WEAVE SPRINT . PAPER II

Dissociation & anxiolytics

The dissociative and conversion disorders with the anxiolytic and hypnotic drugs in one document: dissociation and its classification, conversion or functional neurological disorder with its positive rule-in signs, depersonalisation, amnesia, fugue and the possession-trance states; then the benzodiazepines in depth, buspirone, the z-drugs, the orexin and melatonergic hypnotics and the other anxiolytics, led by the BAP guidelines and the Indian position throughout.

CONTENTS

Dr. Wilfred D'souza · Dr. Niharika Reddy

WEAVE . CENTRE FOR INTEGRATIVE PSYCHIATRY

Contents

Concept / rule Key number Trap

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.

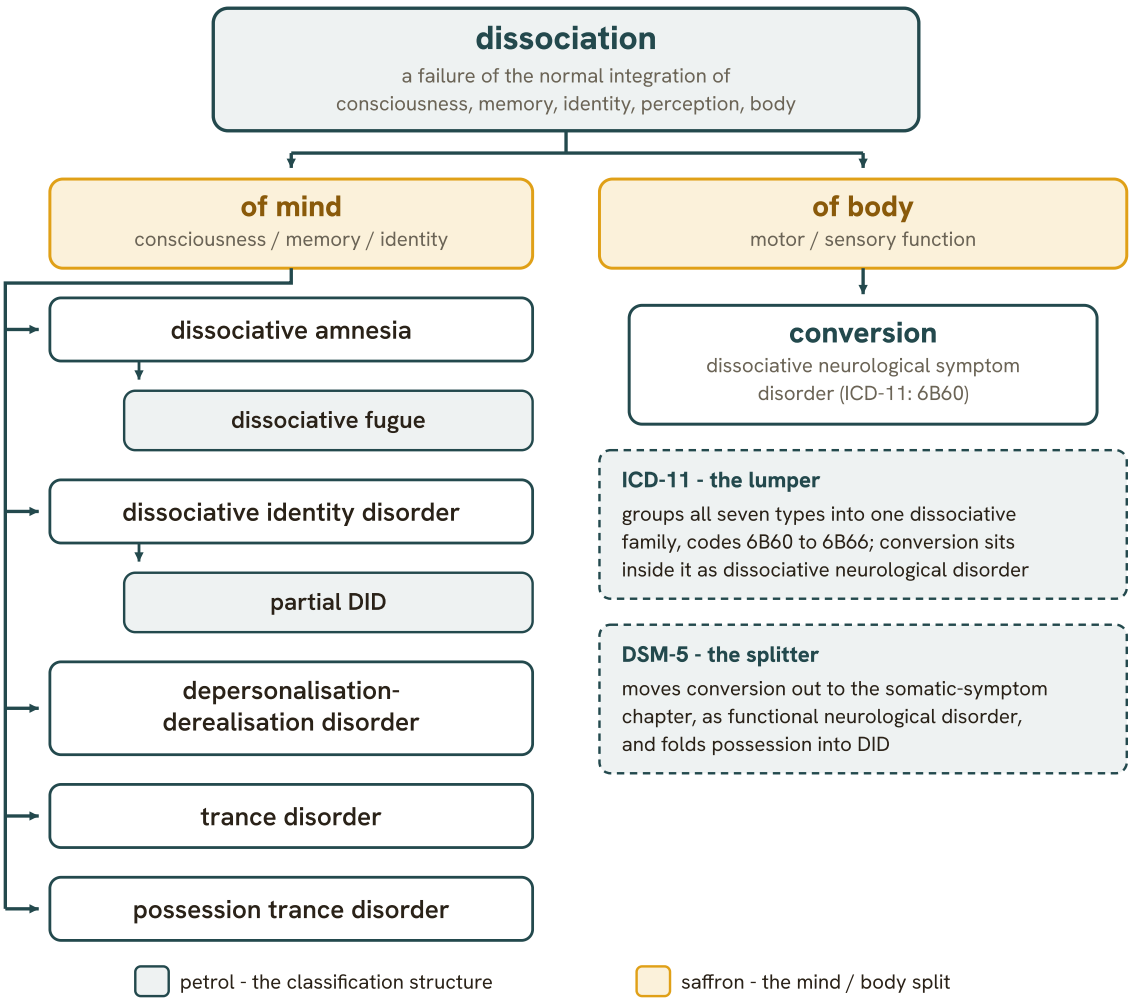


Fig 15.2 The Dissociative Experiences Scale (DES-II) reproduced in full: the 28-item self-report screen for dissociative experiences, scored as the mean of all items.

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.

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.

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.

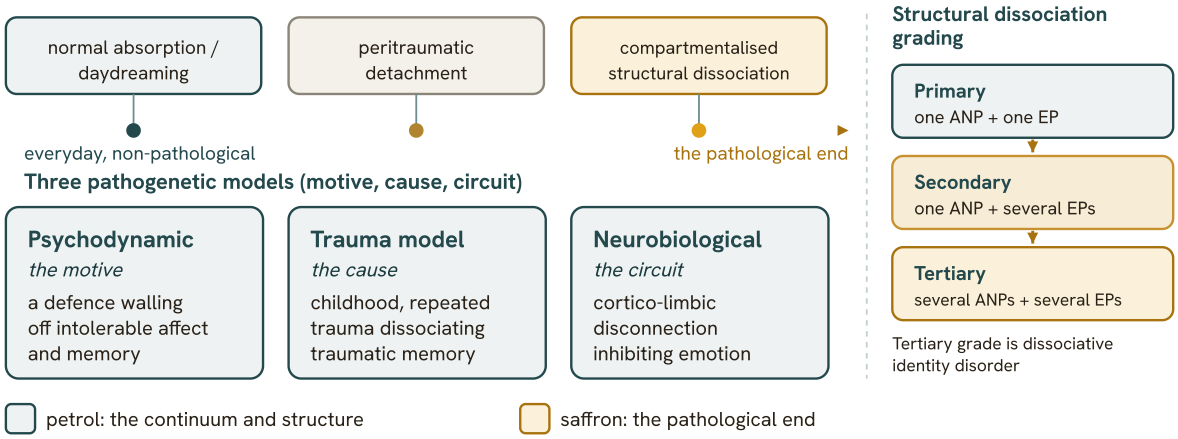
2 · The pathogenesis of dissociation

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.

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 15.5 The positive rule-in signs of a functional neurological disorder, grouped by domain - each one demonstrating internal inconsistency at the bedside.

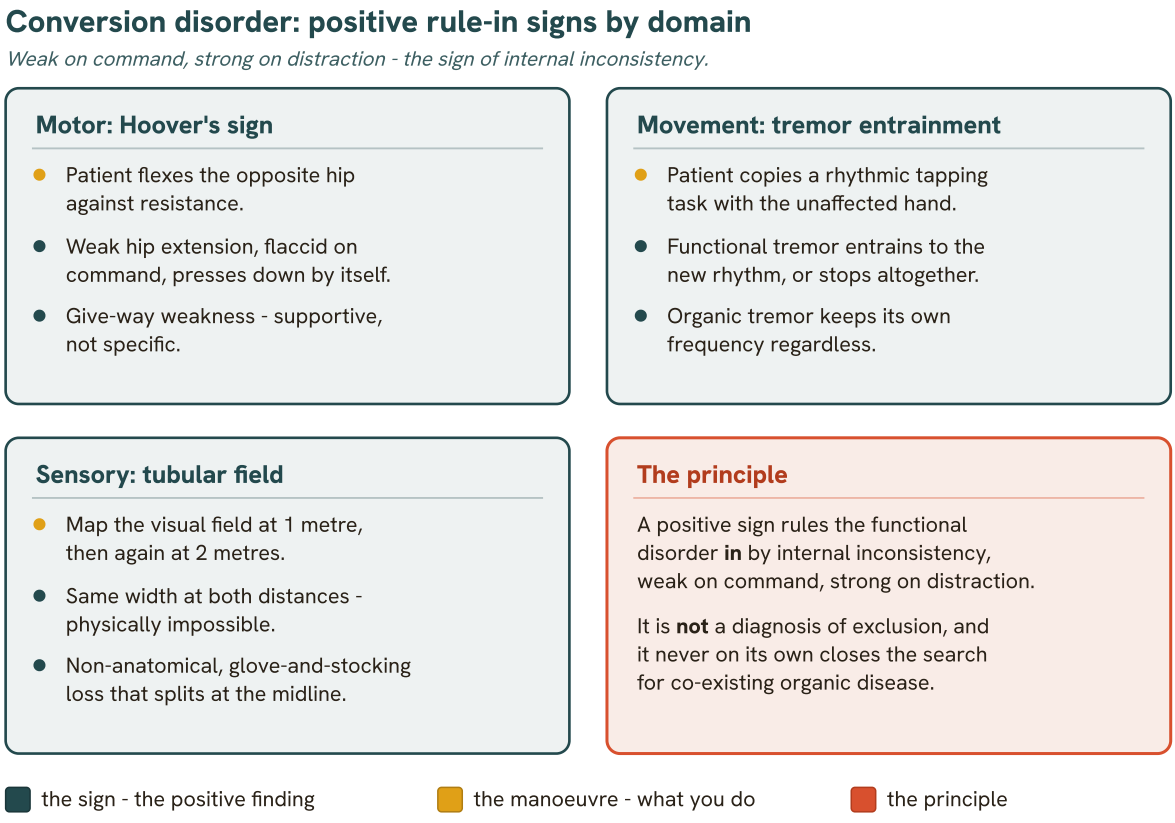


Fig 15.6 How to tell a functional (conversion) presentation from organic disease across five features, and why a positive sign never closes the organic work-up.

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 15.7 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.

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.

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.

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.

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 15.8 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.

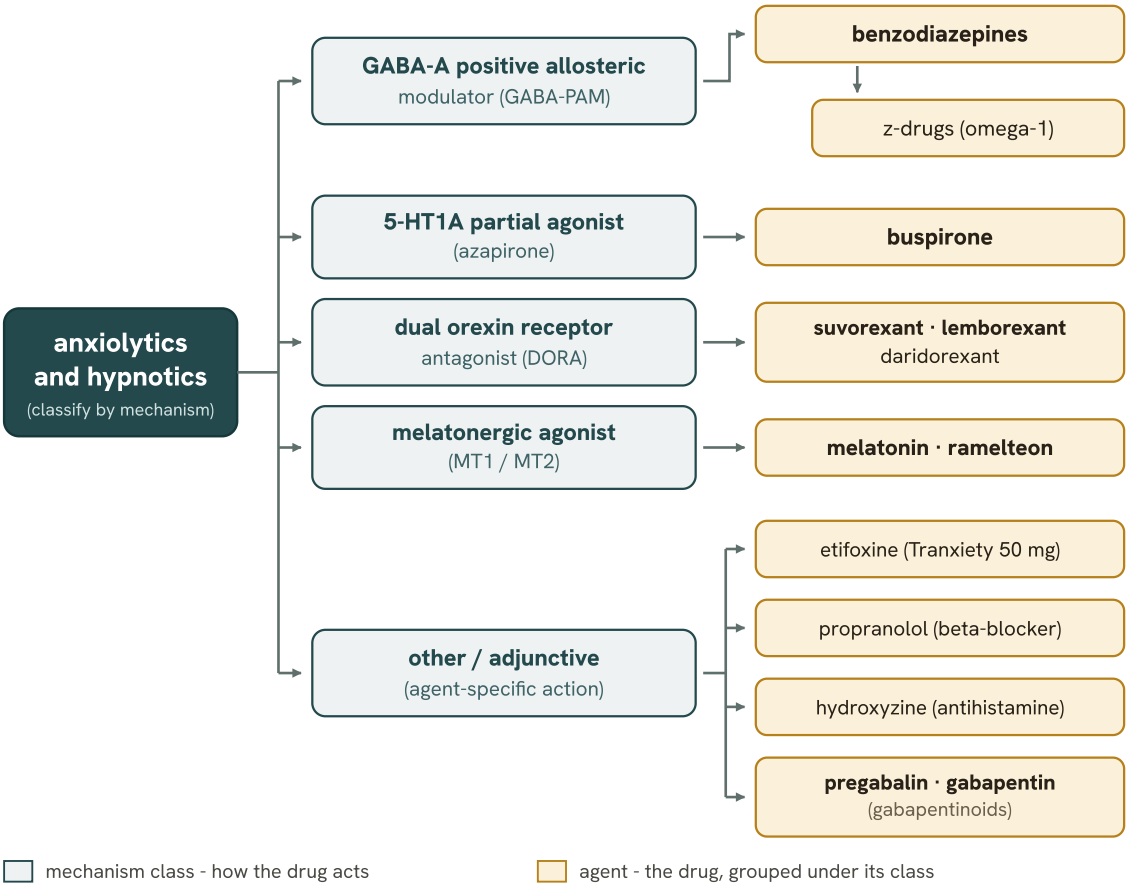


Fig 15.9 The GABA-A receptor is a pentameric chloride channel; the benzodiazepine binds the allosteric site at the alpha/gamma interface and, only with GABA present, raises the frequency of channel opening.

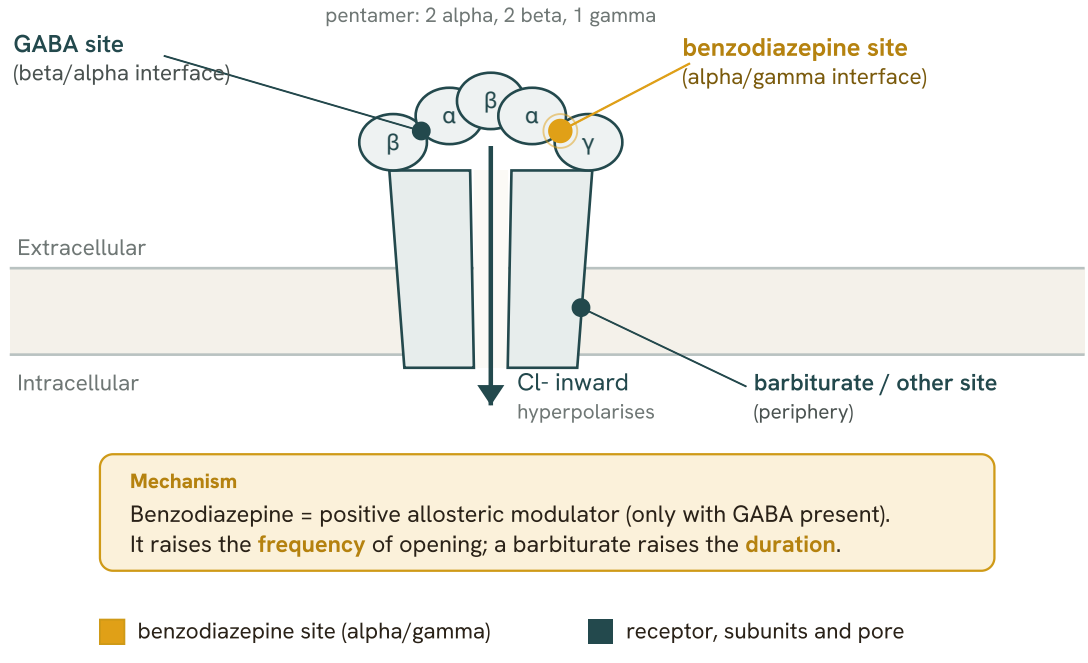


Fig 15.10 The benzodiazepines sorted by elimination half-life into short-, intermediate- and long-acting classes, with the shared long-acting metabolite and the duration-to-use rule.

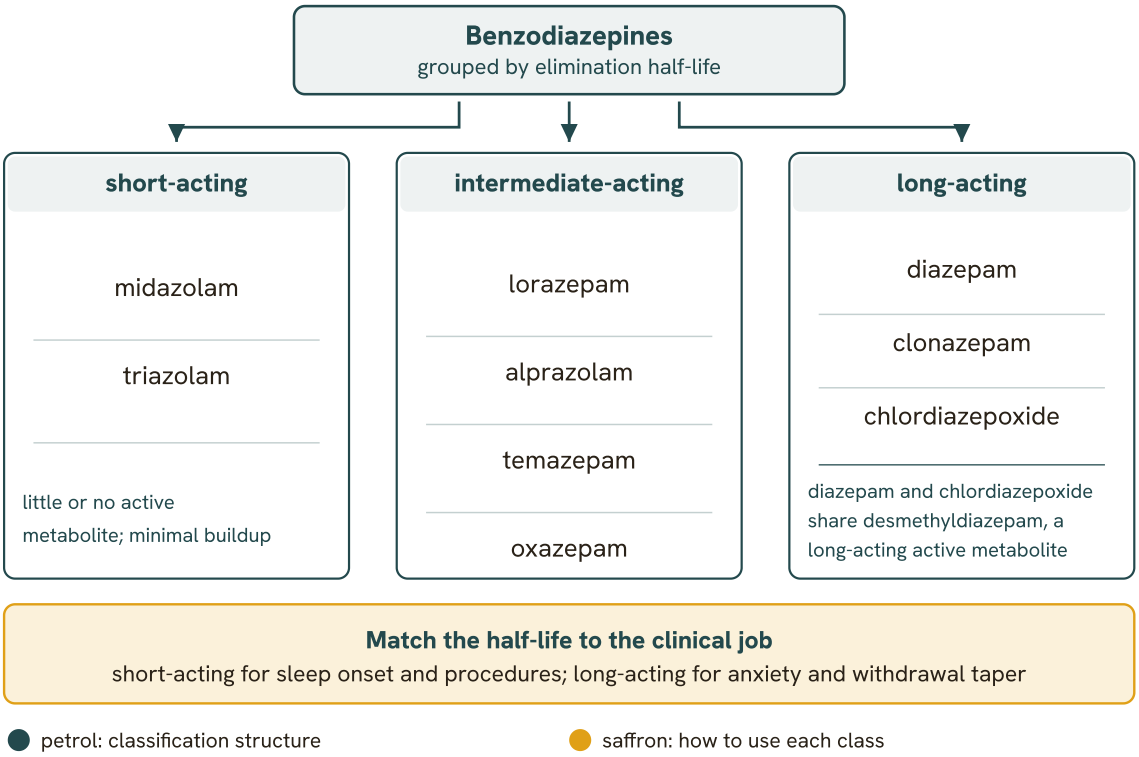
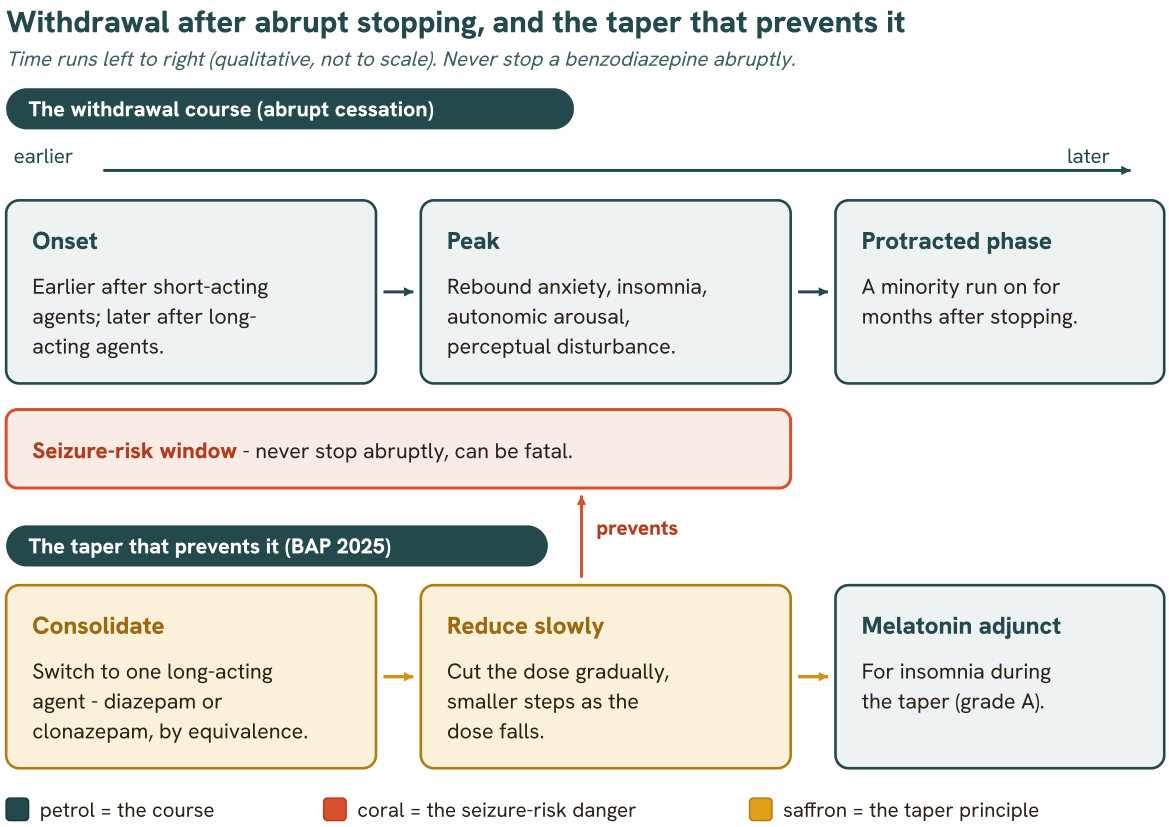


Fig 15.11 The benzodiazepine withdrawal course after abrupt cessation, and the long-acting taper that prevents it.



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

Lorazepam, **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	1 to 2 wk	2 to 4 wk
INTERVAL AT WHICH INTERDOSE REBOUND ANXIETY FOLLOWS A SHORT-ACTING AGENT	DELAY BEFORE WITHDRAWAL STARTS AFTER A LONG-ACTING AGENT IS STOPPED ABRUPTLY	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.

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 SUVOREXANT MEAN HALF-LIFE (HOURS)	**10 to 20** SUVOREXANT DOSE (MG PER DAY)	**25 to 50** DARIDOREXANT DOSE (MG PER DAY)	**7** 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 re-bound 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 15.12 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.

SOURCE HIERARCHY (PSYCHIATRY AND PSYCHOPHARMACOLOGY TEXTS AND CLASSIFICATIONS)

1. Boland R, Verduin ML, eds. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 11th ed. Philadelphia: Wolters Kluwer; 2024. [the dissociative-disorders phenomenology, the psychodynamic, trauma and neurobiological pathogenesis models, the DES means and the anxiolytic and hypnotic pharmacology depth bar]
2. Geddes JR, Andreasen NC, Goodwin GM, eds. New Oxford Textbook of Psychiatry. 3rd ed. Oxford: Oxford University Press; 2020. [Stone and Carson on functional neurological disorder: the positive-sign, rule-in principle, Hoover's sign, tremor entrainment, functional gait and the FND management pillars]

3. Cowen P, Harrison P, Burns T. Shorter Oxford Textbook of Psychiatry. 7th ed. Oxford: Oxford University Press; 2018. [pregabalin as the alternative first-line anxiolytic and its faster onset than an SSRI]
4. Johnstone EC, Owens DGC, Lawrie SM, McIntosh AM, Sharpe M, eds. Companion to Psychiatric Studies. 8th ed. Edinburgh: Churchill Livingstone/Elsevier; 2010. [the dissociative-versus-epileptic seizure semiology (Box 13.12) and the unreliable discriminators; the azapirone family]
5. Tasman A, Kay J, Lieberman JA, First MB, Riba MB, eds. Psychiatry. 4th ed. Chichester: Wiley-Blackwell; 2015. [Janet's desagregation, the z-drug elimination half-lives and the omega-1 selectivity]
6. Stahl SM. Stahl's Essential Psychopharmacology: Neuroscientific Basis and Practical Applications. 5th ed. Cambridge: Cambridge University Press; 2021; and Stahl SM. Stahl's Prescriber's Guide. Cambridge: Cambridge University Press. [the GABA-A benzodiazepine mechanism and subtype pharmacology, the melatonergic MT1/MT2 and orexin systems, the z-drug omega-1 selectivity, buspirone and etifoxine mechanisms]
7. Taylor DM, Barnes TRE, Young AH. The Maudsley Prescribing Guidelines in Psychiatry. 14th ed. Chichester: Wiley-Blackwell; 2021. [the half-lives, the diazepam-equivalence table (Table 3.29), flumazenil, the alprazolam overdose toxicity and the taper]
8. Tripathi KD. Essentials of Medical Pharmacology (KDT). 8th ed. New Delhi: Jaypee; 2019. [the GABA-A chloride-channel mechanism, the frequency-versus-duration barbiturate contrast, the orexin/hypocretin system and chlormezanone]
9. Kaufman DM, Geyer HL, Milstein MJ. Kaufman's Clinical Neurology for Psychiatrists. Philadelphia: Elsevier. [melatonin and ramelteon, the circadian indications and the ramelteon CYP1A2/fluvoxamine interaction]
10. World Health Organization. ICD-11 Clinical Descriptions and Diagnostic Requirements for Mental, Behavioural and Neurodevelopmental Disorders (CDDR). Geneva: WHO; 2024. And: American Psychiatric Association. DSM-5-TR. Washington, DC: APA; 2022. [the dissociative-disorders classification and the ICD-11-versus-DSM-5-TR filing of conversion, trance, possession and partial dissociative identity disorder]
11. Further pharmacology sources consulted for specific dose, half-life and equivalence detail: Saunders JB, et al, eds. Addiction Medicine (Oxford) for the z-drug diazepam equivalences (Table 3.4); the Maudsley Deprescribing Guidelines and Dulcan's Textbook of Child and Adolescent Psychiatry for the z-drug complex-sleep-behaviour caution; Lowinson and Ruiz's Substance Abuse for the z-drug dependence liability.

GUIDELINES (BAP CONSENSUS STATEMENTS PUBMED-VERIFIED; NICE AND IPS CITED AUTHOR-TITLE-YEAR)

1. Baldwin DS, Anderson IM, Nutt DJ, et al; British Association for Psychopharmacology. Evidence-based pharmacological treatment of anxiety disorders, post-traumatic stress disorder and obsessive-compulsive disorder: a revision of the 2005 guidelines. J Psychopharmacol. 2014;28(5):403-439. PMID: 24713617. [the SSRI-first-line position for GAD, buspirone and hydroxyzine as acute-only agents, and the benzodiazepine-sparing sequence]
2. Wilson S, Anderson K, Baldwin D, et al; British Association for Psychopharmacology. Consensus statement on evidence-based treatment of insomnia, parasomnias and circadian rhythm disorders: an update. J Psychopharmacol. 2019;33(8):923-947. PMID: 31271339. [CBT-I first-line; the z-drug, prolonged-release melatonin, low-dose doxepin and suvorexant short-course positions; match the half-life to the insomnia pattern]
3. Sinclair JMA, Kalk NJ, Lingford-Hughes A, et al; British Association for Psychopharmacology. Evidence-based consensus guidelines for the pharmacological management of substance dependence. J Psychopharmacol. 2026;40(2):227-272 (published online 2025). PMID: 41731947. [the benzodiazepine taper: consolidate onto a long-acting agent then reduce slowly; melatonin as the withdrawal-insomnia adjunct. The build fragments cite this as "BAP 2025, Lingford-Hughes et al"; the lead author is Sinclair, with Lingford-Hughes a co-author, corrected here]
4. National Institute for Health and Care Excellence. Generalised anxiety disorder and panic disorder in adults: management (CG113). London: NICE; 2011 (updated). [the SSRI/SNRI/pregabalin stepped-care pathway and the benzodiazepine as a 2 to 4 week crisis measure only]
5. National Institute for Health and Care Excellence. Guidance on the use of zaleplon, zolpidem and zopiclone for the short-term management of insomnia (TA77). London: NICE; 2004 (reviewed 2010). [the z-drugs restricted to short-term use at the lowest effective dose, with no z-drug-to-z-drug switching]
6. Indian Psychiatric Society. Clinical Practice Guidelines (IPS_CPG, 2017). Indian Psychiatric Society. [the Indian insomnia and anxiolytic framing and the benzodiazepine-sparing, short-course position]

INSTRUMENTS AND STUDIES (PUBMED-VERIFIED JOURNAL ARTICLES; TWO INSTRUMENTS NOT MEDLINE-INDEXED)

1. Bernstein EM, Putnam FW. Development, reliability, and validity of a dissociation scale [the Dissociative Experiences Scale, DES]. J Nerv Ment Dis. 1986;174(12):727-735. PMID: 3783140.
2. Carlson EB, Putnam FW. An update on the Dissociative Experiences Scale [the DES-II]. Dissociation. 1993;6(1):16-27. [the journal Dissociation is not MEDLINE-indexed, so cited author-title-year without a PMID]
3. Waller NG, Putnam FW, Carlson EB. Types of dissociation and dissociative types: a taxometric analysis of dissociative experiences [the DES-Taxon, DES-T]. Psychol Methods. 1996;1(3):300-321. [Psychological Methods is not MEDLINE-indexed, so cited author-title-year without a PMID]
4. Nijenhuis ER, Spinhoven P, Van Dyck R, Van der Hart O, Vanderlinden J. The development and psychometric characteristics of the Somatoform Dissociation Questionnaire (SDQ-20). J Nerv Ment Dis. 1996;184(11):688-694. PMID: 8955682.
5. Sierra M, Berrios GE. The Cambridge Depersonalization Scale: a new instrument for the measurement of depersonalization. Psychiatry Res. 2000;93(2):153-164. PMID: 10725532.
6. Dalenberg CJ, Brand BL, Gleaves DH, et al. Evaluation of the evidence for the trauma and fantasy models of dissociation. Psychol Bull. 2012;138(3):550-588. PMID: 22409505.
7. Vonderlin R, Kleindienst N, Alpers GW, Bohus M, Lyssenko L, Schmahl C. Dissociation in victims of childhood abuse or neglect: a meta-analytic review. Psychol Med. 2018;48(15):2467-2476. PMID: 29631646.

Index