Society position and the wider benzodiazepine-sparing principle favour the SSRI or SNRI for sustained anxiety and CBT for insomnia, reserving the benzodiazepine for the genuine short course. The culture-bound relevance is real too: possession, trance and dissociative presentations are common in Indian settings and are frequently met with a reflex sedative, which treats nothing; read them through a cultural formulation (the Consultation-liaison, geriatric & transcultural psychiatry notes) rather than sedating them. The sedative and benzodiazepine use disorder as an addiction is developed in the Substance use disorders notes.

**HOLD THIS**

A benzodiazepine is a GABA-A positive allosteric modulator that raises the *frequency* of chloride-channel opening (barbiturates raise the *duration*); it is never a first-line long-term anxiolytic or hypnotic (NICE: a 2 to 4 week crisis course only); it is withdrawn by consolidating onto a long-acting agent (diazepam or clonazepam) and tapering slowly because *abrupt cessation can cause seizures*; and its antagonist flumazenil can itself precipitate seizures in dependence and mixed overdose.

For the receptor systems behind the GABA-A detail see the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes; for the sedative use disorder as an addiction the Substance use disorders notes; for the lorazepam management of catatonia the Other psychotic disorders, delusional syndromes & catatonia notes; for the SSRIs

and SNRIs as first-line anxiolytics the Mood disorders: pharmacotherapy notes; and for clinical insomnia and CBT for insomnia the Eating & sleep-wake disorders notes.

## 9 · Buspirone and the azapirones

**This is the anxiolytic the examiner uses to test whether you understand what a benzodiazepine is not.** Buspirone is a non-benzodiazepine anxiolytic of the **azapirone** class (chemically an azaspiro-decanedione), and almost every mark on it comes from a single contrast: it is a **5-HT1A partial agonist** that works through slow serotonergic adaptation rather than fast GABAergic inhibition, so it carries a **delayed onset** of two weeks or more and, crucially, **no dependence**, no sedation, no anticonvulsant effect and no abuse potential (Stahl, Prescriber's Guide).

Hold two opposing lists in your head. The benzodiazepine works within minutes, sedates, is a good PRN rescue, and breeds tolerance, dependence and a withdrawal syndrome. Buspirone does the reverse on every count: useless as a PRN because the effect takes a fortnight to appear, but clean on dependence and sedation across long-term use. That is why the guidelines place it as an acute-GAD option sitting *after* an SSRI and *before* a benzodiazepine in the benzodiazepine-sparing ladder, and why the classic trap is to expect it to abort acute anxiety.

### 9.1 Mechanism: a 5-HT1A partial agonist

**Serotonergic, not GABAergic.** Buspirone binds the 5-HT1A receptor as a partial agonist. At the presynaptic somatodendritic autoreceptor its agonism damps 5-HT neuronal firing acutely, while its postsynaptic partial agonism modulates onward serotonergic tone; the therapeutic anxiolysis emerges only once these receptors down-regulate and adapt over weeks, which is precisely why the onset is delayed (Stahl). It *does not act on the benzodiazepine receptor or the GABA-A chloride channel at all*, so it confers none of the GABAergic effects, sedation, muscle relaxation, anticonvulsant cover or cross-tolerance with alcohol and barbiturates. It additionally shows weak dopamine autoreceptor antagonism. For the receptor biology of the 5-HT1A and GABA-A systems, see the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes.

RULE **5-HT1A, weeks, no dependence.** Buspirone is a 5-HT1A partial agonist whose anxiolysis depends on slow receptor adaptation, so it has a delayed onset of two weeks or more and produces no dependence, sedation, anticonvulsant effect or abuse; it is an acute-GAD agent, never a PRN rescue.

### 9.2 The azapirone family

**Buspirone is the only one in routine anxiolytic use.** The **azapirones** are a small family of selective 5-HT1A partial agonists. Buspirone is the marketed anxiolytic; gepirone (extended-release) has since been approved as an *antidepressant* for major depression rather than as an anxiolytic; and ipsapirone, tandospirone and flesinoxan remained investigational 5-HT1A agonists that did not establish a first-line anxiety niche (New Oxford Textbook of Psychiatry;

Companion to Psychiatric Studies). The examinable point is the shared mechanism, not the minor agents.

| AZAPIRONE | 5-HT1A ACTION | ESTABLISHED ROLE |
|---|---|---|
| **Buspirone** | **Selective 5-HT1A partial agonist** | **Anxiolytic for GAD (acute)** |
| **Gepirone (ER)** | 5-HT1A partial agonist | Antidepressant for major depression |
| **Ipsapirone** | 5-HT1A partial agonist | Investigational / research agent |
| **Tandospirone** | 5-HT1A partial agonist | Anxiolytic in some Asian markets |
| **Flesinoxan** | 5-HT1A agonist | Investigational |

The azapirone class shares one mechanism; only buspirone is a mainstream anxiolytic, and gepirone crossed over to depression.

### 9.3 The place in GAD: the guideline ladder

**An acute-GAD option, not first-line, ahead of a benzodiazepine.** BAP is explicit that buspirone (and hydroxyzine) are efficacious in the *acute* treatment of generalised anxiety disorder but that neither has published evidence for relapse prevention (BAP 2014, Baldwin et al.). The first-line pharmacological treatment for GAD is an SSRI, with an SNRI or pregabalin as alternatives; a benzodiazepine is reserved and, in the BAP sequence, considered only after non-response to an SSRI, an SNRI, pregabalin *and* buspirone. NICE CG113 (NICE 2011) builds its stepped care on the SSRI/SNRI/pregabalin sequence and restricts benzodiazepines to short-term crisis use; the Maudsley Prescribing Guidelines carry buspirone as a second-line/adjunctive anxiolytic option. The first-line SSRI position is developed in the Mood disorders: pharmacotherapy notes, and the wider anxiety-disorder pharmacology in the Anxiety, OCD & trauma-related disorders notes.

TRAP **Calling buspirone first-line, or using it as a PRN.** Buspirone is *not* first-line for GAD (an SSRI is) and is worthless taken as-needed for an acute surge; a delayed-onset drug given for immediate relief simply fails and gets abandoned.

### 9.4 Delayed onset: useless as a PRN anxiolytic

**Two weeks to work is the whole clinical personality.** Buspirone takes roughly **2 weeks** before its anxiolytic effect is evident, and if it has not worked within **6 to 8 weeks** it may not work at all (Stahl). Because relief is not tied to the plasma level of any single dose, it cannot be used to abort a panic surge or as an as-needed rescue, the exact niche where a benzodiazepine excels. This delayed onset also drives the higher discontinuation seen versus a benzodiazepine, and it means a patient must never be switched abruptly from a benzodiazepine to buspirone: buspirone does not suppress benzodiazepine withdrawal, so the patient takes a therapeutic buspirone dose for two to four weeks first, and only then is the benzodiazepine tapered (Kaplan and Sadock, CTP 2024).

| FEATURE | BUSPIRONE (5-HT1A PARTIAL AGONIST) | A BENZODIAZEPINE (GABA-A PAM) |
|---|---|---|
| **Onset of anxiolysis** | **Delayed, 2 weeks or more** | **Minutes to hours** |
| **PRN / as-needed rescue** | **No, ineffective acutely** | Yes, effective |
| **Sedation, psychomotor impairment** | **None** | Yes |
| **Anticonvulsant / muscle relaxant** | **None** | Yes |
| **Dependence, tolerance, withdrawal** | **None; taper generally not needed** | Yes; seizure risk on abrupt stop |
| **Respiratory depression** | None | Yes (dose-related) |
| **Abuse potential** | **Low / none** | Present |

The buspirone-versus-benzodiazepine contrast is the single most examinable content on this drug: fast-but-dependent against slow-but-clean.

### 9.5 The safety signature: no dependence, no sedation, no anticonvulsant, no abuse

**Four clean "no"s.** Buspirone does not appear to cause dependence and shows virtually no withdrawal symptoms; it is not habit-forming and a taper is generally not necessary on stopping (Stahl). It causes no sedation, psychomotor impairment, cognitive impairment or respiratory depression (Kaplan and Sadock, CTP 2024), and it is anxioselective with no anticonvulsant or muscle-relaxant properties. This profile makes it attractive where a benzodiazepine is hazardous, in respiratory disease, sleep apnoea, a history of substance misuse, or the older adult, though its efficacy may be diminished in patients previously maintained on a benzodiazepine. Its own side effects are limited and non-sedating: dizziness, headache, nausea and nervousness. The benzodiazepine dependence and withdrawal picture that buspirone deliberately avoids is covered in the Substance use disorders notes.

MNEMONIC

**5-HT1A · TWO WEEKS · NO DEPENDENCE**

The three anchors that answer any buspirone question: the mechanism (a selective 5-HT1A partial agonist azapirone), the delayed onset (two weeks or more, so never PRN), and the clean tail (no dependence, no sedation, no anticonvulsant, no abuse).

### 9.6 Dosing, half-life and pharmacokinetics

**Short half-life, divided dosing, delayed clinical effect.** Buspirone is given at **15 to 30 mg/day** in divided doses, typically a thrice-daily schedule because the elimination half-life is short at only about **2 to 3 hours** (Stahl). A common titration starts around 15 mg daily in divided doses and increases in 5 mg steps every two to three days; the usual effective range is 20 to 30 mg/day, and the maximum is generally 60 mg/day (Stahl; Kaplan and Sadock, CTP 2024). It is metabolised by CYP3A4 to an active metabolite, 1-(2-pyrimidinyl)piperazine (1-PP), so a 3A4 inhibitor (for example a CYP3A4-inhibiting antifungal or nefazodone) raises buspirone levels and a 3A4

inducer such as carbamazepine lowers them. The apparent paradox worth stating in a viva is that a very short-half-life drug still has a delayed clinical onset, because the effect depends on receptor adaptation, not on drug accumulation.

| 2 to 3 | 2 weeks+ | 20 to 30 | 60 |
|---|---|---|---|
| ELIMINATION HALF-LIFE (HOURS) | ONSET BEFORE ANXIOLYTIC EFFECT APPEARS | USUAL EFFECTIVE DOSE (MG PER DAY) | MAXIMUM (MG PER DAY) |

**WORKED EXAMPLE**

A woman in her fifties with chronic generalised anxiety and mild COPD asks for "something for the nerves" and has been buying alprazolam from a chemist for months. She wants immediate relief. Buspirone is a reasonable maintenance anxiolytic here precisely because it will not sedate or depress respiration and carries no dependence, but she must be told it will take about two weeks to help and is useless as a tablet-when-anxious. The correct plan is to start an SSRI as the first-line agent for her GAD, use buspirone as an add-on or alternative rather than a benzodiazepine, and, because she is already on alprazolam, keep the alprazolam running at a therapeutic buspirone dose for two to four weeks before tapering it slowly; buspirone will not cover benzodiazepine withdrawal.

**INDIA**

Buspirone (marketed in India as Buspin, verify locally) matters most in India as the answer to a real problem: benzodiazepine over-prescription, with alprazolam (alprazolam; Alprax or Restyl) as the default anxiolytic reached for reflexively and often continued long-term. The Indian Psychiatric Society position, in line with BAP and NICE, is that an SSRI or SNRI is first-line for GAD and that a benzodiazepine is a short-course measure only, not a maintenance anxiolytic. Buspirone is a genuinely useful non-dependence-forming alternative in the patient who would otherwise be parked on alprazolam, and the exam-safe line is that its delayed onset and lack of a "kick" are exactly why patients (and prescribers) drift back to the benzodiazepine, which is the behaviour the guidelines are trying to correct.

**PEARL**

A drug can have a very short half-life and still a long onset. Buspirone clears in two to three hours yet takes two weeks to work, because anxiolysis rides on 5-HT1A receptor adaptation, not on plasma concentration. State that dissociation and the mechanism mark is yours.

**GOOD TO REMEMBER**

- **Buspirone (the drug):** a selective 5-HT1A partial agonist of the azapirone class, dosed at 15 to 30 mg/day in divided doses (maximum 60 mg/day) with a short elimination half-life of about 2 to 3 hours but a delayed onset of two weeks or more, so it is never a PRN anxiolytic; it is efficacious in acute GAD (no relapse-prevention evidence) and its signature is no dependence, no sedation, no anticonvulsant effect and no abuse potential.

**HOLD THIS**
Buspirone is a 5-HT1A partial agonist azapirone with a short half-life (about 2 to 3 hours) but a delayed onset of two weeks or more, so it is useless as a PRN rescue; it has no dependence, sedation, anticonvulsant effect or abuse, and it sits in acute GAD after an SSRI and before a benzodiazepine.

## 10 · The z-drugs

**The z-drugs are the examiner's test of whether you can separate a chemical class from a mechanism.** The **z-drugs** (zolpidem, zopiclone, zaleplon and eszopiclone) are **non-benzodiazepine hypnotics**: they belong to different chemical families from the benzodiazepines, yet they act at the very same GABA-A benzodiazepine site, and specifically at its **benzodiazepine-1 (omega-1)** subtype. That single fact answers most questions on them, because it explains both why they work as hypnotics and why "non-benzodiazepine" does not mean non-addictive.

Almost every mark comes from four discriminators: the receptor (an omega-1-selective GABA-A positive allosteric modulator), the half-lives (short, and different from each other, which is how you choose one), the guideline place (a short-term hypnotic after CBT-I, never a long-term one), and the signature caution (**complex sleep behaviour**). Hold those four and the topic is yours.

### 10.1 The class: four non-benzodiazepine hypnotics

**One shared site, three chemical families.** The z-drugs are grouped by their shared action, not their structure. Zolpidem is an imidazopyridine, zaleplon a pyrazolopyrimidine, and zopiclone and its active S-enantiomer eszopiclone are cyclopyrrolones (Kaplan and Sadock, CTP 2024). None is a benzodiazepine, so the "z-drug" or "non-benzodiazepine hypnotic" label is a statement about chemistry; the pharmacology is benzodiazepine-like because the binding site is shared. Zopiclone is available across most of the world including India; eszopiclone (Lunesta) is chiefly a US agent, and zaleplon has been withdrawn in several markets. For the GABA-A receptor biology and the wider receptor systems, see the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes.

### 10.2 Mechanism: the benzodiazepine-1 (omega-1) site

**A GABA-A positive allosteric modulator, but selective for the alpha-1 subtype.** Like a benzodiazepine, a z-drug is a **positive allosteric modulator** at the GABA-A receptor's benzodiazepine site, enhancing GABA-driven chloride-channel opening; it has no effect without GABA present (Stahl). The difference is selectivity. Whereas benzodiazepines bind fairly non-selectively across GABA-A receptors carrying the alpha-1, -2, -3 and -5 subunits, the z-drugs bind more selectively to receptors that contain the alpha-1 subunit, the population classically called the benzodiazepine-1 (BZ1, omega-1) receptor (Kaplan and Sadock, CTP 2024; Tasman). Because the alpha-1 subtype mediates sedation and hypnosis (and the amnestic effect), this selectivity gives a relatively cleaner hypnotic with comparatively less of the anxiolytic, myorelaxant and anticonvulsant action that the alpha-2, -3 and -5 subtypes carry. The examinable line is that the

z-drugs share the benzodiazepines' GABA-positive-allosteric-modulator mechanism but are a different chemical class acting preferentially at omega-1.

RULE **Same site, different molecule, same short-course rule.** A z-drug is a non-benzodiazepine hypnotic that still modulates the GABA-A benzodiazepine site, selectively at the omega-1 (alpha-1) subtype; it is a short-term hypnotic after CBT-I, not a long-term one.

### 10.3 Half-lives, and matching the drug to the insomnia pattern

**The half-life is how you choose between them.** All four are short-acting, but they differ enough to matter clinically. Elimination half-lives run from the ultrashort zaleplon at about **1 hour** (1 to 1.5 hours), through zolpidem at roughly **2.4 hours** and zopiclone at about **5.2 hours**, to eszopiclone as the longest at approximately **6 hours** (Tasman; Saunders, Addiction Medicine). The practical rule (which BAP states directly) is to match the half-life to the problem: a very short-acting agent (zaleplon, zolpidem) for sleep-onset insomnia, and a slightly longer-acting agent (zopiclone) for sleep-maintenance insomnia where the patient wakes in the second half of the night (BAP 2019). The trade-off is symmetrical: the shortest half-lives give the least next-morning hangover but the greatest rebound on stopping.

| Z-DRUG | CHEMICAL CLASS | APPROX HALF-LIFE (HOURS) | BEST INSOMNIA MATCH |
|---|---|---|---|
| **Zaleplon** | Pyrazolopyrimidine | ~1 (1 to 1.5) | Sleep-onset; middle-of-night dosing |
| **Zolpidem** | Imidazopyridine | ~2.4 | Sleep-onset |
| **Zopiclone** | Cyclopyrrolone | ~5.2 | Sleep-maintenance |
| **Eszopiclone** | Cyclopyrrolone (S-zopiclone) | ~6 | Sleep-maintenance (US agent) |

Ascending half-life is the memorable spine: zaleplon < zolpidem < zopiclone < eszopiclone, and it drives the onset-versus-maintenance choice.

| ~1 | 2.4 | 5.2 | 5 to 10 |
|---|---|---|---|
| ZALEPLON HALF-LIFE (HOURS) | ZOLPIDEM HALF-LIFE (HOURS) | ZOPICLONE HALF-LIFE (HOURS) | ZOLPIDEM DOSE (MG PER DAY) |

MNEMONIC

**zAleplon · zOlpidem · zOpiclone · Eszopiclone**

Read left to right for ascending half-life (roughly 1, 2.4, 5.2 and 6 hours). The two at the short end (zaleplon, zolpidem) are your sleep-onset drugs; the two at the long end (zopiclone, eszopiclone) hold sleep through the night.

### 10.4 The guideline place: a short-term hypnotic after CBT-I

**BAP leads: CBT-I first, then a short course of a GABA-positive hypnotic.** BAP is explicit that CBT-I (with sleep restriction and stimulus control) is first-line for chronic insomnia, and that a pharmacological agent is offered where CBT-I fails, is unavailable, or the patient cannot engage with it (BAP 2019, Wilson et al.). When a drug is used, a GABA-positive-allosteric-modulator

hypnotic, meaning a benzodiazepine or a z-drug such as zolpidem or zopiclone, is the short-term option, with the half-life matched to the insomnia pattern (BAP 2019). NICE restricts the z-drugs (zaleplon, zolpidem and zopiclone) to the short-term management of insomnia at the lowest effective dose, and advises against switching from one z-drug to another if the first has not helped (NICE TA77 2004, reviewed 2010). The Maudsley Prescribing Guidelines carry the same short-course, lowest-dose framing, and the Indian Psychiatric Society (IPS_CPG 2017) mirrors the short-term hypnotic position for insomnia in the Indian setting. The wider treatment of clinical insomnia and CBT-I itself is developed in the Eating & sleep-wake disorders notes.

TRAP **Treating a z-drug as dependence-free because it is "non-benzodiazepine".** The z-drugs cause tolerance, dependence, rebound insomnia and complex sleep behaviours; prescribing one long-term for chronic insomnia contradicts BAP and NICE, which both restrict them to short-term use after CBT-I.

### 10.5 Doses and the diazepam equivalence

**Low milligram doses at night; roughly a diazepam-5-mg equivalent each.** Usual adult doses are zolpidem **5 to 10 mg** at night (immediate-release; prolonged-release 6.25 to 12.5 mg), zaleplon **5 to 10 mg**, zopiclone **3.75 to 7.5 mg**, and eszopiclone **1 to 3 mg** (Tasman; Geriatric Psychiatry Study Guide). In the older adult the dose is halved (zolpidem 5 mg, prolonged-release 6.25 mg; zopiclone 3.75 mg) because of falls, next-morning impairment and delirium risk. For the withdrawal-and-taper arithmetic that a benzodiazepine consolidation uses, the verified equivalences are that zolpidem 10 mg, zopiclone 7.5 mg and zaleplon 10 mg are each approximately equivalent to diazepam 5 mg (Saunders, Addiction Medicine, Table 3.4). The benzodiazepine-equivalence switch and the slow taper themselves belong to the Substance use disorders notes.

| Z-DRUG | USUAL DOSE (MG PER DAY) | OLDER-ADULT DOSE (MG PER DAY) | APPROX DIAZEPAM-5-MG EQUIVALENT |
|---|---|---|---|
| **Zolpidem** | **5 to 10 (PR 6.25 to 12.5)** | 5 (PR 6.25) | **10 mg** |
| **Zaleplon** | 5 to 10 | 5 | **10 mg** |
| **Zopiclone** | **3.75 to 7.5** | 3.75 | **7.5 mg** |
| **Eszopiclone** | 1 to 3 | 1 | Not established here |

Doses are single-figure milligrams at night; the diazepam equivalence is the verified figure to quote when consolidating before a taper.

### 10.6 The z-drug-specific cautions: complex sleep behaviour, dependence, next-day impairment

**Three cautions define the class.** First, **complex sleep behaviour**: patients can carry out complex acts (sleep-walking, sleep-driving, sleep-eating, and even sleep-cooking or telephoning) while not fully awake and with no memory of the event afterwards. This prompted an FDA labelling change requiring warnings of complex sleep-related behaviours, and zolpidem in particular is associated with sleepwalking, sleep-driving and hallucinations (Dulcan; Maudsley Deprescribing Guidelines). Second, dependence: the z-drugs carry genuine abuse liability,

tolerance and a withdrawal syndrome, so "non-benzodiazepine" is not a safety guarantee (Lowinson and Ruiz). Third, next-day impairment: residual sedation, psychomotor and driving impairment, and, in the older adult, falls, fracture and delirium, which is why the 2019 Beers Criteria advise avoiding this class in older people (Kaplan and Sadock, CTP 2024). The sedative and z-drug use disorder as an addiction is covered in the Substance use disorders notes.

**COMMON TRAP**

A patient reports night-eating, an out-of-place car journey, or a partner's account of a full conversation, all with dense amnesia, and is worked up for a dissociative or nocturnal-seizure disorder. If they are on zolpidem or another z-drug, the first move is to stop the drug: this is a complex sleep behaviour until proven otherwise, and it is a reason to discontinue, not merely to reduce.

### 10.7 Choosing, dosing short, and stopping

**Lowest dose, shortest course, taper alongside CBT-I.** The management pattern that satisfies the guidelines is: reserve the z-drug for when CBT-I is not the route, pick the agent by half-life (onset versus maintenance), start at the lowest effective dose, and cap the intended duration to a short course rather than open-ended repeats. On stopping, taper slowly rather than ceasing abruptly, and deliver CBT-I concurrently during the taper to improve the outcome; melatonin is a useful adjunct for the insomnia that surfaces during withdrawal (BAP 2019). Abrupt cessation after regular use risks rebound insomnia and rebound anxiety, and where a z-drug has been co-prescribed with a benzodiazepine the seizure risk of the benzodiazepine dominates the taper plan.

**WORKED EXAMPLE**

A man in his forties with three weeks of sleep-onset insomnia after a job loss asks for "a sleeping tablet". CBT-I is offered first. Because he cannot access it quickly and the problem is falling asleep rather than staying asleep, a short course of zolpidem 10 mg at night (the short half-life suiting sleep-onset) is reasonable, explicitly time-limited and at the lowest effective dose. He is warned about next-morning driving and about complex sleep behaviours, told not to combine it with alcohol, and booked for CBT-I so the drug can be tapered rather than repeated indefinitely. Had he been waking at 4 a.m., zopiclone (longer half-life) would have been the better match.

**INDIA**

The z-drugs sit against a real Indian backdrop of benzodiazepine over-prescription, with alprazolam (Alprax or Restyl) as the default anxiolytic and hypnotic reached for reflexively and often continued long-term. Zolpidem (Indian brands Zolfresh or Nitrest) and zopiclone (Zopicon) are widely prescribed and easily obtained, and the same short-course discipline that the Indian Psychiatric Society applies to benzodiazepines applies to them: a hypnotic is a short-term measure after CBT-I, not a standing prescription. The exam-safe framing is that the z-drugs are marketed as the "safer, non-benzodiazepine" alternative, but they carry dependence, rebound and complex sleep behaviours of their own, so they do not solve the over-prescription problem, they simply relabel it.

**PEARL**

Selectivity, not chemistry, is the answer to "how do z-drugs differ from benzodiazepines". Both are GABA-A benzodiazepine-site positive allosteric modulators; the z-drugs are chemically distinct and bind preferentially at the omega-1 (alpha-1) subtype, which is why they are marketed as selective hypnotics yet still share the dependence and withdrawal liabilities.

**GOOD TO REMEMBER**

- **Zolpidem (imidazopyridine):** an omega-1-selective GABA-A positive allosteric modulator dosed 5 to 10 mg at night (prolonged-release 6.25 to 12.5 mg; 5 mg in the older adult), half-life about 2.4 hours, best for sleep-onset insomnia; the z-drug most linked to sleep-driving and sleepwalking; 10 mg approximately equals diazepam 5 mg. Indian brands Zolfresh, Nitrest.
- **Zopiclone (cyclopyrrolone):** the same omega-1-selective mechanism, dosed 3.75 to 7.5 mg at night (3.75 mg in the older adult), half-life about 5.2 hours, better for sleep-maintenance; 7.5 mg approximately equals diazepam 5 mg. Indian brand Zopicon.
- **Zaleplon (pyrazolopyrimidine):** the ultrashort z-drug, half-life about 1 hour, dosed 5 to 10 mg, useful for sleep-onset or middle-of-night dosing with the least next-morning hangover; 10 mg approximately equals diazepam 5 mg.
- **Eszopiclone (S-enantiomer of zopiclone):** the longest-acting z-drug, half-life about 6 hours, dosed 1 to 3 mg, chiefly a US agent (Lunesta) for sleep-maintenance insomnia.

**HOLD THIS**

The z-drugs (zolpidem, zopiclone, zaleplon, eszopiclone) are non-benzodiazepine hypnotics that bind selectively at the benzodiazepine-1 (omega-1, alpha-1) subtype of the GABA-A receptor; match half-life to the insomnia pattern (short-acting zolpidem or zaleplon for onset, longer zopiclone for maintenance), use them short-term after CBT-I, and watch for complex sleep behaviour, dependence and next-day impairment.

## 11 · Dual orexin receptor antagonists

**This is the competitor-delta hypnotic: the newest way to make someone sleep without touching the GABA-A channel at all.** The dual orexin receptor antagonist class (a **DORA**) contains suvorexant, lemborexant and daridorexant, and its entire examinable identity is one mechanistic pivot: instead of adding inhibitory GABAergic sedation the way a benzodiazepine or a Z-drug does, a DORA *subtracts the wake signal* by blocking the orexin (hypocretin) wake-promoting system, so the patient falls asleep because arousal is switched off rather than because the brain is chemically sedated.

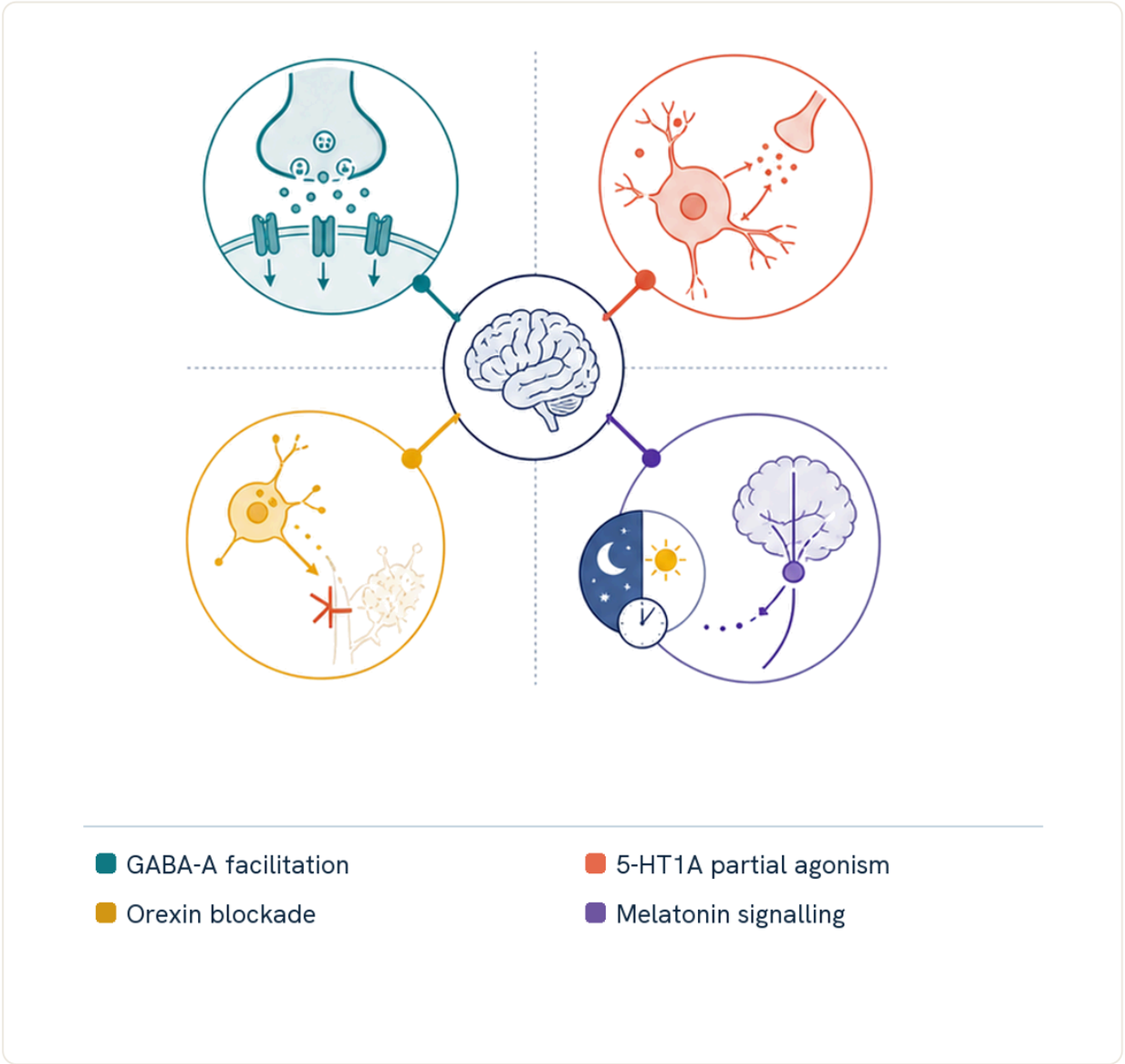



**Fig 15.15** Four pharmacological systems used across anxiety and insomnia: GABA-A facilitation, serotonin 5-HT1A partial agonism, blockade of orexin-mediated wake drive and melatonin-linked circadian signalling.

For a low-yield, current-through-2026 class the mark is won by holding three things: the target (orexin, acting at the OX1 and OX2 receptors), the class action (dual antagonism of both, promoting sleep by removing wakefulness), and the fact that suvorexant is the agent named in the BAP 2019 insomnia evidence base among the pharmacological options when CBT-I is not the route. Everything else is dose detail. Where these agents sit relative to CBT-I and the GABAergic hypnotics belongs to the Eating & sleep-wake disorders notes.

### 11.1 The target: the orexin/hypocretin wake system

**Orexin stabilises wakefulness; lose it and you get narcolepsy.** **Orexin** A and orexin B (also called hypocretin, the same peptide under two names) are excitatory neuropeptides cleaved from a single precursor, released by a small cluster of neurons in the lateral hypothalamus that act as the master "wake-stabilising" switch (Stahl; KDT). They project to and drive the arousal nuclei, the noradrenergic locus coeruleus, the histaminergic tuberomammillary nucleus and the wider reticular activating system, and they signal through two G-protein-coupled receptors, the OX1 receptor and the OX2 receptor, whose stimulation raises arousal and wakefulness. The clinching

physiology, and a favourite viva mirror, is that selective loss of orexin neurons is the lesion in narcolepsy with cataplexy (hypocretin deficiency); a DORA therefore reproduces a slice of that orexin-deficient state on purpose, at night, to let sleep come. The receptor biology of the arousal systems is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes.

### 11.2 The class action: block wakefulness, do not add sedation

**Dual OX1 and OX2 antagonism, not a GABA-A modulator.** A DORA is a competitive antagonist at *both* orexin receptors; by occupying OX1 and OX2 it removes the orexin drive onto the arousal network, so wakefulness is no longer actively maintained and the brain transitions into sleep (Stahl; KDT: "suvorexant blocks both OX1R and OX2R"). This is the whole point of the class: it initiates and maintains sleep without the positive allosteric modulation of the GABA-A chloride channel that a benzodiazepine or a Z-drug relies on, and it therefore lacks their signature of dependence, tolerance and a seizure-risk withdrawal to the same degree, while tending to preserve more of normal sleep architecture (Stahl notes the effect comes "without the side effects expected of a benzodiazepine or Z-drug hypnotic"). The GABAergic mechanism it deliberately avoids is the anchor of the Benzodiazepines topic and the wider Neurochemistry notes.

RULE **Subtract wakefulness, do not add sedation.** A DORA blocks OX1 and OX2 to switch off the orexin wake drive, so it promotes sleep without GABA-A modulation, and that non-GABAergic mechanism is the single fact every DORA question is testing.

PEARL

A DORA is pharmacologically induced, time-limited narcolepsy. Narcolepsy with cataplexy is the disease of orexin loss; a dual orexin receptor antagonist manufactures a brief orexin-blocked state at bedtime to produce sleep. State that mirror and the mechanism mark is yours, and it also explains the class cautions: next-morning somnolence and rare sleep paralysis, hypnagogic hallucinations or cataplexy-like phenomena.

### 11.3 The three agents and their doses

**Suvorexant, lemborexant, daridorexant, each once nightly.** All three are taken as a single dose shortly before bed. Suvorexant (international brand Belsomra) was the first, at **10 to 20 mg** nocte; lemborexant (Dayvigo) at **5 to 10 mg** nocte; and daridorexant (Quviviq), the most recently introduced agent, at **25 to 50 mg** nocte (Maudsley Prescribing Guidelines; KDT; Practical Psychopharmacology). The verified timing anchor for daridorexant is that it is taken within 30 minutes of going to bed with at least **7 hours** remaining before the planned waking time, precisely to keep the next-morning residual effect down; the same "leave a full sleep window" logic applies across the class.

| AGENT (INTERNATIONAL BRAND) | NIGHTLY DOSE (MG PER DAY) | KEY POINT |
|---|---|---|
| **Suvorexant (Belsomra)** | **10 to 20** | First DORA; CYP3A4-metabolised, mean half-life about 12 hours; a stopping taper is not required |
| **Lemborexant (Dayvigo)** | **5 to 10** | Dual OX1/OX2 antagonist; cap at 5 nocte and use caution above it in patients 65 years and older (fall risk) |
| **Daridorexant (Quviviq)** | **25 to 50** | Most recent agent; take within 30 minutes of bed with at least 7 hours of sleep opportunity remaining |

The three marketed dual orexin receptor antagonists and their once-nightly dose ranges; the numbers rise as the agents get newer.

| 12 | 10 to 20 | 25 to 50 | 7 |
|---|---|---|---|
| SUVOREXANT MEAN HALF-LIFE (HOURS) | SUVOREXANT DOSE (MG PER DAY) | DARIDOREXANT DOSE (MG PER DAY) | MINIMUM SLEEP WINDOW REMAINING (HOURS) |

TRAP **Dosing without a full night ahead.** Taking a DORA when fewer than about seven hours of sleep remain, or the morning before driving, invites next-morning somnolence and impaired driving; the drug switches wakefulness off, and that suppression has to be allowed to wear off before the patient needs to be alert.

### 11.4 Place in therapy, cautions and the India reality

**Not first-line, and still a controlled hypnotic.** CBT-I is first-line for chronic insomnia; a DORA is a pharmacological option only where CBT-I fails, is unavailable or is declined, and the accompanying figure sketches the orexin wake-switch and where the class acts. BAP 2019 (Wilson et al.) names suvorexant among the evidence-based pharmacological choices in that scenario, and the class initiates *and* maintains sleep. Despite the lower dependence signal relative to GABAergic hypnotics, these agents are still handled as controlled/scheduled drugs, and the examinable cautions are next-morning somnolence and driving impairment, the sleep-paralysis and hypnagogic-hallucination/cataplexy-like phenomena that follow from the mechanism, complex sleep behaviours as a class labelling concern shared with the Z-drugs, and CYP3A4 interactions for suvorexant. Their positioning against CBT-I sits in the Eating & sleep-wake disorders notes, and the benzodiazepine dependence they are designed to sidestep in the Substance use disorders notes.

**INDIA**

The genuine Indian angle for this class is an absence. Indian insomnia and anxiety prescribing still defaults reflexively to a benzodiazepine, with alprazolam (alprazolam; Alprax or Restyl) the habitual first reach and often continued long-term, which is exactly the over-prescription problem the Indian Psychiatric Society position and the BAP/NICE short-course rule are trying to correct. A non-GABAergic hypnotic that carries far less dependence liability is precisely what that pattern needs, yet the dual orexin receptor antagonists remain a boutique, largely unavailable and prohibitively priced option in Indian practice as of 2026, so they do not yet displace the alprazolam habit. The exam-safe line is to know the mechanism and name the class as the dependence-sparing future of hypnotic prescribing, while noting it is not yet the ground reality in India.

**GOOD TO REMEMBER**

- **Suvorexant (Belsomra):** the first dual orexin receptor antagonist, blocking OX1 and OX2 to remove the orexin wake drive; dosed 10 to 20 mg nocte, CYP3A4-metabolised with a mean half-life of about 12 hours, and no taper needed on stopping.
- **Lemborexant (Dayvigo):** a dual OX1/OX2 antagonist for insomnia at 5 to 10 mg nocte, with the specific caution to cap at 5 mg and use care above it in patients 65 years and older because of fall risk.
- **Daridorexant (Quviviq):** the most recent DORA at 25 to 50 mg nocte, taken within 30 minutes of bed with at least 7 hours before planned waking to limit next-morning residual effect.

**HOLD THIS**

The DORAs (suvorexant 10 to 20 mg, lemborexant 5 to 10 mg, daridorexant 25 to 50 mg nocte) block both OX1 and OX2 to switch off the orexin/hypocretin wake system, promoting sleep by removing wakefulness rather than by GABAergic sedation, which is why they largely sidestep benzodiazepine-style dependence.

## 12 · Melatonin and ramelteon

**These are the hypnotics the examiner uses to show you what a benzodiazepine is not.** Melatonin and ramelteon are **melatonergic** agents: agonists at the MT1 and MT2 receptors of the suprachiasmatic nucleus, the brain's circadian pacemaker. They coax sleep by nudging the body clock rather than by depressing the cortex, so unlike a benzodiazepine or a z-drug they carry no dependence, no next-day cognitive impairment, no complex sleep behaviour and no controlled-substance status (Kaplan and Sadock, CTP 2024; Stahl; Kaufman's Clinical Neurology). That clean tail, set against a genuinely modest hypnotic effect, is the whole examinable delta.

Two facts carry most of the marks. First, prolonged-release **melatonin** is the guideline option in the patient over 55 and in the circadian sleep disorders, and it is the endorsed adjunct for insomnia *during a benzodiazepine taper* (BAP). Second, **ramelteon** is the prescription MT1/MT2 **circadian hypnotic** for sleep-onset insomnia. Keep both apart from agomelatine, the melatonergic *antidepressant*, which is developed in the Mood disorders: pharmacotherapy notes (agomelatine cross-ref).

### 12.1 Mechanism: MT1/MT2 melatonergic agonism

**Melatonergic, not GABAergic.** Endogenous **melatonin** is synthesised from serotonin in the pineal gland and signals darkness to the suprachiasmatic nucleus. Its two G-protein-coupled receptors divide the labour: stimulation of the MT1 receptor suppresses the firing of the suprachiasmatic pacemaker and so *promotes sleep onset*, while stimulation of the MT2 receptor mediates the *phase-shifting*, circadian-entraining effect (Stahl, Prescriber's Guide). Ramelteon is a full agonist at both MT1 and MT2. Because the drug works through the physiological sleep-timing system and not by opening the GABA-A chloride channel, it produces none of the sedation-by-CNS-depression, muscle relaxation, anticonvulsant cover, respiratory depression or cross-tolerance that define a GABAergic hypnotic. For the GABA-A receptor and the wider receptor systems, see the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes.

#### MT1 receptor

Suppresses suprachiasmatic-nucleus firing; the sleep-*onset*-promoting arm of melatonergic signalling.

#### MT2 receptor

Mediates the *phase-shift* of the circadian clock; the entrainment arm used in jet lag and delayed sleep phase.

RULE **MT1 onset, MT2 phase, no dependence.** Melatonin and ramelteon are MT1/MT2 agonists acting on the circadian clock, not the GABA-A channel, so they cause no dependence, no cognitive impairment, no complex sleep behaviour and are not controlled substances.

### 12.2 Prolonged-release melatonin: the over-55 and circadian indications

**The over-55 hypnotic and the body-clock drug.** BAP names prolonged-release **melatonin** as an option to improve sleep-onset latency and sleep quality in patients over 55 years (BAP 2019, grade Ib), and the melatonergic agonists are the rational choice in the circadian rhythm sleep disorders, jet lag, shift work and delayed sleep phase, where the aim is to re-time rather than to sedate (Kaufman's Clinical Neurology; Kaplan and Sadock, CTP 2024). The licensed prolonged-release preparation is given as a **2 mg** bedtime dose; its effect on sleep latency is real but modest, comparable to other non-benzodiazepine hypnotics rather than to a benzodiazepine (Kaplan and Sadock, CTP 2024). Match a short-acting melatonergic agent to sleep-*onset* difficulty. Clinical insomnia, the sleep-wake disorders and CBT-I as the first-line are covered in the Eating & sleep-wake disorders notes.

GOOD TO REMEMBER

- **Melatonin (the drug):** an MT1/MT2 melatonergic agonist derived from serotonin in the pineal; the prolonged-release form is dosed at 2 mg at bedtime and is the BAP option for sleep-onset latency in patients over 55 and for the circadian sleep disorders; it does not enable benzodiazepine discontinuation on its own but is the endorsed adjunct for insomnia during the taper. Avoid in moderate-to-severe hepatic impairment.

### 12.3 Melatonin as an adjunct in benzodiazepine withdrawal

**It settles the insomnia; it does not do the taper.** BAP endorses **melatonin** as a useful adjunct for managing insomnia during benzodiazepine withdrawal (BAP 2025, grade A, strong). The important qualifier is that melatonin has *not* been shown to enable benzodiazepine discontinuation itself: a systematic review of six trials found it no better than placebo for that endpoint (Kaplan and Sadock, CTP 2024). So its role is symptomatic, to soften rebound insomnia while the actual work, consolidation onto a longer-acting benzodiazepine and a slow graded reduction, proceeds. The equivalence switch and the taper schedule, and the sedative or benzodiazepine use disorder in the addiction sense, are covered in the Substance use disorders notes.

TRAP **Leaning on melatonin to prevent withdrawal seizures.** Melatonin manages the insomnia of a benzodiazepine taper only; it does not cover the withdrawal syndrome or protect against the seizure. The slow graded reduction, never melatonin, is what prevents that.

### 12.4 Ramelteon: the MT1/MT2 circadian hypnotic

**An 8 mg, sleep-onset, non-scheduled hypnotic.** **Ramelteon** is a synthetic melatonin analogue and a full MT1/MT2 agonist, approved for insomnia characterised by difficulty with sleep *onset*. It is given as an **8 mg** dose about 30 minutes before bedtime (reports of efficacy span 4 to 64 mg), with no adjustment needed in renal impairment; use it cautiously in moderate hepatic impairment and avoid it in severe (Kaplan and Sadock, CTP 2024). The effect on sleep latency is small, roughly 4 minutes in a large meta-analysis, and is non-dose-dependent. Ramelteon is extensively metabolised by **CYP1A2**, so fluvoxamine, a potent CYP1A2 inhibitor, can raise ramelteon to toxic concentrations and is effectively contraindicated with it (Kaufman's Clinical Neurology). Its elimination half-life is short, under 2 hours, which is why it does not produce a next-day hangover. Being a melatonergic agonist, it is not a controlled substance.

| 2 | 8 | 55 | < 2 |
|---|---|---|---|
| PROLONGED-RELEASE MELATONIN (MG AT BEDTIME) | RAMELTEON (MG AT BEDTIME) | AGE FROM WHICH PR MELATONIN IS A BAP OPTION (YEARS) | MELATONERGIC AGONIST ELIMINATION HALF-LIFE (HOURS) |

**GOOD TO REMEMBER**

- **Ramelteon (the drug):** a full MT1/MT2 melatonergic agonist for sleep-onset insomnia, dosed at 8 mg about 30 minutes before bedtime, with a short elimination half-life under 2 hours and no dependence or controlled-substance status; it is metabolised by CYP1A2, so co-prescription with fluvoxamine is dangerous, and it is used cautiously in hepatic impairment.

### 12.5 The class signature, and the agomelatine cross-ref

**Clean where the GABAergic hypnotics are dirty.** Set against a benzodiazepine or a z-drug, the melatonergic agonists reduce sleep latency and improve subjective sleep quality but, crucially, do not impair cognition, cause imbalance, induce dangerous complex sleep behaviour or fall into the controlled-substance category (Kaufman's Clinical Neurology). That is exactly the profile that makes them attractive where a benzodiazepine or a z-drug is hazardous, in the older adult, in respiratory disease and in anyone with a substance-misuse history, though the trade-off is a

decidedly gentler hypnotic effect. The wider family also includes tasimelteon (licensed for Non-24-Hour Sleep-Wake Disorder in the blind) and agomelatine, the melatonergic *antidepressant*, which combines MT1/MT2 agonism with 5-HT2C antagonism, is dosed at 25 mg at bedtime and needs liver-function monitoring; agomelatine is developed as an antidepressant in the Mood disorders: pharmacotherapy notes (agomelatine cross-ref). The z-drug complex-sleep-behaviour and dependence cautions that the melatonergic agents avoid are covered with the benzodiazepine-receptor hypnotics.

| FEATURE | MELATONERGIC AGONIST (MELATONIN, RAMELTEON) | GABA-A HYPNOTIC (BENZODIAZEPINE, Z-DRUG) |
|---|---|---|
| **Target** | **MT1/MT2 on the suprachiasmatic clock** | **GABA-A chloride channel (PAM)** |
| **Mode of action** | Re-times sleep, promotes onset | Sedation by CNS depression |
| **Hypnotic effect size** | **Modest** | Larger, more reliable |
| **Dependence, tolerance, withdrawal** | **None** | Yes; seizure risk on abrupt stop |
| **Cognitive impairment, imbalance** | **None** | Yes |
| **Complex sleep behaviour** | **Not a feature** | Yes (notably the z-drugs) |
| **Controlled substance** | **No** | Yes |

The melatonergic-versus-GABAergic contrast is the single most examinable point on these agents: clock-timing and clean against channel-opening and dependent.

**INDIA**

**Melatonin** is available over the counter in India (Meloset or Zytonin; verify the brand and strength locally), which is precisely why it matters against the real Indian problem: the over-prescription of benzodiazepines, with alprazolam (alprazolam; Alprax or Restyl) reached for reflexively as the default anxiolytic and hypnotic and too often continued long-term. A non-dependence-forming, non-controlled melatonergic hypnotic is a genuinely useful alternative in the patient who would otherwise be parked on alprazolam for sleep, and it is consistent with the Indian Psychiatric Society and BAP position that CBT-I is first-line and a benzodiazepine is a short-course measure only. Ramelteon is not widely marketed in India, so melatonin is the melatonergic agent you will actually reach for.

**PEARL**

Split the receptor to answer the mechanism question: MT1 quietens the suprachiasmatic pacemaker and brings sleep *on*, MT2 *shifts* the clock. That single distinction explains why these drugs help sleep-onset insomnia and jet lag but sedate far less forcefully than a benzodiazepine.

**HOLD THIS**

Melatonin and ramelteon are MT1/MT2 melatonergic agonists acting on the suprachiasmatic clock, not the GABA-A channel: prolonged-release melatonin 2 mg for the over-55 and as the taper-insomnia adjunct, ramelteon 8 mg for sleep-onset insomnia, both with a short half-life, a modest effect, and no dependence or controlled-substance status.

## 13 · Etifoxine and the non-benzodiazepine anxiolytics

**This is the competitor-delta drug: the anxiolytic that quiets anxiety without being a benzodiazepine.** Etifoxine is **a non-benzodiazepine anxiolytic** licensed in parts of Europe, and its whole exam value is a single claim, that it can match a benzodiazepine for short-term anxiety while carrying less of the benzodiazepine tail (rebound, central-nervous-system side effects). Kaplan and Sadock (CTP 2024) confirm it as a non-benzodiazepine that **modulates the GABA-A receptor**, and it is the one **novel anxiolytic** the examiner is likely to name against lorazepam and alprazolam.

Hold the frame that makes the marks. A benzodiazepine works fast but breeds tolerance, dependence and rebound. A translocator protein ligand such as etifoxine works on the same final channel by a different route, boosting the brain's own calming neurosteroids, so the trials show comparable anxiolysis with a cleaner discontinuation. The trap is to read "non-benzodiazepine" as "first-line": an SSRI remains first-line for generalised anxiety, and these agents are alternatives or short-course options, developed in the Anxiety, OCD & trauma-related disorders notes and the Mood disorders: pharmacotherapy notes.

### 13.1 What etifoxine is: a novel non-benzodiazepine anxiolytic

**A European anxiolytic that is not a benzodiazepine.** Etifoxine (a benzoxazine derivative) is marketed as an anxiolytic for the psychosomatic manifestations of anxiety and, in the trial literature, for the anxious subtype of adjustment disorder; Kaplan and Sadock (CTP 2024) describe it plainly as "a nonbenzodiazepine drug that modulates GABA-A receptors available in Europe" (New Oxford Textbook of Psychiatry likewise lists it as a non-BZD anxiolytic). It is not a controlled substance and it is not licensed in India through the mainstream pharmacopoeia, which is why the standard Indian textbooks (the Companion) do not carry a dose for it; treat it as the examinable European comparator drug rather than a routine Indian prescription.

### 13.2 Mechanism: GABA-A modulation plus the translocator protein

**Two routes to the same chloride channel.** Etifoxine acts by a dual mechanism. First, it is a direct positive modulator at the **GABA-A receptor** (Kaplan and Sadock, CTP 2024, confirm the GABA-A modulation). Second, and this is the part worth a mark, it is a ligand of the mitochondrial 18 kDa translocator protein (TSPO, the old peripheral benzodiazepine receptor), and by stimulating TSPO it drives **neurosteroidogenesis**, raising synthesis of the neurosteroid allopregnanolone, itself a powerful positive allosteric modulator of GABA-A. So etifoxine augments GABAergic inhibition both directly and indirectly through its own endogenous neurosteroid, a mechanism distinct from the benzodiazepine site. The GABA-A receptor and the

wider neurosteroid systems are developed in the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes.

RULE **GABA-A directly, and again through the translocator protein.** Etifoxine is a non-benzodiazepine anxiolytic with a dual action, direct GABA-A modulation plus TSPO-driven neurosteroid (allopregnanolone) synthesis; it matches a benzodiazepine for short-term anxiety but is an alternative to it, never a substitute for a first-line SSRI.

### 13.3 The competitor-delta evidence: non-inferior to a benzodiazepine, better tolerated

**Same anxiolysis, cleaner tail.** The examinable data are head-to-head. A controlled protocol randomised **191** patients with DSM-IV adjustment disorder to etifoxine or lorazepam over **4 weeks**; on the Hamilton Anxiety Rating Scale (the primary outcome) etifoxine was *not inferior* to lorazepam, was better tolerated, produced more responders, and left fewer patients with **rebound anxiety** (Kaplan and Sadock, CTP 2024). A parallel comparison against alprazolam gave similar results, and after the drug was stopped the HAM-A scores kept falling in the etifoxine group while they rose again in the alprazolam group, which also carried more central-nervous-system adverse events. New Oxford records the same signal, a single-study superiority of etifoxine over lorazepam. The reading is that the benzodiazepine buys speed at the price of rebound and CNS load, while the translocator-protein route holds the gain after withdrawal.

| AXIS | ETIFOXINE (GABA-A + TSPO) | A BENZODIAZEPINE (GABA-A PAM) |
|---|---|---|
| **Site of action** | **GABA-A modulation plus TSPO neurosteroid synthesis** | Benzodiazepine site on GABA-A |
| **Short-term anxiolysis (HAM-A)** | **Non-inferior to lorazepam / alprazolam** | Effective |
| **Tolerability, CNS adverse events** | **Better tolerated, fewer CNS events** | Sedation, psychomotor impairment |
| **After discontinuation** | **Gains maintained; less rebound anxiety** | Rebound anxiety, scores rise |
| **Controlled-substance / dependence liability** | Not scheduled; low reported abuse | Scheduled; dependence and withdrawal |

The competitor-delta in one grid: etifoxine matches a benzodiazepine on anxiety scores but is better tolerated and holds the gain after stopping.

TRAP **Reading "novel" and "non-benzodiazepine" as "first-line".** A non-inferiority signal against lorazepam does not make etifoxine a first-line anxiolytic; an SSRI is first-line for generalised anxiety, and etifoxine (like hydroxyzine) is an alternative or short-course option, not the front of the ladder.

### 13.4 Dose and the Indian brand

**The 50 mg capsule, given a few times a day.** Etifoxine is supplied as a **50 mg** capsule and given in divided doses, with a usual daily dose of about **150 to 200 mg/day** (the standard European product information; the parsed textbook corpus confirms the drug and its mechanism but does not carry an exact dose, so treat the milligrams as standard-source rather than corpus-verified). In

India, Wilfred flags the brand **Tranxiety** (etifoxine 50 mg); the Indian brand and strength could not be independently confirmed against the Companion or the Maudsley Prescribing Guidelines, so it is carried as Wilfred-provided and flagged for a local check. A rare but recognised safety signal with etifoxine is idiosyncratic hepatocellular injury and severe cutaneous reactions, which is worth naming as the signature caution and is flagged as not corpus-confirmed.

| 50 | 150 to 200 | 191 | 4 |
|---|---|---|---|
| ETIFOXINE CAPSULE STRENGTH (MG) | USUAL ETIFOXINE DOSE (MG PER DAY) | PATIENTS IN THE LORAZEPAM NON-INFERIORITY TRIAL | COMPARISON-TRIAL DURATION (WEEKS) |

**INDIA**

Etifoxine matters in India as an answer to a genuine problem rather than as a common prescription. The problem is benzodiazepine over-prescription, with alprazolam (Alprax or Restyl) reached for reflexively as the default anxiolytic and continued far past any crisis. The Indian Psychiatric Society, in line with BAP and NICE, holds that an SSRI or SNRI is first-line for generalised anxiety and that a benzodiazepine is a short-course measure only, not a maintenance drug. A non-benzodiazepine anxiolytic that holds its gain after withdrawal is exactly the kind of agent that answers the alprazolam-default habit, and Wilfred flags a local brand (Tranxiety, etifoxine 50 mg); the exam-safe line is that etifoxine is a European comparator drug with a favourable discontinuation profile, not an established Indian first-line, and the brand and dose need local verification. The benzodiazepine dependence and withdrawal picture it is contrasted against is covered in the Substance use disorders notes.

### 13.5 The other non-benzodiazepine anxiolytics: a compact class

**A small family, mostly historical or niche.** Beyond etifoxine and the 5-HT1A azapirone buspirone, the label "non-benzodiazepine anxiolytic" gathers a scatter of agents. Chlormezanone is the historical one to name: a centrally acting drug with both anti-anxiety and neuro-hypnotic (muscle-relaxant) actions used for tension states (KDT, Essentials of Medical Pharmacology), which was largely withdrawn from world markets in the 1990s after reports of severe cutaneous reactions and hepatotoxicity, so it survives as an exam name rather than a live prescription (the withdrawal reason is flagged as not corpus-confirmed). Hydroxyzine (Atarax) is the H1-antihistamine anxiolytic that BAP names, with buspirone, as efficacious in acute generalised anxiety. Two further historical roots complete the picture: the carbamate meprobamate, the mid-century "minor tranquilliser" the benzodiazepines displaced, and tofisopam, an atypical 2,3-benzodiazepine promoted as a non-sedating daytime anxiolytic. Buspirone itself is developed in its own topic.

| AGENT | CLASS / MECHANISM | STATUS |
|---|---|---|
| **Etifoxine** | **GABA-A modulator + TSPO neurosteroid ligand** | Licensed anxiolytic (Europe) |
| **Buspirone** | 5-HT1A partial agonist (azapirone) | Current; acute GAD (own topic) |
| **Hydroxyzine** | H1 antihistamine | Current; acute GAD option (BAP) |
| **Chlormezanone** | **Central anxiolytic / muscle relaxant** | Historical; largely withdrawn |
| **Meprobamate** | Carbamate | Historical; displaced by benzodiazepines |
| **Tofisopam** | Atypical 2,3-benzodiazepine (non-sedating) | Niche; limited markets |

The compact class: etifoxine is the live novel agent, chlormezanone and meprobamate are the historical names, hydroxyzine the current antihistamine option.

**PEARL**

The translocator protein has two lives on this paper. As a drug target it is how etifoxine raises its own GABA-modulating neurosteroid; as an imaging marker (TSPO PET tracers such as [11C]PK11195) it flags activated microglia in the neuroinflammation literature. Same protein, one pharmacological and one radiological. Say which you mean and the mark is yours.

**GOOD TO REMEMBER**

- **Etifoxine (the drug):** a non-benzodiazepine, novel anxiolytic acting by a dual mechanism, direct GABA-A modulation plus translocator-protein (TSPO)-driven neurosteroid (allopregnanolone) synthesis; supplied as a 50 mg capsule at about 150 to 200 mg/day, it was non-inferior to lorazepam and alprazolam on the HAM-A over 4 weeks with better tolerability and less rebound anxiety; the signature caution is rare hepatic and severe cutaneous reactions (dose and brand standard-source, not corpus-verified).
- **Chlormezanone (historical):** a centrally acting anxiolytic with muscle-relaxant / neuro-hypnotic properties for tension states, largely withdrawn from world markets for severe skin and liver reactions; an exam name, not a current prescription.
- **Hydroxyzine (the class option):** an H1-antihistamine anxiolytic that BAP names, alongside buspirone, as efficacious in acute generalised anxiety, with no dependence liability.

**HOLD THIS**

Etifoxine is a non-benzodiazepine anxiolytic that hits GABA-A two ways, directly and through translocator-protein-driven neurosteroid synthesis, and in adjustment-disorder anxiety it was non-inferior to lorazepam and alprazolam on the HAM-A while causing less rebound and fewer CNS effects, yet it is an alternative to a benzodiazepine, never a replacement for a first-line SSRI.

## 14 · Beta-blockers, antihistamines and gabapentinoids as anxiolytics

**These are the adjunctive and off-label anxiolytics, and they earn their marks by testing one clinical reflex: can you match a drug to a symptom?** Grouped here are three non-serotonergic,

non-benzodiazepine options reached for around the edges of anxiety management: a **beta-blocker anxiolytic** for the autonomic and performance-anxiety symptoms, a sedating **antihistamine anxiolytic** with genuine acute-generalised-anxiety evidence, and the two **gabapentinoid** agents, one of which is a licensed alternative first-line treatment for generalised anxiety disorder (GAD). None is a first-line agent for the psychic anxiety of GAD except pregabalin, and the single most current examinable point is that the gabapentinoids are no longer regarded as dependence-free.

Hold the group as a spread of receptor targets: propranolol at the peripheral **beta-adrenergic receptor**, hydroxyzine at the H1 histamine receptor, and pregabalin and gabapentin at the alpha-2-delta subunit of the voltage-gated calcium channel. Where each SSRI and SNRI sits as the true first-line anxiolytic belongs to the Mood disorders: pharmacotherapy notes, and the wider place of the anxiolytics across the anxiety disorders to the Anxiety, OCD & trauma-related disorders notes; this topic owns the adjuncts.

### 14.1 Propranolol: the beta-blocker anxiolytic

**Blocks the body, not the mind.** Propranolol (Ciplar or Inderal) is a non-selective **beta-adrenergic antagonist** whose anxiolytic action is peripheral: it abolishes the somatic, sympathetically driven symptoms of anxiety, the palpitations, tachycardia, tremor and dry mouth, without touching the cognitive dread itself (Kaplan and Sadock, CTP 2024; Practical Psychopharmacology). Its established anxiety niche is social anxiety disorder, performance type: a single dose of **10 to 40 mg** is taken about an hour before the feared performance, with a test dose tried on an ordinary occasion beforehand. Because relief comes from switching off the peripheral adrenergic surge rather than from sedation, it is a useful non-dependence-forming alternative to a benzodiazepine for the situational performer, the musician or the examinee with a pounding heart. It has a short half-life of about **3 to 5 hours** (immediate-release). Cautions are cardiorespiratory: avoid in asthma and COPD (beta-2 bronchoconstriction), in significant bradycardia or heart block, and do not withdraw abruptly in the cardiac patient. The beta-1-selective agent atenolol is an alternative. It has no useful effect on the pervasive worry of GAD.

> **PEARL**
>
> The beta-blocker treats the pulse, not the panic. Propranolol strips out the palpitations and tremor of the performance surge by blocking peripheral beta-adrenergic drive, but leaves the anticipatory dread intact, which is exactly why it shines for the time-limited somatic storm of stage fright and fails as a treatment for generalised, cognitively driven anxiety.

### 14.2 Hydroxyzine: the antihistamine anxiolytic

**A sedating H1 antagonist with real acute-GAD evidence.** Hydroxyzine (Atarax) is a first-generation piperazine antihistamine; its central antagonism of the **H1 histamine receptor** produces the sedation and anxiolysis that make it an **antihistamine anxiolytic** rather than merely an allergy drug. It carries a place most of this group lacks: BAP is explicit that hydroxyzine (alongside buspirone) is efficacious in the *acute* treatment of generalised anxiety disorder, though neither has published evidence for relapse prevention (BAP 2014, Baldwin et al.). The anxiety

dose is **50 to 100 mg** up to four times a day, and the elimination half-life is long at about **20 hours** (Stahl, Prescriber's Guide). It is not a controlled drug and does not cause dependence, so it is a reasonable short-term or as-needed alternative to a benzodiazepine. The examinable cautions are its sedation (warn about driving and machinery), its anticholinergic effects (dry mouth, constipation) and QT prolongation with cardiac conduction delay at higher doses.

RULE **Match the drug to the symptom.** Propranolol for the somatic autonomic surge of performance anxiety and hydroxyzine for short-term or acute generalised anxiety; of this whole group only pregabalin is a licensed alternative first-line agent for GAD, and an SSRI remains the true first-line drug.

### 14.3 The gabapentinoids: one mechanism, not a GABA drug

**Alpha-2-delta ligands despite the name.** The **gabapentinoid** class holds two agents, pregabalin and gabapentin, and the trap built into the name is that neither acts on the GABA-A receptor or on GABA metabolism at all (Kaplan and Sadock, CTP 2024; Stahl). Both bind the **alpha-2-delta subunit** of voltage-gated (P/Q-type) calcium channels on presynaptic terminals, reducing calcium influx and dampening the release of excitatory neurotransmitters such as glutamate, noradrenaline and substance P; the net effect is a reduction in neuronal excitability that translates into anxiolytic, analgesic and anticonvulsant activity. Both are excreted largely unchanged by the kidney rather than metabolised in the liver, so they carry few pharmacokinetic drug interactions but need dose reduction in renal impairment. The fast, frequency-increasing GABA-A mechanism they are so often confused with is the benzodiazepine's, developed in the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes.

### 14.4 Pregabalin: the licensed alternative first-line agent

**The one non-serotonergic drug licensed for GAD.** Pregabalin (Pregeb or Pregastar) is licensed for generalised anxiety disorder in the UK and Europe, and both BAP and NICE place it as an alternative initial treatment where an SSRI or SNRI is unsuitable or not tolerated, and as an augmentation option after non-response to an SSRI or SNRI (BAP 2014; NICE CG113). Its different side-effect profile and faster onset than an SSRI are exactly why it is useful in the patient who cannot tolerate serotonergic agents (Shorter Oxford Textbook of Psychiatry). The dose is **150 to 600 mg** per day in two to three divided doses, and the half-life is short at about **5 to 7 hours** (Stahl). The signature caution, and the reason it is a scheduled/controlled drug (Schedule V in the United States), is a genuine dose-related misuse and dependence potential with euphoria and a withdrawal syndrome; it must never be stopped abruptly (seizure risk), and it can cause sedation, dizziness, peripheral oedema and weight gain, with the anticonvulsant-class suicidality signal to bear in mind.

### 14.5 Gabapentin: the off-label cousin

**Off-label, weaker evidence, the same cautions.** Gabapentin (Gabapin or Gabantin) is used off-label for anxiety, with controlled-trial evidence in social phobia (social anxiety disorder) and a post-hoc signal in panic disorder, but it is not licensed for GAD and does not carry pregabalin's guideline status (Kaplan and Sadock, CTP 2024). When used for anxiety the dose is broadly **900**

**to 1800 mg** per day in three divided doses, titrated upward over several days, with the same short half-life of about **5 to 7 hours** and the same renal excretion. Crucially it shares pregabalin's dependence and misuse liability and its abrupt-withdrawal seizure risk, so it is not the safe, consequence-free benzodiazepine substitute it is sometimes assumed to be. The sedative and benzodiazepine dependence picture these agents are compared against is developed in the Substance use disorders notes.

TRAP **Treating a gabapentinoid as dependence-free.** Pregabalin and gabapentin are now recognised drugs of misuse with a withdrawal syndrome; never stop either abruptly (seizure risk) and screen actively for misuse, especially alongside opioid or benzodiazepine use, rather than reaching for them as a "safe" alternative.

| AGENT (INDIAN BRAND) | CLASS / TARGET | USUAL DOSE (MG PER DAY) | HALF-LIFE (HOURS) | PLACE IN ANXIETY |
|---|---|---|---|---|
| **Propranolol (Ciplar, Inderal)** | Non-selective beta-adrenergic antagonist | **10 to 40 (single pre-event dose)** | 3 to 5 (IR) | **Somatic symptoms of performance / social anxiety** |
| **Hydroxyzine (Atarax)** | H1 antihistamine antagonist | **50 to 100, up to four times a day** | about 20 | **Efficacious in acute GAD (no relapse-prevention data)** |
| **Pregabalin (Pregeb, Pregastar)** | Alpha-2-delta calcium-channel ligand | **150 to 600** | 5 to 7 | **Licensed alternative first-line for GAD; misuse potential** |
| **Gabapentin (Gabapin, Gabantin)** | Alpha-2-delta calcium-channel ligand | **900 to 1800** | 5 to 7 | Off-label (social phobia evidence); misuse potential |

The adjunctive and off-label anxiolytics at a glance; the accompanying figure maps each agent to its receptor target and its symptom niche.

| 10 to 40 | 150 to 600 | 900 to 1800 | 5 to 7 |
|---|---|---|---|
| PROPRANOLOL PRE-EVENT DOSE (MG PER DAY) | PREGABALIN DOSE (MG PER DAY) | GABAPENTIN DOSE (MG PER DAY) | GABAPENTINOID HALF-LIFE (HOURS) |

INDIA

These agents matter in India as the answer to a real problem: benzodiazepine over-prescription, with alprazolam (alprazolam; Alprax or Restyl) the reflexive default anxiolytic, often begun from the outset and continued long-term. The Indian Psychiatric Society position, in line with BAP and NICE, is that an SSRI or SNRI is first-line for GAD and that a benzodiazepine is a short-course measure only. Propranolol for the situational performer, hydroxyzine for short-term anxiety, and pregabalin as the licensed alternative are all genuine non-benzodiazepine options in the patient who would otherwise be parked on alprazolam. The important caveat is that the gabapentinoids are themselves a recognised and rising drug of misuse, so they are a considered swap and not a free pass.

**GOOD TO REMEMBER**

- **Propranolol (Ciplar / Inderal):** a non-selective beta-adrenergic antagonist that treats the peripheral autonomic symptoms (palpitations, tremor) of performance / social anxiety at a single 10 to 40 mg dose about an hour before the event; short half-life about 3 to 5 hours, no effect on psychic anxiety, avoid in asthma and bradycardia.
- **Hydroxyzine (Atarax):** a sedating H1 antihistamine anxiolytic efficacious in acute GAD (BAP), dosed 50 to 100 mg up to four times a day with a long half-life of about 20 hours; not a controlled drug and no dependence, but sedating and anticholinergic.
- **Pregabalin (Pregeb / Pregastar):** an alpha-2-delta calcium-channel gabapentinoid, licensed and guideline-endorsed as an alternative first-line treatment for GAD, at 150 to 600 mg per day in divided doses (half-life about 5 to 7 hours); signature caution is a genuine misuse and dependence potential with a seizure-risk withdrawal, so never stop abruptly.
- **Gabapentin (Gabapin / Gabantin):** an off-label alpha-2-delta anxiolytic (evidence in social phobia) at about 900 to 1800 mg per day in three divided doses (half-life about 5 to 7 hours), sharing pregabalin's misuse liability and abrupt-withdrawal seizure risk.

**HOLD THIS**

Of the adjuncts, propranolol (a beta-adrenergic antagonist, 10 to 40 mg before a performance) treats only the somatic surge, hydroxyzine (an H1 antihistamine, efficacious in acute GAD) is a non-dependence-forming short-term option, and the gabapentinoids act on the alpha-2-delta calcium-channel subunit (not GABA-A), with only pregabalin licensed as an alternative first-line agent for GAD and both now recognised as drugs of misuse.

## Apply and consolidate

### 15 · Worked cases

**This closing set of cases lands where the paper actually lives, at the bedside and the prescription pad.** Four **worked cases** gather the dissociative, conversion and anxiolytic threads into the four decisions the examiner most reliably tests: rule a **functional neurological disorder** in on a positive sign, separate a **dissociative** from an **organic** amnesia, taper a benzodiazepine dependence, and select the right anxiolytic or hypnotic for a specific patient. Each vignette is reasoned rather than recited, and each is a worked example of one two-beat method: name the positive finding that rules the decision in, then keep screening or apply the guideline anchor.

The textbook spine is the one carried through the block: Stone and Carson and DSM-5-TR for the conversion signs, DSM-5-TR and ICD-11 for the dissociative criteria, and BAP, NICE and the Maudsley Prescribing Guidelines for the taper and the agent choice, with the Indian Psychiatric Society for the Indian layer. Nothing new is introduced here; the cases only make the earlier topics decide something.

#### 15.1 How to work a case: rule in, then keep screening

**A positive finding first, an open mind second.** Every case on this material is worked in two beats. The first beat is a **rule-in**: elicit the sign or the pattern that makes the diagnosis positively, a Hoover's sign, an entraining tremor, a retrograde memory loss with identity gone but new learning intact, or an interdose rebound that betrays dependence. The second beat is the

safeguard: a positive sign never closes the organic work-up, and a diagnosis never licenses the wrong drug. This is the Stone and Carson logic for conversion generalised to the whole block, and it is why the reflex sedative, reached for so readily in Indian practice, is so often the wrong answer. Two of the cases are diagnostic (conversion, dissociative amnesia) and two are managerial (the taper, the agent choice), but the shape is identical.

### 15.2 Case one: functional leg weakness and a positive Hoover's sign

**Weak on command, strong on distraction.** case: a woman in her late twenties develops a dense weakness of the right leg over several hours after a bitter family confrontation, with no sensory level, normal reflexes and a flexor plantar response. Direct hip extension of the "weak" leg is flaccid. You cup a hand under the right heel and ask her to flex the *left* hip against your other hand; as she does, the right heel drives firmly down into your palm. That is a positive Hoover's sign, the extensors she could not fire on command firing automatically when attention moves to the other leg (Kaufman; Kaplan and Sadock, CTP 2024). It rules a functional neurological disorder in at the bedside, in seconds and without a scanner. A give-way weakness (ratchety power that builds then suddenly collapses) supports the picture but is not specific, because pain and poor effort in genuine disease can produce it too.

RULE **Rule in, then still screen.** A functional disorder is diagnosed on a positive sign of internal inconsistency (weak on command, strong on distraction), never on the absence of a lesion or the presence of a stressor; the positive sign does not close the organic search in a progressive or atypical case.

**WORKED EXAMPLE**

The Hoover manoeuvre doubles as the first move of treatment. Having shown the patient that her heel pressed down of its own accord, you can say that a leg which moves automatically is a leg that *can* move, replacing "there is nothing wrong" with "here is what is wrong, and here is why it can recover". The demonstrated sign proves the diagnosis and its reversibility in one gesture.

### 15.3 Case one continued: the functional tremor that entrains

**A tremor that copies the other hand is functional.** The same patient, at a later review, has a coarse tremor of the right hand. You ask her to copy a slow rhythmical tapping with the *left* hand; the right-hand tremor shifts its frequency to match the tapping, then stops, and she cannot keep the copied rhythm going. That is a positive tremor entrainment test, the movement-domain equivalent of Hoover's sign: an organic tremor holds its own frequency regardless, a functional one entrains, stops or disrupts the copied rhythm (DSM-5-TR; New Oxford 2024). The unifying mechanism across both signs is that the symptom depends on abnormally focused attention, so diverting attention exposes the intact pathway. The visual analogue the examiner likes is the tubular field, a visual-field defect the same width at 1 metre as at 2 metres, which is physically impossible and so rules the functional picture in.

**GOOD TO REMEMBER**

- **Functional neurological disorder / conversion disorder (the syndrome):** defining criterion is a motor or sensory symptom, genuinely experienced, shown by a *positive* sign of internal inconsistency to be incompatible with neurological disease (Hoover's sign, tremor entrainment, tubular field); it is a *rule-in* diagnosis, not one of exclusion, no stressor is required (DSM-5-TR) and la belle indifference is non-diagnostic. First step: elicit a positive sign at the bedside, then keep screening for coexisting organic disease.

## 15.4 Case two: a fugue told apart from an organic amnesia

**Direction of the loss, and the fate of identity.** case: a man in his thirties is brought in by railway police, found several hundred kilometres from home, unable to give his name, address or occupation, and calm rather than distressed. The sensorium is clear, there are no focal neurological signs, and he learns and recalls a new list of three objects without difficulty; a relative later reports he left home the morning after a large debt notice arrived. The reasoning is a positive comparison, not a hunch. Intact new learning, a clear sensorium, a *lost personal identity* and a clear psychological precipitant point to a dissociative fugue with generalised dissociative amnesia, because the organic amnestic syndrome is the mirror image, an anterograde failure to form new memory with personal identity preserved. Loss of the name is a psychogenic flag until proven otherwise. Screening for dissociation itself uses the Dissociative Experiences Scale, where a mean around **30** flags a probable dissociative disorder, set out in the accompanying scale plate.

The rule-in does not end the work. Criterion C still requires the organic mimics to be excluded: transient global amnesia (an abrupt, isolated anterograde amnesia with repetitive questioning, identity preserved, resolving within some hours), a complex partial seizure or epileptic automatism (confusion, no assumption of a new identity, an abnormal EEG), the amnestic (Korsakoff) syndrome and a substance blackout. Only when those are cleared does the label fix, and the treatment is safety, contact with family and a gentle, phased recovery of the memory, never a sedative.

**GOOD TO REMEMBER**

- **Dissociative amnesia and fugue (the syndrome):** DSM-5-TR Criterion A is an inability to recall important autobiographical information, usually traumatic or stressful, that is inconsistent with ordinary forgetting (localised is the commonest type); fugue is the specifier "with dissociative fugue", apparently purposeful travel or bewildered wandering with amnesia for identity. The deficit is retrograde and reversible; the first step is to exclude an organic cause (Criterion C) and to check whether personal identity is lost, which points away from an organic amnesia.

## 15.5 Case three: consolidating an alprazolam dependence onto a long-acting agent

**Consolidate, convert, then come down.** case: a man has been taking alprazolam (Alprax, Restyl) for insomnia and worry, the dose creeping up over many months, and now describes anxiety and tremor 3 to 4 hours after each tablet that he reads as the illness returning. That interdose rebound, 3 to 4 hours after a short-acting high-potency agent, is the fingerprint of dependence, not relapse. The evidence-based taper (BAP 2025; Maudsley) begins by *consolidating* him onto a single long-acting benzodiazepine, diazepam (Valium, Calmpose) or clonazepam (Clonotril,

Rivotril), using the verified diazepam-equivalence below; where the liver is impaired, lorazepam (Ativan, Loraz) or oxazepam is used instead. The Maudsley table deliberately omits alprazolam, so no alprazolam equivalence is asserted here; in practice the difficult final alprazolam doses are eased by substituting clonazepam (Kaplan).

| BENZODIAZEPINE | DOSE EQUAL TO DIAZEPAM 10 MG (MG) |
|---|---|
| **Diazepam** | 10 |
| **Clonazepam** | **0.5** |
| **Lorazepam** | **1** |
| **Chlordiazepoxide** | 25 |

The consolidation switch uses the verified diazepam-equivalent doses (Maudsley Prescribing Guidelines, Table 3.29); alprazolam is not tabulated there, so its equivalence is not asserted.

### 15.6 Case three continued: reducing slowly and managing the withdrawal

**Small proportional steps, and never an abrupt stop.** Once consolidated, the dose comes down *gradually*: patients often tolerate reductions of about 10 percent of the last dose every 2 to 4 weeks (roughly 5 to 10 percent per month), the steps becoming smaller as the dose falls because the dose-to-effect relationship is hyperbolic, so the final reductions before cessation are often less than 1 mg of diazepam-equivalent (Maudsley). The usual maximum diazepam dose for anxiety is around 30 mg daily. Melatonin (Meloset, Zytonin) is a useful adjunct for the insomnia of withdrawal (BAP grade A). The withdrawal to manage is rebound anxiety and insomnia, autonomic arousal and perceptual disturbance, beginning within hours to a few days of stopping a short-acting agent but only 1 to 2 weeks after a long-acting one; the danger that governs the whole plan is *seizures*. The benzodiazepine use disorder as an addiction is developed in the Substance use disorders notes.

**TRAP** **Stopping the benzodiazepine abruptly.** Abrupt cessation risks a withdrawal syndrome of rebound anxiety, autonomic arousal and, dangerously, *seizures* (Maudsley: it can be fatal); the whole point of consolidating onto a long-acting agent and reducing in small proportional steps is to never stop suddenly.

| | | | |
|---|---|---|---|
| **10%** USUAL TAPER STEP OF THE LAST DOSE EVERY 2 TO 4 WEEKS | **0.5 mg** CLONAZEPAM EQUAL TO DIAZEPAM 10 MG (MAUDSLEY) | **30** USUAL MAX DIAZEPAM FOR ANXIETY (MG PER DAY) | **3 to 4 h** INTERDOSE REBOUND AFTER A SHORT-ACTING AGENT |

**GOOD TO REMEMBER**

- **Diazepam (Valium, Calmpose):** the long-acting anchor of the taper, oral 4 to 40 mg per day in divided doses; a GABA-A positive allosteric modulator with the long-lived active metabolite nordiazepam; verified equivalence, diazepam 10 mg equals clonazepam 0.5 mg equals lorazepam 1 mg equals chlordiazepoxide 25 mg (Maudsley Table 3.29).
- **Alprazolam (Alprax, Restyl):** short-to-intermediate high-potency benzodiazepine, anxiolytic range 1 to 4 mg per day, with 5 to 6 mg per day reserved for panic; the default Indian anxiolytic, the most toxic in overdose and the hardest to taper, with interdose rebound at 3 to 4 hours and a manufacturer short-course limit of 2 to 4 weeks.

### 15.7 Case four: selecting the anxiolytic to the patient and the goal

**Match the agent to the target, and spare the benzodiazepine.** case: three patients ask for "something for the nerves", and the right answer differs for each. A junior doctor with palpitations and hand tremor only before an examination viva needs the *somatic* surge blunted for a discrete event: propranolol (Ciplar, Inderal) 10 to 40 mg taken about 20 to 30 minutes before the performance blocks the peripheral autonomic symptoms without sedation or dependence (Kaplan and Sadock, CTP 2024). A woman with chronic generalised anxiety and mild COPD, already buying alprazolam from a chemist, needs a sustained non-dependence anxiolytic: an SSRI is the first-line agent, with buspirone (Buspin), a 5-HT1A partial agonist with no dependence or respiratory depression, as the benzodiazepine-sparing option, provided she is told it takes about a fortnight to work and is useless taken as-needed. A student with a short run of stress-related sleep-onset insomnia needs the briefest possible cover: zolpidem (Zolfresh, Nitrest) 10 mg at bedtime for 7 to 10 nights, or melatonin, with CBT for insomnia as the durable answer. The accompanying figure lays these choices out as an agent-selection map.

| PATIENT AND GOAL | FIRST CHOICE (INDIAN BRAND) | DOSE (MG PER DAY) | WHY NOT A BENZODIAZEPINE |
|---|---|---|---|
| **Performance or situational anxiety (palpitations, tremor)** | propranolol (Ciplar, Inderal) | **10 to 40** | blunts the autonomic surge without sedation or dependence |
| **Sustained GAD with respiratory disease or a substance history** | SSRI first-line; buspirone (Buspin) as the non-dependence add-on | buspirone 15 to 30 | no dependence, no respiratory depression, but two weeks to work |
| **Short-term sleep-onset insomnia** | zolpidem (Zolfresh, Nitrest) or melatonin (Meloset, Zytonin) | zolpidem 10 | z-drug complex-sleep-behaviour and dependence caution; CBT-I is durable |
| **Acute severe crisis, alcohol withdrawal, status epilepticus, catatonia** | a benzodiazepine (diazepam or lorazepam) | **short course only** | this IS the benzodiazepine's genuine niche, time-limited |

The agent-selection map: the anxiolytic is matched to the patient and the goal, and the benzodiazepine is spared for its genuine acute niche. The SSRIs that outrank all of these for sustained anxiety are developed in the Mood disorders: pharmacotherapy notes.

### 15.8 Case four continued: applying the short-course rule

**Not first-line, not for maintenance.** The anchor that decides all three sub-cases is the **short-course rule**: a benzodiazepine is not a first-line long-term anxiolytic or hypnotic. For generalised anxiety an SSRI is first-line (BAP 2014; NICE CG113 names sertraline first), and NICE says a benzodiazepine may be offered for generalised anxiety disorder only as a short-term crisis measure of no more than 2 to 4 weeks; for chronic insomnia CBT for insomnia is first-line (BAP 2019), with a z-drug or benzodiazepine only as a brief option matched to the pattern. Two corollaries fall out for this case. The delayed onset of buspirone means the woman must not be switched abruptly from alprazolam to buspirone, which does not cover benzodiazepine

withdrawal; she takes a therapeutic buspirone dose for two to four weeks first, and only then is the alprazolam tapered. And flumazenil, the benzodiazepine antagonist, is not a shortcut out of dependence, because it can precipitate seizures in a dependent patient. The wider anxiety pharmacology sits in the Anxiety, OCD & trauma-related disorders notes, and clinical insomnia and CBT-I in the Eating & sleep-wake disorders notes.

**GOOD TO REMEMBER**

- **Propranolol (Ciplar, Inderal):** a peripheral beta-adrenergic blocker for somatic and performance anxiety, 10 to 40 mg taken about 20 to 30 minutes before a performance (Kaplan and Sadock, CTP 2024); it blunts palpitations and tremor without sedation or dependence and has no effect on the psychic component.
- **Buspirone (Buspin):** a 5-HT1A partial agonist, 15 to 30 mg per day (maximum 60 mg per day), short half-life of about 2 to 3 hours but a delayed onset of two weeks or more, so never a PRN rescue; no dependence, sedation, anticonvulsant effect or abuse.
- **Zolpidem (Zolfresh, Nitrest):** a z-drug at the benzodiazepine-1 (omega-1) GABA-A subtype, 10 mg at bedtime for 7 to 10 nights (short half-life about 2.5 hours); the signature caution is complex sleep behaviours with dependence and next-morning impairment.

### 15.9 The single method behind all four

**One move, four applications.** Stand back and the four cases are one skill practised four ways. The two diagnostic cases rule a functional or dissociative disorder *in* on a positive finding, then keep the organic search open. The two managerial cases apply the short-course discipline, consolidating and tapering a dependence rather than continuing it or stopping it abruptly, and matching the anxiolytic to the patient rather than reaching for the default alprazolam. Across all four the reflex to resist is the same, the sedative given in place of an assessment, which is precisely the habit the Indian benzodiazepine-sparing position exists to correct.

**INDIA**

Every case here has an Indian edge. The benzodiazepine over-prescription problem is real, and **alprazolam** (Alprax, Restyl) is the default anxiolytic reached for reflexively and continued long-term; benzodiazepines are among the most frequently prescribed drugs, and a benzodiazepine should not ordinarily run beyond 2 to 4 weeks. The Indian Psychiatric Society position, in line with BAP and NICE, is that an SSRI or SNRI is first-line for generalised anxiety and that a benzodiazepine is a short-course measure only, so buspirone and propranolol are genuinely useful in the patient who would otherwise be parked on alprazolam. The culture-bound edge is just as genuine: possession and trance states, in which a deity, spirit or ancestor is felt to take over, are a common idiom of distress and can present with the amnesia and purposeful travel of case two; ICD-11 keeps these as separate categories (trance disorder 6B62, possession trance disorder 6B63), and a sanctioned, non-distressing ritual state is not a disorder at all. Read them through a cultural formulation, not a sedative, as developed in the Consultation-liaison, geriatric & transcultural psychiatry notes.

**PEARL**

The most powerful move in three of these four cases is a demonstration rather than a drug: the patient's own Hoover's sign or entraining tremor proves the functional diagnosis and its reversibility, the intact new learning proves the amnesia is not organic, and the interdose rebound proves the anxiety is dependence, not relapse. In each, the finding that rules the diagnosis in is also the thing you show the patient to explain it.

**HOLD THIS**

Win each case with a positive move: rule a functional disorder in on a sign that is weak on command but strong on distraction (Hoover, entrainment, tubular field), separate a dissociative from an organic amnesia by a retrograde loss with identity gone but new learning intact, and treat a benzodiazepine dependence by consolidating onto a long-acting agent (diazepam or clonazepam) and tapering slowly because abrupt cessation causes *seizures*, since a benzodiazepine is never the first-line long-term anxiolytic.

For the receptor detail behind the benzodiazepine mechanism see the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes; for the video-EEG separation of dissociative from epileptic events the Epilepsy & psychiatry notes; for the lorazepam challenge and benzodiazepine management of catatonia the Other psychotic disorders, delusional syndromes & catatonia notes; for grounding, abreaction and hypnosis in the recovery of dissociative memory the Psychotherapies, clinical assessment, MSE & nosology notes; and for the somatic-symptom, functional and factitious boundary the Somatic-symptom, sexual, impulse-control disorders & suicide notes.

## 16 · Consolidation

**The last step is not new material, it is retrieval.** This closing topic exists to turn a chapter you have *read* into a chapter you can *reproduce* under exam pressure. The evidence is one-directional: testing yourself on half-learned material, **retrieval practice**, fixes it far more durably than reading it again, and the most efficient way to study this material is to **cover the answer** and force each fact out of memory before checking it. Everything below is built for that, a **mnemonic** that reassembles the whole chapter from one line, answer-free **retrieval prompt** lists, two answer vaults to check against, and a whole-chapter **synthesis map** to reproduce.

Use it actively, not by rereading. Say the master mnemonic once, then close the notes and work the retrieval prompts with the answer covered; only then uncover the Good-to-remember vaults and mark what you missed. Finish by reproducing the synthesis map on a blank sheet. Do those three things and the chapter is consolidated; the gaps you find are exactly where to go back. The wider study technique of active recall and spaced retrieval sits alongside the clinical material, and the grounding and hypnosis techniques touched on across the dissociative half are developed in the Psychotherapies, clinical assessment, MSE & nosology notes.

**Fig 15.16** The whole-chapter synthesis map as a cover-the-columns grid: each dissociative entity reduced to its one discriminator and first step, and each anxiolytic-hypnotic class to its mechanism, use and key caution.

| A. Dissociative and conversion: entity, discriminator, first step | | |
|---|---|---|
| **entity** | **the one discriminator** | **the first step** |
| **dissociative amnesia and fugue** | retrograde loss, intact new learning; fugue is a specifier | exclude an organic amnestic cause |
| **dissociative identity disorder** | two or more identity states, plus recurrent amnesia | confirm the dissociative-amnesia criterion |
| **depersonalisation-derealisation** | detachment with intact reality testing ('as if') | confirm reality testing is intact (not psychosis) |
| **conversion / functional neurological disorder** | a positive rule-in sign, not a diagnosis of exclusion | elicit a positive sign |
| **possession and trance** | involuntary, outside accepted cultural or religious practice | apply the cultural-exclusion criterion |

| B. Anxiolytics and hypnotics: class, mechanism, use and key caution | | |
|---|---|---|
| **class** | **mechanism** | **use and the key caution** |
| **benzodiazepines** | GABA-A positive allosteric modulator; frequency, not duration | short course only; dependence and seizure on abrupt withdrawal |
| **z-drugs (zolpidem, zopiclone)** | benzodiazepine-1 (omega-1) GABA-A subtype | short-term sleep onset; complex sleep behaviours |
| **buspirone** | 5-HT1A partial agonist | sustained anxiety, spares BZD; delayed onset, no dependence |
| **DORAs (suvorexant, lemborexant)** | orexin (hypocretin) receptor antagonist | insomnia; blocks the wake-promoting orexin system |
| **melatonergic (melatonin, ramelteon)** | MT1 / MT2 receptor agonist | sleep onset; prolonged-release an option over age 55 |
| **adjuncts (propranolol, hydroxyzine, pregabalin)** | beta-blocker, H1 antihistamine, alpha-2-delta ligand | somatic and acute anxiety; none is a GABA-A hypnotic |

structure: section and column headers · the exam anchor to hold · the danger to hold

## 16.1 The master mnemonic, paid off

**One line rebuilds the whole chapter.** The chapter opened by seeding a single **mnemonic**, and this is where it is redeemed. *MIND SPLITS, BODY SPEAKS, GABA SLEEPS* is not decoration, each beat is a doorway into a third of the material, and saying the line should make the branches beneath it unfold. **MIND SPLITS** recovers the psychoform dissociative disorders of consciousness, memory and identity; **BODY SPEAKS** recovers conversion, the somatoform half; **GABA SLEEPS** hands over to the anxiolytic and hypnotic pharmacology that acts on the GABA-A receptor. If the line is solid, the chapter is recoverable from it.

MNEMONIC

### MIND SPLITS, BODY SPEAKS, GABA SLEEPS

**MIND SPLITS** unfolds to the mind-side entities, dissociative *amnesia* (autobiographical memory), *identity* disorder and its partial form (identity), *depersonalisation-derealisation* (self and reality), and *trance* and *possession trance* (awareness and identity-replacement). **BODY SPEAKS** unfolds to the one body-side entity, conversion or dissociative neurological symptom disorder, diagnosed by positive signs. **GABA SLEEPS** unfolds to the benzodiazepine and its comparators, the mechanism (frequency, not duration), the half-life classes, dependence and the taper, flumazenil, and the short-course rule. Three beats, three branches, the whole chapter.

## 16.2 How to run the recall

**Cover, retrieve, check, redraw.** The mechanics of **active recall** for this chapter are fixed and worth stating so the rest of the topic is usable. First, **cover the answer**, keeping the two Good-to-remember vaults below hidden. Second, work each **retrieval prompt** out loud or on paper from memory. Third, uncover the vault and mark every miss. Fourth, close on the **synthesis map** and **redraw from memory** on a blank sheet. The order matters, because the effortful pull of retrieval is what lays down the memory, so a prompt you struggle with and then check is worth more than a paragraph reread. That is why the prompt lists below are deliberately answer-free.

RULE **Three anchors hold the chapter.** One split (dissociation of the mind versus dissociation of the body), one mechanism (the benzodiazepine raises the *frequency* of chloride-channel opening, not its duration), and one management rule (the benzodiazepine is a short-course drug, never a first-line long-term anxiolytic). Recover those three and the detail hangs off them.

## 16.3 Retrieval prompts: the dissociative discriminators

**Cover the vaults below and answer from memory.** Each prompt targets the single discriminator the examiner tests, not a general description. Work them as **retrieval practice**, then check against the criterion vault.

1. Name the seven ICD-11 dissociative types and, for each, the one integrative function that has failed.
2. What single discriminator separates dissociative amnesia from an organic amnestic syndrome, and is fugue a disorder or a specifier?
3. Beyond two or more distinct personality states, which criterion is mandatory for dissociative identity disorder, and what two competing models explain its cause?
4. What distinguishes partial dissociative identity disorder from the full disorder?
5. What keeps depersonalisation-derealisation disorder on the non-psychotic side of the line, and what scale threshold is conventionally diagnostic?
6. State the cultural-exclusion criterion that both trance disorder and possession trance disorder must satisfy, and how the two differ.
7. Where does conversion sit in ICD-11 versus DSM-5-TR, and why is that the pivotal filing difference?

### 16.4 Retrieval prompts: the conversion signs and the measure

**Still covered.** These carry the highest bedside yield of the mind-and-body half, the positive signs that rule conversion *in*, and the screen that flags dissociation for interview.

1. Name the five positive rule-in signs of a functional neurological symptom, and say what each one demonstrates.
2. Is conversion a diagnosis of exclusion, and is an identifiable psychological stressor required? What is the diagnostic status of la belle indifference?
3. Which single sign would you elicit first at the bedside for a functional leg weakness, and what is the positive finding?
4. What is the conventional Dissociative Experiences Scale (DES) screening cut-off, and what do you do for a patient who scores above it?
5. What does the DES-Taxon subscale add, and what does the SDQ-20 capture that the DES does not?

**GOOD TO REMEMBER**

- **Dissociative amnesia:** defining criterion is an inability to recall important autobiographical information inconsistent with normal forgetting; the deficit is *retrograde* with intact new learning (the discriminator from organic amnesia); fugue is a specifier. First step: exclude an organic amnestic cause.
- **Dissociative identity disorder:** two or more distinct personality states (or possession) *plus* recurrent dissociative amnesia; the amnesia criterion is the discriminator; trauma / structural-dissociation model versus the sociocognitive (fantasy) model.
- **Depersonalisation-derealisation disorder:** persistent detachment with *intact reality testing* and an "as if" quality, the intact reality check being the discriminator from psychosis; a Cambridge Depersonalisation Scale score of 70 or more is the conventional threshold.
- **Trance / possession trance disorder:** narrowed awareness with identity intact (trance) versus identity replaced by an external agent with amnesia (possession); both require the state to be involuntary, distressing and *outside accepted cultural or religious practice*. First step: apply the cultural-exclusion criterion.
- **Dissociative / functional neurological symptom disorder:** a neurological symptom with a *positive* sign of internal inconsistency (Hoover's sign, tremor entrainment, give-way weakness, tubular visual field, astasia-abasia); it is *not* a diagnosis of exclusion, no stressor is required, and la belle indifference is unreliable. First step: elicit a positive sign.

### 16.5 Retrieval prompts: the benzodiazepine mechanism and kinetics

**Cover the dose vault below.** The pharmacology half rewards the mechanism first, then the half-life sort, because everything clinical follows from those two.

1. State the benzodiazepine mechanism at the GABA-A receptor, which parameter of chloride-channel opening it raises, where its binding site lies, and how the barbiturate differs.
2. Why is a benzodiazepine relatively safe in a pure overdose while a barbiturate is not?
3. Sort lorazepam, alprazolam, clonazepam, diazepam and chlordiazepoxide into short-to-intermediate versus long-acting, and name the shared active metabolite of the long-acting agents.

4. Which three benzodiazepines have no active metabolite, making them the liver-safe choice, and what mnemonic names them?
5. Which receptor subtype carries sedation, and which carries anxiolysis?

### 16.6 Retrieval prompts: withdrawal, taper and selection

**Still covered.** These are the single most examinable management statements of the material, so retrieve them cleanly and check against the dose vault.

1. What are the dangerous features of benzodiazepine withdrawal, and why must the drug never be stopped abruptly?
2. Describe the taper, onto which agent you consolidate and at what rate you reduce.
3. Name the benzodiazepine antagonist, its half-life problem, and its signature hazard.
4. What is the first-line pharmacological treatment for generalised anxiety disorder, precisely where does the benzodiazepine sit, and what is the NICE ceiling on a benzodiazepine course?
5. What is first-line for chronic insomnia, and which non-benzodiazepine anxiolytic works only after a delay and carries no dependence?

| 30 | 0.5 to 6 | 20 to 50 | 10% |
|---|---|---|---|
| DES SCREENING CUT-OFF | ALPRAZOLAM ANXIETY DOSE (MG PER DAY) | DIAZEPAM ELIMINATION HALF-LIFE (HOURS) | TAPER: FRACTION OF LAST DOSE EVERY 2 TO 4 WEEKS |

**GOOD TO REMEMBER**

- **Diazepam (Valium, Calmpose):** GABA-A positive allosteric modulator; 4 to 40 mg per day in divided doses; long-acting through the active metabolite nordiazepam; the consolidation anchor of the taper, with diazepam 10 mg equal to clonazepam 0.5 mg, lorazepam 1 mg and chlordiazepoxide 25 mg (Maudsley).
- **Alprazolam (Alprax, Restyl):** short-to-intermediate (half-life 6 to 20 hours), high-potency; anxiety 1 to 4 mg per day (5 to 6 mg per day is the panic figure, not the anxiolytic range); the most toxic benzodiazepine in overdose and the hardest to taper, and the Indian default anxiolytic despite both.
- **Clonazepam (Clonotril, Rivotril):** long-acting (half-life 30 to 40 hours), high-potency; panic 0.5 to 2 mg per day; the other taper-consolidation agent and the substitute that eases the final doses of alprazolam.
- **Lorazepam (Ativan, Loraz):** short-to-intermediate (half-life 10 to 20 hours), no active metabolite so liver-safe; oral 2 to 6 mg per day; the catatonia challenge agent at 1 to 2 mg.
- **Buspirone (Buspin):** a 5-HT1A partial agonist, 20 to 30 mg per day; a *delayed* onset of 2 weeks or more with no dependence, sedation or anticonvulsant effect, so it spares the benzodiazepine in sustained anxiety but is useless acutely.
- **Zolpidem (Zolfresh, Nitrest):** a z-drug at the benzodiazepine-1 (omega-1) GABA-A subtype, 10 mg at bedtime for short-term use; the signature caution is complex sleep behaviours, dependence and next-day impairment.
- **Flumazenil (the antagonist):** a competitive benzodiazepine-receptor antagonist with a short half-life of 41 to 79 minutes (so re-sedation and repeat dosing); it can *precipitate seizures* in benzodiazepine dependence and in mixed or cyclic-antidepressant overdose, so it is used selectively.

### 16.7 The whole-chapter synthesis map

**Cover the branches and redraw from memory.** The final and highest-value exercise is to reproduce the chapter as one picture. The accompanying figure sets out the **synthesis map** as a single tree, a trunk labelled by the master mnemonic, three primary branches (mind, body, GABA), and the named entities and anchors hanging off each. Study it once, then cover every branch and **redraw from memory** on blank paper, writing the discriminator or the dose at each leaf. The mind branch should regrow its five entities, each with its failed integrative function; the body branch its one entity with the positive-sign list; the GABA branch the mechanism, the half-life classes, the withdrawal-and-taper, flumazenil and the short-course rule. A branch you cannot regrow is a branch to reread. This cover-the-branches redraw is the most compact single test of whether the chapter is genuinely consolidated.

**INDIA**

Two Indian threads run the length of this chapter and belong on the synthesis map. On the pharmacology side, India carries a genuine benzodiazepine over-prescription problem, with **alprazolam** the reflex default anxiolytic despite being the most toxic in overdose and the hardest to taper; the Indian Psychiatric Society position and the wider benzodiazepine-sparing principle favour the SSRI or SNRI for sustained anxiety and CBT for insomnia, keeping the benzodiazepine to a genuine short course. On the dissociative side, trance, possession and conversion presentations are among the most frequent dissociative pictures in Indian practice, which makes the cultural-exclusion criterion and a proper cultural formulation, not a reflex sedative, the examinable core. The transcultural reading is expanded in the Consultation-liaison, geriatric & transcultural psychiatry notes.

**TRAP** **The chapter's recurring errors, in one line.** Missing an organic cause behind a "conversion" picture, diagnosing dissociative identity disorder without the dissociative-amnesia criterion, reading depersonalisation-derealisation as psychosis, starting a benzodiazepine for chronic anxiety or insomnia, and stopping a benzodiazepine abruptly and risking a seizure, five traps, one per section.

**HOLD THIS**

If you can say *MIND SPLITS, BODY SPEAKS, GABA SLEEPS* and, from it, regrow the mind-side discriminators, the conversion positive signs, the DES cut-off of 30, the frequency-not-duration mechanism and the never-abrupt, short-course-only benzodiazepine rule, the chapter is consolidated; wherever you cannot, that gap is the reread.

For the receptor systems behind the GABA-A detail see the Neurochemistry, Neuroendocrinology, Neuroimaging & Basic Psychopharmacology notes; for the sedative and benzodiazepine use disorder as an addiction the Substance use disorders notes; for the SSRIs and SNRIs that outrank the benzodiazepine as first-line anxiolytics the Mood disorders: pharmacotherapy notes; and for chronic insomnia and CBT for insomnia the Eating & sleep-wake disorders notes.

## Previously asked

This territory is examined at two levels at once: the syndrome (the dissociative and conversion presentations, the positive signs that rule them in, the organic differential that must be excluded and the management that follows) and the pharmacology (the benzodiazepines and the wider anxiolytic and hypnotic agents, their receptor action and the dependence and withdrawal they carry). The dissociative and conversion questions on record are written in the older hysteria vocabulary, so the same case can be labelled hysterical unconsciousness, conversion disorder or functional neurological disorder across sittings, and a strong answer reconciles the ICD-11 and DSM-5 naming with the term used in the stem. Every long essay ends in a differential and a guideline-anchored management plan, so the organic-versus-functional decision and the first-line plan must be exam-ready. The genuine questions on record are grouped below. The clinical anxiety and obsessive-compulsive disorders these agents actually treat are developed in the Anxiety, obsessive-compulsive and trauma- and stressor-related disorders notes; the somatic-symptom and illness-anxiety boundary that sits beside conversion is developed in the Somatic-symptom, sexual, impulse-control disorders and suicide notes; and the GABA-A receptor pharmacology behind the anxiolytics and hypnotics is developed in the Neurochemistry, neuroendocrinology, neuroimaging and basic psychopharmacology notes, while the dissociative classification, the positive functional signs, the conversion management and the anxiolytic and hypnotic agents are owned here.

### HOW THIS TERRITORY IS ACTUALLY TESTED

Q **Conversion disorder and the dissociative presentation of unconsciousness.** "Define consciousness. Differential diagnosis for unconsciousness in middle aged male. How to investigate and manage a case of Hysterical Unconsciousness?" has come as a full 25-mark essay, "Discuss etiology, clinical features and management of Hysterical Conversion Disorder" has come as a 25-mark essay, and "Clinical features and differential diagnosis of Conversion Disorder" has come as a 25-mark essay in its own right. Prepare the conversion and functional-neurological presentation, the positive signs that rule the disorder in (Hoover's sign, the hip-abductor and give-way patterns, tremor entrainment and the tubular visual field), the organic differential for unconsciousness that must be excluded first, the dissociative mechanism behind the symptom and the caveat that la belle indifference is neither sensitive nor specific, and the management line (exclude organic disease, explain the diagnosis as a real and reversible one, physiotherapy-led rehabilitation and treatment of the comorbid depression or anxiety). **25-mark essay, 2013 / 2014 / 2016**

Q **Benzodiazepine dependence and the anxiolytic and hypnotic agents.** "Benzodiazepine Dependence" has come as a standalone 10-mark note. Prepare the receptor pharmacology (the GABA-A benzodiazepine site and its chloride-channel action), the tolerance and dependence that follow continued use, the withdrawal syndrome with its rebound anxiety, perceptual disturbance and seizure risk, the over-prescription context that drives the problem in Indian practice where a somatising anxious patient is handed alprazolam or clonazepam rather than started on the agent that treats the disorder, and the management (a slow graded taper, the switch to a long-acting agent such as diazepam, and the reservation of these drugs for short-term crisis use only). **10-mark note, 2016**

Prepare this material to be diagnosed and treated, not just recited: the organic-versus-functional differential, the positive functional-sign figures, the dissociative classification set against the ICD-11 and DSM-5, the dose-anchored good-to-remember boxes and the benzodiazepine taper schedule in these notes are built to the way the block is examined. The dissociative amnesia and fugue, the depersonalisation and derealisation, the dissociative identity and the possession and trance

presentations are not on the question record but sit squarely inside the classification the stems ask you to apply, and the non-benzodiazepine anxiolytics and hypnotics (the Z-drugs, buspirone, melatonin and ramelteon, and flumazenil as the antidote) are catalogued as an essay only in the basic-pharmacology paper, so both are prepared here to the same depth. The clinical anxiety and obsessive-compulsive disorders these agents treat live in the Anxiety, obsessive-compulsive and trauma- and stressor-related disorders notes; the somatic-symptom and illness-anxiety boundary in the Somatic-symptom, sexual, impulse-control disorders and suicide notes; and the receptor-level pharmacology in the Neurochemistry, neuroendocrinology, neuroimaging and basic psychopharmacology notes.

## Quick review in one table

| THE DISSOCIATIVE DISORDERS OF THE MIND: THE DISCRIMINATOR AND THE FIRST STEP | |
|---|---|
| THE ORGANISING SPLIT | Dissociation of the **mind** (consciousness, memory, identity) versus the **body** (motor and sensory control). ICD-11 keeps conversion inside the family (6B60); DSM-5-TR exports it to the somatic disorders. First step: name which integrative function came apart and the diagnosis names itself. |
| DISSOCIATIVE AMNESIA (AND FUGUE) | Discriminator: a **retrograde** loss of autobiographical memory with **new learning intact** and identity often lost (the mirror of organic amnesia); localised is commonest, fugue is a specifier. First step: exclude an organic amnestic cause. |
| DISSOCIATIVE IDENTITY DISORDER | Discriminator: two or more distinct identity states **plus recurrent dissociative amnesia** (the required, most-tested criterion; drop it and it is other specified dissociative disorder). First step: confirm the amnesia criterion. Debate: trauma model versus sociocognitive (fantasy) model. |
| PARTIAL DISSOCIATIVE IDENTITY DISORDER | Discriminator: one dominant state runs daily life, intruded on by non-dominant states that do **not** take executive control (full disorder = states seize control, partial = they only intrude); an ICD-11 category, filed under other specified dissociative disorder in DSM-5-TR. |
| DEPERSONALISATION-DEREALISATION DISORDER | Discriminator: persistent detachment from self and/or world with an "as if" quality and **intact reality testing** (the separator from psychosis). First step: confirm reality testing is preserved. A Cambridge Depersonalisation Scale score of 70 or more is the conventional threshold. |
| TRANCE DISORDER | Discriminator: narrowed awareness of surroundings with a small, seemingly uncontrolled behavioural repertoire, the **self still intact** (no identity replacement); normal EEG even during episodes. First step: apply the cultural-exclusion criterion and clear the organic mimics. |
| POSSESSION TRANCE DISORDER | Discriminator: the sense of identity is **replaced by an external possessing agent** (spirit, deity, demon) with amnesia; the external attribution separates it from dissociative identity disorder. First step: apply the cultural-exclusion criterion (a sanctioned, non-distressing ritual is not a disorder). |
| **CONVERSION, ITS SEIZURE FORM, AND MEASURING DISSOCIATION** | |
| CONVERSION (DISSOCIATIVE NEUROLOGICAL SYMPTOM DISORDER) | Discriminator: a motor or sensory symptom with a **positive sign of internal inconsistency** (Hoover's sign, tremor entrainment, tubular field), weak on command but strong on distraction; a **rule-in** diagnosis, no stressor required, la belle indifference unreliable. First step: elicit a positive sign. |

| CONVERSION, ITS SEIZURE FORM, AND MEASURING DISSOCIATION | |
|---|---|
| DISSOCIATIVE (NON-EPILEPTIC) CONVULSIONS | Discriminator: a seizure-like attack with preserved awareness in bilateral shaking, closed and resisted eyes, a long fluctuating course, no post-ictal prolactin rise, and a **normal ictal video-EEG** (the gold standard). Tongue-biting and incontinence do not distinguish it. First step: capture the attack on video-EEG. |
| MEASURING DISSOCIATION (DES) | The Dissociative Experiences Scale is the self-report screen; a **mean of 30 or more** flags a probable dissociative disorder and prompts a structured interview (SCID-D, DDIS). The DES-Taxon isolates pathological dissociation; the SDQ-20 captures somatoform dissociation. A high score is a flag, never a diagnosis. |

| THE GABA-A SEDATIVE-HYPNOTICS: MECHANISM, USE AND CAUTION | |
|---|---|
| BENZODIAZEPINES (THE CLASS) | A **positive allosteric modulator** at the GABA-A receptor (benzodiazepine site, alpha-gamma interface): raises the **frequency** of chloride-channel opening only when GABA is present (barbiturates raise the **duration**). GABA-dependence gives a ceiling, so relatively safe in pure overdose. Alpha-1 sedation, alpha-2 anxiolysis. |
| AGENTS AND HALF-LIFE | Short-to-intermediate: lorazepam (10 to 20 h, no metabolite, liver-safe), **alprazolam** (12 to 15 h, high-potency). Long-acting through shared nordiazepam: diazepam, chlordiazepoxide; clonazepam (30 to 40 h). LOT group (lorazepam, oxazepam, temazepam) has no active metabolite. Alprazolam **1 to 4 mg per day for anxiety** (5 to 6 mg per day only for panic), most toxic in overdose, hardest to taper. |
| WITHDRAWAL AND TAPER | Withdrawal: rebound anxiety, perceptual disturbance and autonomic arousal, with the criterion-level dangers of **seizures**, psychosis and delirium. **Never stop abruptly.** Taper: consolidate onto a long-acting agent (diazepam or clonazepam), reduce about 10% of the last dose every 2 to 4 weeks (diazepam 10 = clonazepam 0.5 = lorazepam 1 = chlordiazepoxide 25 mg). |
| FLUMAZENIL (THE ANTAGONIST) | The competitive benzodiazepine-receptor **antagonist**; short half-life 41 to 79 minutes, so re-sedation and repeat dosing. Signature caution: it can **precipitate seizures** in benzodiazepine dependence and in mixed or cyclic-antidepressant overdose, so it is used selectively, not routinely. |
| Z-DRUGS (ZOLPIDEM, ZOPICLONE, ZALEPLON, ESZOPICLONE) | Non-benzodiazepine hypnotics that still modulate the GABA-A benzodiazepine site, selective for the **omega-1 (alpha-1)** subtype. Use: a short-term hypnotic after CBT for insomnia, matched by half-life (zolpidem 5 to 10 mg for onset, zopiclone for maintenance). Caution: **complex sleep behaviour**, dependence, next-day impairment. |

| THE NON-GABA ANXIOLYTICS AND THE OTHER HYPNOTICS | |
|---|---|
| BUSPIRONE (AZAPIRONE) | A **5-HT1A partial agonist**, not a GABA drug; 15 to 30 mg per day (short half-life 2 to 3 h). Use: acute GAD, sitting after an SSRI and before a benzodiazepine. Caution: a **delayed onset of 2 weeks or more**, so useless as a PRN, but no dependence, sedation or anticonvulsant effect. |
| GABAPENTINOIDS (PREGABALIN, GABAPENTIN) | **Alpha-2-delta calcium-channel ligands** (despite the name, not GABA drugs). Pregabalin 150 to 600 mg per day is the licensed **alternative first-line** for GAD; gabapentin 900 to 1800 mg per day is off-label. Caution: now recognised drugs of **misuse and dependence**, never stop abruptly (seizure). |
| PROPRANOLOL (BETA-BLOCKER) | A peripheral **beta-adrenergic antagonist**: strips the palpitations and tremor, not the dread. Use: the somatic symptoms of **performance and social anxiety**, a single 10 to 40 mg dose about an hour before the event. Caution: no effect on psychic anxiety; avoid in asthma and bradycardia. |
| HYDROXYZINE (ANTIHISTAMINE) | A sedating **H1 antagonist**; 50 to 100 mg up to four times a day (half-life about 20 h). Use: efficacious in **acute GAD** (BAP), no dependence, not controlled. Caution: sedation, anticholinergic effects, and QT prolongation at higher doses. |
| ETIFOXINE | A non-benzodiazepine anxiolytic with a **dual action**: direct GABA-A modulation plus translocator-protein (TSPO)-driven neurosteroid (allopregnanolone) synthesis; about 150 to 200 mg per day. Non-inferior to lorazepam and alprazolam over 4 weeks with less rebound. Caution: rare hepatic and severe cutaneous reactions; an alternative, not a first-line SSRI. |
| MELATONERGIC AGONISTS (MELATONIN, RAMELTEON) | **MT1/MT2 agonists** on the suprachiasmatic clock (MT1 onset, MT2 phase-shift), not the GABA channel. Use: prolonged-release melatonin 2 mg for the over-55 and as a taper-insomnia adjunct; ramelteon 8 mg for sleep-onset. Caution: a modest effect, no dependence; ramelteon with fluvoxamine is dangerous (CYP1A2). |
| DUAL OREXIN RECEPTOR ANTAGONISTS (DORAS) | **Dual OX1/OX2 orexin (hypocretin) antagonists** that promote sleep by subtracting wakefulness, not by adding GABAergic sedation. Use: insomnia when CBT for insomnia is not the route (suvorexant 10 to 20, lemborexant 5 to 10, daridorexant 25 to 50 mg nocte). Caution: next-morning somnolence, so leave a 7-hour sleep window. |

| THE GUIDELINE SPINE AND THE HARD RULES | |
|---|---|
| ANXIETY, FIRST-LINE | **An SSRI or SNRI is first-line** for GAD; pregabalin and buspirone are the alternatives, hydroxyzine and buspirone cover acute anxiety, and propranolol the performance type. A benzodiazepine is not first-line and not for maintenance. |

| THE GUIDELINE SPINE AND THE HARD RULES | |
|---|---|
| INSOMNIA, FIRST-LINE | **CBT for insomnia is first-line** for chronic insomnia; a hypnotic (benzodiazepine, z-drug, melatonin or a DORA) is a short-term option only, matched by half-life to the onset-versus-maintenance pattern. |
| THE BENZODIAZEPINE SHORT-COURSE RULE | A benzodiazepine is a **2 to 4 week crisis measure** at most (NICE), then tapered, and is **never stopped abruptly** because withdrawal seizures can be fatal. India's alprazolam-default habit is exactly the pattern the benzodiazepine-sparing position corrects. |
| THE DISSOCIATIVE FIRST-MOVE | For a possession, trance or dramatic dissociative presentation the first move is a **cultural formulation and exclusion of an organic cause**, not a reflex sedative; a sanctioned, non-distressing ritual is not a disorder, and dissociation has no benzodiazepine indication. |
| THE FIVE TRAPS | Missing an organic cause behind a "conversion"; diagnosing dissociative identity disorder **without the amnesia** criterion; reading depersonalisation-derealisation as psychosis; starting a benzodiazepine for chronic anxiety or insomnia; and stopping a benzodiazepine abruptly. |

## References

The dissociative, conversion and anxiolytic-and-hypnotic content is grounded in the standard psychiatry and psychopharmacology sources (the source hierarchy below), with Kaplan and Sadock as the depth bar for the dissociative phenomenology, the three pathogenesis models and the scale cut-offs, the New Oxford Textbook (Stone and Carson) for the positive-sign, rule-in approach to functional neurological disorder, and the Maudsley Prescribing Guidelines and Stahl as the depth bar for the receptor mechanisms, the half-life classes, the doses, the diazepam-equivalence table and the taper. The management is guideline-first, led by the British Association for Psychopharmacology (anxiety 2014, insomnia 2019, substance dependence), with NICE and the Indian Psychiatric Society. Every criterion, receptor detail, dose, half-life, equivalence and first-line or short-course statement was checked against these sources and the adversarial content pass; anything that could not be confirmed was omitted and flagged rather than guessed. Each journal PMID below was verified against PubMed this build and matched on title, first author and year; identifiers that resolved to the wrong paper were corrected or dropped. Books, classifications and guideline documents without a MEDLINE record are cited by author, title and year without a PMID.

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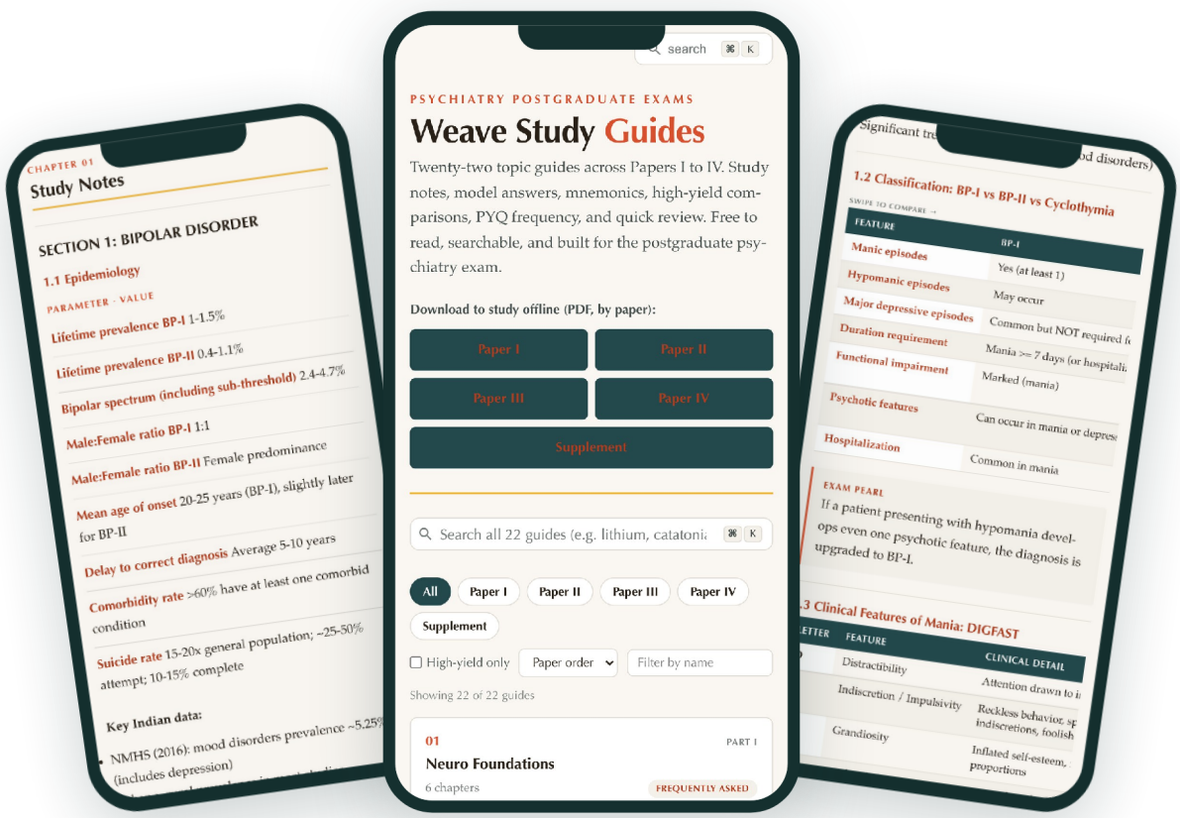
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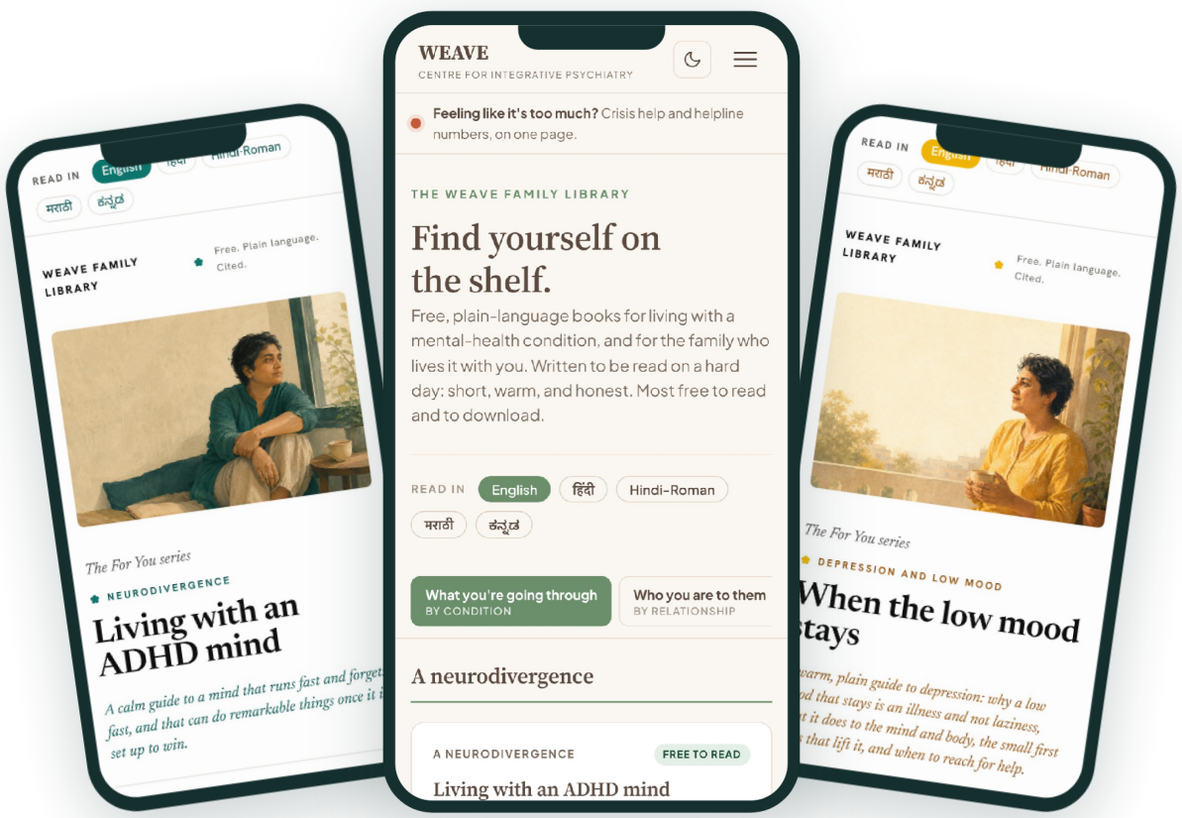
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